The role of familiarity and similarity in action understanding and imitation: investigating mirror neurons in Saudi children with ASD

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Abbreviations

Autism Spectrum Disorder (ASD)

Attention deficit hyperactivity disorder (ADHD)

Typically developed (TD)

Motor evoked potentials (MEPs)

Electroencephalography (EEG)

Autism Diagnostic Interview –Revised (ADI-R)

Mirror Neurons (MNs)

Magnetoencephalography (MEG)

Analysis of Variance (ANOVA)

Left central electrode (C3)

Right central electrode (C4)

Central midline electrode (Cz)

Functional Magnetic resonance image (fMRI)

Kilohm (KW)

Magnetic Resonance Image (MRI)

Number of subjects (n)

P value (P)

Standard errors (SE)

Transcranial magnetic stimulation (TMS)

Positron emission tomography (PET)

Theory of Mind (ToM)

Abstract

'Mirror Neuron Theory' is a brain process model which is based on a direct-matching model, that encodes the motor features, mental states, and the goal of observed actions onto the observer's own motor system. MNs abnormalities and Autism Spectrum Disorder (ASD) have been empirically associated as they are alleged to represent the neural basis of deficits in social competence and imitative learning in ASD.

Neurophysiological evidences nonetheless appear to validate the enhanced activity of MNs when utilizing a familiar agent (person) with ASD. Similar evidence suggests influence of the individual's own culture, compared to others, on modulating the mirror neuron; however, this hypothesis has never been tested on an ASD group. Other behavioural data show that the use of typically developing peers as models in a social intervention setting with ASD was advocated for its significant outcomes, but the impact of age similarity on modulating MNs in ASD children was not directly investigated.

In these four EEG experiments, we investigate the effect of observing a familiar person, a person from a similar age group and someone from a similar ethnic group, performing actions, on the capacity of understanding and imitation of others' actions. Additionally, we consider if observing a prime, familiar person, similar ethnic-person, or similarly-aged person would facilitate action understanding and imitation if this action were then seen performed later by an unfamiliar person, dissimilar-ethnic person, or dissimilarly-aged person, in young children with ASD, compared to a control group.

Participants watched people performing gestures, crossing familiarity of the person (parent/ stranger), similarity of the person's age (child/ adult), or of the person's ethnicity (Saudi/ European), with familiarity of the action (meaningful/ meaningless). MNs activity was indexed by alpha (8-12 Hz), low beta (13-20 Hz), and theta (5.5-7.5Hz) desynchronization over the sensorimotor cortex. Behavioural performance was recorded through the imitation stage.

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1 Literature Review

In Chapter 1, we will present some of the related work, theoretical background and concepts concerning the experiments conducted in this thesis. In particular, we will define ASD and the models used to explain its etiology. We will focus on the MN system and its role in social cognition, with reference to abnormalities in this MN system, and how this might relate to the ASD clinical profile.

A brief overview of familiarity and similarity will be provided, along with a review of the literature regarding their effectiveness in modulating MNs, and their effects on action understanding and imitation, which relate to our investigation. Finally, we will lay down a practical overview of the experimental paradigm being employed in the current series of experiments, and introduce the methodological techniques. A more detailed introduction to each of these subjects is included in the pertaining chapters. In the experiments conducted in this thesis, we touch upon three main cognitive mechanisms: 'motor reasoning,' 'familiarity or similarity,' and 'imitation,' and examine the interaction between them.

1.1 Autism Spectrum Disorders ASD

Seventy years ago, Leo Kanner (1943) and Hans Asperger (1944) were the first to label a specific set of brain development disorders as Autism Spectrum Disorders (ASD). ASD is a pervasive neurodevelopmental behavioural disorder that is characterised by widespread abnormalities in social interactions, abnormal functioning in use of language, imaginative or symbolic play, and restricted and repetitive interests and behaviours, with a typical onset prior to 36 months (American Psychiatric Association, 2000).

Under ASD, the range of conditions varies: in classic autism the individual displays severely impaired learning and intellectual abilities. Asperger syndrome includes in its characteristics social and communication impairments with preserved language skills and intellectual abilities (Attwood, 2006). Lastly, pervasive developmental disorder, not otherwise specified (PDD-NOS)can be diagnosed when the criteria for autism or Asperger syndrome are not met (Johnson & Myers, 2007). ASD prevalence estimations

reveal that 1 in 88 individuals are affected, and this figure varies by gender, with 1 in 54 boys and 1 in 252 girls currently diagnosed with the disorder (2012 CDC estimate). The controversy surrounding ASD etiology has led to the development of several models (e.g. genetic, environmental factors, and brain process) through which its core deficits are described.

Although a number of consistent findings have emerged from these models, it has not yet been firmly established whether these etiologies are primary causal or ancillary (Bailey et al., 1995; Rodier & Hyman, 1998). Within this framework, ASD is considered to be heterogeneous, in cause and in its clinical profile. It is found to be associated with a number of clinical abnormalities such as epilepsy, attention deficit hyperactivity disorder (ADHD) and various brain pathologies. There are a number of indicators and symptoms, rather than clinical criteria, that are manifested by individuals with ASD, for example, difficulty in joint attention related tasks, which refers to the ability to use eye contact and gestures in order to coordinate attention with another individuals (e.g. pointing to an object) to share a specific experience or interesting event (Bruner and Sherwood, 1983).

The second indicator is a difficulty in Theory of Mind (ToM) related tasks which comprise a difficulty in attributing mind-states, beliefs, intentions, and feelings to others (Baron-Cohen, 2000). This, as a result, leads to reciprocal difficulty in speculating on the appropriate course of social interactions with others (Baron-Cohen, 1995). From a different perspective, some genetic data has led to the argument that single-gene disorder is found to be associated with ASD. Nonetheless, single-gene accounts for only 10% - 20% of cases within the ASD population. On the other hand, genetic syndromes occur in individuals cleared of ASD diagnoses, while other researchers suggest that environmental factors (e.g. parental age or exposure to toxins) are likely to be the cause (NIMH, 2007).

1.2 Mirror neurons MNs

The MN system is one of the brain process models which, in principle, is based on a direct-matching model (Rizzolatti & Sinigaglia, 2010), that encodes the motor features,

emotions, mental states, and the goal of others 'actions onto the observer's own motor system (Barsalou, 1999; Goldman, 2006; Gallese, Keysers, & Rizzolatti, 2004). The function of 'encoding' represents the ability to understand actions, goals and intentions using MNs, which in turn, seems to explain social skills, including theories of 'mindreading' (Gallese et al., 2004).

About 18 years ago, a subtype of sensorimotor neurons was discovered in the ventral premotor cortex of the macaque monkey brain using single cell recording (Rizzolatti et al., 1996; Gallese et al., 1996) and subsequently in the inferior parietal lobe (PF/ PFG) (Gallese et al., 2002; Fogassi, Ferrari, Gesierich, Rozzi, Chersi, & Rizzolatti, 2005). These neurons were primarily found to increase in activity when a monkey performs goal-directed hand action (transitive action), or when a monkey passively observes such a hand action performed by a conspecific towards a target. However, these neurons seem to fail to fire in the absence of a visual or acoustic target that the animal is aware of, which suggests that MNs in monkeys are specifically restricted to the visual and auditory perception of actions directed towards a target (Keysers et al., 2003).

Mirror neurons are believed to exist in humans, and are thought to comprise, beside transitive actions, intransitive and ostensive communicative gestures (Jacob, 2008). Preliminary work emerged by Fadiga, Fogassi, Pavesi & Rizzolatti (1995), who conducted a transcranial magnetic stimulation (TMS) study whereby electromagnetic induction is used to induce an electric current; hence, when applied to the scalp, neurons underneath the scalp actively fire as a result of the induced electric current (Jalinous & Freeston, 1985).

This method was used to show the link between the primary motor cortex stimulation and recorded muscle action potentials reflecting the human homunculus. In Fadiga et al.'s study, TMS was applied to the primary motor cortex of participants while they observed the researcher grasping an object, or observing a flashing light. Cortex excitability was measured using motor evoked potentials (MEPs) from the participants' hand muscles. Stimulation was applied to the left hemisphere hand area of the sensory motor cortex (homunculus) and muscles in the right hand were recorded while

participants observed the grasping actions or flashing light. Findings revealed that motor-evoked potentials recorded from hand muscles increased exclusively during the observation of hand movements.

Research that records brain electrophysiology, whereby an Electroencephalogram (EEG) measures the brain ś electrical activity as the voltage fluctuations that are formed by neural activity causing current flows, agrees with these findings. Specifically, studies have shown that when a human subject passively observes hand movements there is a desynchronization in the oscillatory activity in the mu (8-12Hz) and beta (15-30Hz) bands over the sensorimotor cortex similar, although weaker, to that occurring during self-execution actions (Cochin, Barthelemy, Lejeune & Martineau, 1998; Hari, Levanen & Raij, 2000). Further congruent evidence has been revealed by two important functional Magnetic Resonance Imaging (fMRI) studies.

Firstly, Buccino et al., (2001) showed that the corresponding neural structures that are involved during active execution are also recruited during action observation of different effectors (hand, foot, and mouth). Nonetheless, it could still be argued that the corresponding neural activity of observing and executing was generated by different neural populations within the same voxel. This debatable point requires evidence of individuals employing the same neural populations during both execution and observation of the same action.

Such evidence was provided by Kilner, Neal, Weiskopf, Friston, and Frith, (2009), in their cross-modal repetition suppression study. Their philosophy was built on the fact that participants, while observing and executing the same action, should manifest reduced responses in the inferior frontal gyrus (IFG), thus making it evident that the same population of neurons have been recruited during both tasks of the same action.

Much of the current empirical evidence that supports the existence of MNs in human, discussed earlier, has used indirect measuring methods (e.g. neuroimaging and electrophysiology studies), which means that their existence remains under debate. Nonetheless, recently, Mukamel Ekstrom**,** Kaplan, Iacoboni, and Fried (2010) have successfully carried out the first single-neuron recording study. They recorded

extracellular activity from 1177 cells in medial frontal and temporal cortices during passive observation, executing hand grasping actions and facial emotional expressions. Their findings revealed neural responsiveness from two major areas during observation and execution of the actions: the supplementary motor area and hippocampus.

Identifying the brain region related to MNs was a major area of interest. Use of fMRI helped researchers initially to localize two main areas: the ventral premotor cortex of the inferior frontal gyrus (IFG) and the superior parietal lobe. The homologue brain areas in monkeys are area F5 and the Inferior Parietal Lobule (IPL), which were shown to be active when the monkey performs an action and when the performance of the action is observed (Rizzolatti & Craighero, 2004).

Following this study, a number of other cortical areas have been related to MNs activity, such as the Superior Temporal Sulcus (STS), supplementary motor areas, Broca's area, primary somatosensory cortex, insula and the Anterior Cingulate Cortex (ACC) (Rizzolatti & Fabbri-Destro, 2008). Within the realm of social cognitive neuroscience, researchers have inspected a cluster of social cognitive mechanisms such as mental states, attributing intentions, inferring, action perception and social communication (ToM). The neural networks that underlie these processes are collectively known as the 'social brain' (see figure 1.1) (Frith & Frith, 2010), which comprises (IFG), (pSTS), (ACC), the medial prefrontal cortex (mPFC), the inferior parietal lobe (IPL), the temporoparietal junction (TPJ), the intraparietal sulcus (IPS), the amygdala and insula.

A number of cortical areas that are important for social cognition seem to have equivalent connections to the MNs areas (Uddin, Iacoboni, Lange & Keenan, 2007). In that regard, MNs dysfunction is thought to emerge in individuals who experience social deficits, as represented by poor communication, poor understanding of other's intentions and actions, poor imitation ability, and delayed language acquisition. Critically these impairments represent the main features that are manifested in individuals with ASD (Welsh, Ray, Weeks, Dewey & Elliott, 2009). Hence, identifying the possible properties, functions, and cognitive mechanisms underlying these neurons has become scientifically controversial.

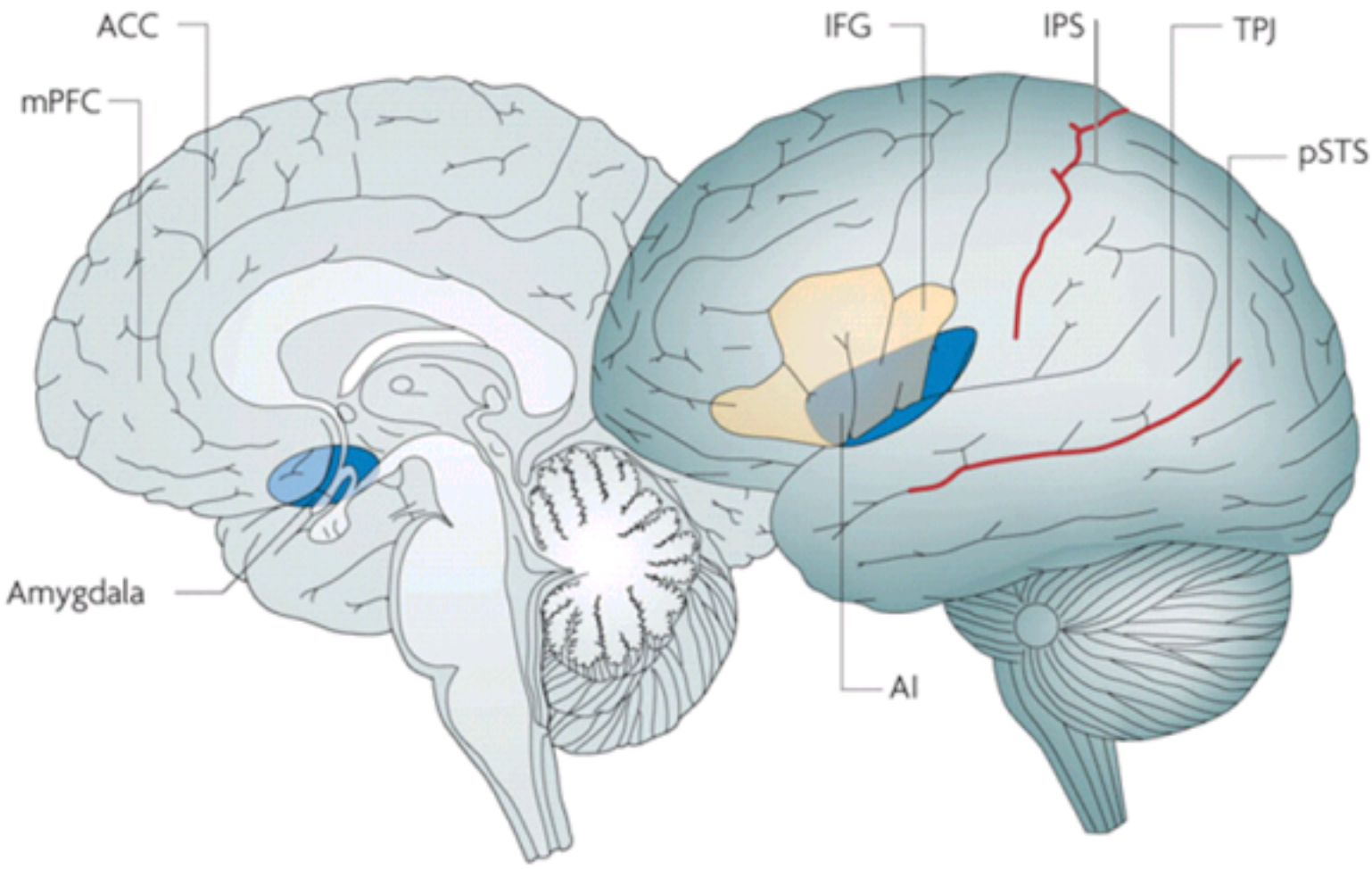

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Figure 1 Illustration shows the main brain areas associated with social cognition. This comprises the medial prefrontal cortex (mPFC), the posterior superior temporal sulcus (pSTS), anterior cingulate cortex (ACC), anterior insula, the temporoparietal junction.

1.3 Functions of MNs

In the years following the discovery of MNs, a number of theories have surfaced stressing the functions and significance of these neurons. Resultantly, an equal number of theories have risen that deny the significance of MNs specified in the former kind of studies criticising them as exaggerated or merely speculation. Some researchers stress the role of MNs in direct motor resonance functions and goal-understanding (Rizzolatti & Fabbri-Destro, 2013; Gallese, 2014; Ramachandran & Oberman, 2006; Keysers & Gazzola, 2009) while other researchers state that MNs merely help in anticipating the outcome of an observed action (Southgate et al., 2009; Southgate & Begus, 2013; Cook et al., 2014; Southgate et al., 2014).

In their study, Rizzolatti and Fabbri-Destro (2013) state that the behaviours of others

represent their intentions and goals which may be recognised by those observing them. Referencing the TMS studies, the authors argue that humans are capable of coding both transitive and intransitive gestures using the mirror mechanism. Rizzolatti and Fabbri-Destro (2013) also contend that the velocity of both executed and imagined movements result in similar cortical frequency bands. While providing evidence from several studies, the authors presented the theory that humans have the direct mapping mechanism of the observed movements on the motor cortex (Arbib et al., 2008). In addition to that, humans are capable of recognising the emotions of others by activating the same emotions in themselves which can also be explained by the mirror mechanism (Fogassi, 2014).

In an attempt to evaluate the role of MNs in ASD, Rizzolatti and Fabbri-Destro (2013), focused on ASD children and typically developing (TD) children and highlighted the differences in responsiveness of children towards observation of object-directed action. The results of the study showed the lack of mirror mechanism in ASD children.

The research was done using two investigative actions: action observation and understanding of the performer's intentions. While TD children were successful in identifying both the action and intention, ASD children were unable to comprehend the intention behind the action. Thus, the study attempted to prove that the occurrence of ASD may be due to a lack of or deficiency of MNs.

Gallese (2014) uses MNs to explain intersubjectivity or the theory of embodied simulation (ES). Intersubjectivity is the conceptualisation of a psychological relationship between people, and according to Gallese (2014), the discovery of MNs provides a fresh perspective to explain interpersonal understanding, mindreading, and understanding emotions and sensations. Previously, empathy was described as the source of an individual's social cognition; however with the emergence of understanding of MNs, intersubjectivity can become the focus in understanding human social intelligence. In addition, the theory of ES challenges the widespread belief that folk psychology is the only way to describe interpersonal understanding.

Ramachandran and Oberman (2006) call MNs the great leap forward in human

evolution and present the significance of MNs by linking them to some of the major human functions. The authors attempt to establish a relationship between MNs and imitation learning in human evolution. According to the authors, MNs are responsible for the emergence of language and the "big bang of human evolution" (Ramachandran & Oberman, 2006, p.4). The authors place mirror mechanism right in the middle of co-evolution which resulted in the development of sophisticated tools, transmission of knowledge, and establishment of cultures and societies.

The great leap in human evolution or the sudden expansion of sophisticated technology and behaviour resulted from imitation learning and emulation. By linking MNs with the emergence of language, the theory emphasises the communicative actions triggered by MNs. While Ramachandran and Oberman (2006) are highly optimistic about the functions and significance of MNs, Heyes (2010) directly opposes the view presented by Ramchandaran and Oberman (2006). Heyes (2010) states that the ongoing arguments pertaining to MNs only imply that they are the cells responsible for action understanding and they may be a by-product of associative learning.

Heyes (2010) critically evaluates the role of MNs from the perspective of associative learning and highlights that there are a number of factors that show MNs as the product of social interaction. The author bases this argument on the increasing evidence that MNs are responsible for a number of social cognitive functions and they are not active in action understanding. In addition to that, the associative learning perspective of MNs implies that these cells result from sensorimotor experience which is also linked with social interaction.

The arguments presented by Heyes (2010) are also accepted by Cook et al. (2014) who argue that MNs result from sensorimotor associative learning. The authors disregard the research available on MNs due to its lack of direction, and suggest that a novel approach should be taken to evaluate the functions of MNs in prospective research studies. The authors argue that during two decades of research since the discovery of MNs, there is no concrete evidence that these cells exist in humans, and nothing to suggest whether they are actually responsible for all the functions that have been associated with these cells in other studies.

A number of researchers believe that MNs were adapted genetically for action understanding and they are a result of evolution in order to equip the carriers with socio-cognitive function (Rizzolatti & Fabbri-Destro, 2013; Gallese, 2014; Ramachandran & Oberman, 2006). In contrast, Cook et al. (2014) argue that MNs are forged by processes of associative learning during the phase of individual development. Even though MNs may be responsible for certain psychological functions, this does not conclusively indicate that these neurons have a clear evolutionary purpose.

Cook et al. (2014) support their argument with evidence suggesting that: MNs fail to encode intentions behind actions on a consistent basis; the context-sensitive nature of associative learning explains the properties of MNs comprehensively; MNs can be developed and modified using sensorimotor training and human infants are exposed to sufficient sensorimotor training to help them develop MNs through associative learning (Bonaiuto, 2014; Cook & Bird, 2012; Duran et al., 2014; Oosterhof et al., 2014).

Perkins et al. (2010) add to the criticism towards theories suggesting roles of MNs in human cognitive development. The authors argue that there is no conclusive evidence suggesting the existence of MNs in humans. In addition to that, the MNs observed in Macaques form a minority of only 6% of observed cells. The authors also argue that the theory of imitation learning is too complex to be explained simply by a mirror mechanism.

Southgate et al. (2009) also argue that over two decades, very little evidence has arisen which proves the existence of MNs in infancy. Although the authors agree that some parts of the brain show similar patterns while performing and observing the same action, this behaviour cannot be conclusively linked to the existence of a mirror mechanism in infancy. The authors conducted a study involving nine month old infants by identifying individual frequency ranges using EEG. The researchers identified sensorimotor alpha band activity in infants while they reached for objects or observed the same action being performed by someone else. The results showed that observing the same action resulted in motor activation in infants, however, this activity began before observing the action and as soon as it could be anticipated. Thus, the authors

concluded that the motor activation was not entirely triggered by visual input but by the infant's understanding of the action that was about to take place, though it should be noted that the results provide evidence of action anticipation within the context of object-directed action.

In another study, Southgate and Begus (2013) also argued that the inferences generated by the brain from motor activation and the overall mechanism at work in such activation are unknown. The authors challenge the theories that suggest that motor activation is the result of goal identification, and assert that the actual scenario is contrary to the popular theories, that is, action anticipation results in motor activation.

Action anticipation was discussed within the context of motor chains in similar work by Cattaneo and colleagues (Cattaneo et al., 2007). EMG was recorded from ASD and typically developing children during execution and observation of object-hand actions. Participants were requested to pick up a piece of food to eat or to place on their shoulder, and thus, the action involved three steps: reaching, grasping, and finally placing the food. The observation conditions entailed observing similar actions of execution conditions performed by someone else.

The EMG activity was recorded from the mouth-opening mylohyoid muscle which is supposed to present activity in the face of grasped food intended for eating, but not intended for placing elsewhere. Results revealed that only TD children activate the MH muscle during the grasping phase when observing someone else performing the same action, proposing anticipation of the action. The activity was recorded from the parietal MNs area, and thus the researchers propose that the abnormal activity recorded in ASD, reflects abnormal functioning of MNs.

Gallese and Sinigaglia (2014) challenge the arguments presented by Cook et al. (2014), Southgate et al. (2009), and Southgate and Begus (2013) with regard to the relationship between MNs and action understanding, and the primacy of mirror mechanism. According to the authors, a mirror mechanism is present in humans at birth which can be modulated by motor experiences and enhanced further through visuomotor experiences. Unlike the theory of development of MNs through associative learning as

presented by Cook et al. (2014), Gallese and Sinigaglia (2014) present the theory that the presence of mirror mechanism at birth contributes towards understanding actions. The authors also reference the studies conducted by Cannon and Woodward (2012) and Kanakogi and Itakura (2011) to strengthen their argument regarding the primacy of mirror mechanism. In addition to that, Oberman et al. (2014) also agree that development of MNs in humans cannot be based solely on associative learning.

Although multiple theories explain the functions of MNs, the majority of these theories have been generated from object-directed actions studies. There is a lack of studies evidencing the functions of MNs through the use of intransitive actions, particularly, communicative gestures.

The arguments presented by Heyes (2010) and Cook et al. (2014) also hinder acquisition of an understanding of the role of MNs in the context of communicative gestures using the existing theories. These authors argued that MNs develop as a result of sensorimotor associative learning; however, they do not explain the role of communicative gestures in development of MNs. These theories show that the visual system and motor system collectively help in the development of correlated MNs. On the other hand, in the context of communicative gestures, the motor system is not object-directed.

Montgomery et al. (2007) shed light upon the relevance of MNs in the context of communicative hand gestures. The authors present results from neuroimaging experiments which identified inferior parietal lobule (IPL) and frontal operculum as major parts of MNs. According to the authors, MNs are activated when an individual observes the execution of actions including object-directed hand movements; however, the previous studies suggest that no such activation is detected when communicative hand gestures not involving an object are observed. The authors conducted an fMRI experiment which required the participants to view, imitate, and produce both communicative gestures and object-directed hand actions. As per the results, both communicative hand gestures and object-directed hand actions activated the MNs in participants in a similar manner.

Mainieri et al. (2013) conducted a study into whether MNs behave in a similar manner during observation and execution of communicative gestures. The study included observation and execution of social, non-social, and meaningless gestures. The results highlighted that the gestures with communicative intentions activated the MNs while the gestures that lacked any communicative intention did not activate the MNs in the similar manner. Therefore, it can be inferred that MNs play a role in interpretation of hand gestures that represent a communicative intention. This finding can also be linked with the arguments presented by Ramachandran and Oberman (2006) who state that MNs are situated at the core of language development among humans.

Corballis (2010) presents the idea that language evolved from manual gestures. Initially, language merely existed in a symbolic form which was further enhanced over a long period of time. Eventually, facial and vocal elements were incorporated and grammar was introduced. The author argues that MNs played the core role in development of language as they helped individuals in understanding and imitating communicative gestures.

Schippers and Keysers (2011) explain the role of MNs in active guessing and passive observing of gestures. According to the authors, MNs act as a feedback control system which assists in the flow of information during observation of gestures. When an individual actively attempts to interpret the meaning of a communicative gesture, the MNs become activated. On the other hand, when an individual passively observes a gesture without attempting to interpret its meaning or background intention, the MNs remains relatively passive.

Increasing numbers of researchers have revealed evidence for the involvement of MNs in language perception (Rizzolatti, & Arbib, 1998; Arbib, 2008), as well as comprehension of motor actions (Di Pellegrino et al., 1992; Schroeder et al., 2008). *'The association of perception to action theory'* (Rizzolatti & Craighero, 2004) is one of the pieces of evidence for direct matching of the low-level motor movement theory, which is represented by the mirror neuron mechanisms.

The exceptional view of one theory of language acquisition, 'motor theory of speech

perception ’relies heavily on observation of the articulatory gestures of the speaker (e.g., movements of the tongue, lips, and mouth), rather than the acoustic cues of speech sounds. In order to acquaint oneself with spoken language, articulatory actions have to be represented in the listener ś (agent ś) brain, meaning that the regions which are associated with speech production would also become activated when the listener observes articulatory gestures.

In fact, the recent discovery of mirror neurons has granted further support to the idea of the involvement of the motor system in auditory speech perception (Wan et al., 2010). It seems that speech perception is basically a multi-modal experience whereby the development of language characteristically must occur in the presence of facial gestures and manual gesticulations (Arbib, 2005). Thus, action understanding and mental simulations of sensory-motor structures are essential for language comprehension (Gallese, 2005), and are thought to underlie the experiences of other people which are believed to be crucial for effective communication and social interaction.

Hauk and colleagues (2004) conducted an fMRI study with normally developing individuals, and validated the involvement of the motor areas in speech perception. Their neuroimaging study demonstrated that when an individual reads a sentence that includes motor words (e.g. head, leg or hand), their related regions in the sensorimotor cortex become activated as if that action had been executed. Wilson and colleagues (2004) similarly found that motor regions of speech production become activated when people are listening to speech sounds.

Kilner and Friston (2014) state that one view of the function of MNs that is commonly accepted is their ability to transform visual information into meaningful knowledge therefore giving rise to the ability to understand non-verbal communication. While this view emphasises the role of MNs in enhancing understanding of communicative actions, other views describe the significance of MNs in performing and understanding transitive and object-directed actions. Researchers have used MNs as their focal point to explain evolution and emergence of intellect among humans. Ramachandran and Oberman (2006) emphasise the significance of discovery of MNs by stating it to be the

most important finding of that decade. MNs show increased neural activity when hiding the final part of a goal-directed reaching action and in response to acoustic stimuli that is produced by the action (Kohler et al., 2002), which might suggest MNs involvement in goal prediction (Umiltà et al., 2001).

In addition, the MNs system seems to activate in similar ways for active action performance and passive observed actions by simulating the motor system; this finding suggests a role for MNs in action comprehension, imitation (Rizzolatti & Sinigaglia 2010), and emotion understanding (Keysers & Gazzola, 2006). It has been widely speculated that these neurons are the source behind functions like empathy, automatic imitation, language, and self-awareness (Perkins et al., 2010; Cannon et al., 2012; Tidemann, 2011). Some theories of MNs functions drawn from object-directed action may not be entirely applicable in the context of communicative actions, in particular, in theories that require object involvement, motor chain actions, or open-ends actions.

On the other hand, a plethora of data on MNs functions have been reported through brain lesions studies. Both classic and contemporary research has demonstrated the impact of lesions to various sections of the brain and performance on cognitive and motor tasks (Drewe, 1974; Wang et al., 2011; Westlake & Nagarajan, 2011). For example, Drewe (1974) conducted an early study demonstrating how the effect and type of brain lesion observed in a particular participant impacted on performance differently in a card sorting task. More recently, Arevalo et al. (2012) showed that the relationship between brain lesions and task performance can reveal critical information regarding the role of finite cognitive structures in human functioning. These findings lend support to the role of specific cognitive structures and the regulation of different task modalities.

Recent research (i.e., Arevalo et al., 2012) has explored the relationship between brain lesions and MNs in human. Studies have shown that lesions to these areas have produced changes in participants' ability to process effector-related stimuli, as well as action, execution and observation (Kemmerer & Gonzalez-Castillo, 2010; Arevalo et al., 2012). Additionally, relationships between MNs and object-related actions are still mostly speculative (Buxbaum, Kyle, & Menon, 2005).

Based on these lesion studies, it seems reasonable to assume that lesions to MNs areas in the brain would also result in cognitive domains impairment, for instance, poor performance on imitation and action understanding (Rizzolati, Cattaneo, Fabbri-Destro, & Rozzi 2014). Research has shown that lesions to motor neurons areas can interfere with automatic imitation capabilities (Heyes, 2010). These activities are designed to assess advanced motor processing (Rizzolati et al., 2014). Research has shown that lesions to motor neurons areas delay this process as well as the actual ability to interpret actions (Vivanti et al., 2011).

Rizzolatti and colleagues (2014) conducted a recent review of the cortical mechanisms underpinning actions regulated by MNs. According to these authors, it is now widely recognised that the motor system is not simply a manufacturer of movements, as was believed in previous decades, but a network of advanced cognitive processes. Identification of MNs and their relationship with goal-directed actions and action understanding has been beneficial in further understanding the neural mechanisms underlying human behaviour (Tranel, Kemmerer, Adolphis, Damasio, & Damasio, 2003; Rozzolatti et al., 2014).

Additionally, Urgesi, Candid, and Avenanti (2014) conducted a meta-analysis of lesion-symptom mapping studies in brain-injured patients, illustrating the role of MNs in perceiving and understanding the actions of others. Strong effects were demonstrated across neuropsychological studies for temporal-, parietal-, and frontal-lesion-related deficits in both visual perception and action understanding (Urgesi et al., 2014). It is suggested that MNs represent the key anatomical substrate governing these neuropsychological deficits (Urgesi et al., 2014).

Though understanding of MNs and their role in imitation and action understanding has progressed considerably in recent years, some limitations related to both technology and research design are evident that potentially detract from the ability to draw causal conclusions about these physiological structures. Furthermore, the MNs theory has drawn criticism for both philosophical and practical reasons. One fundamental problem with research in MNs, which appears on other cognitive neuroscience research, is the predominant reliance on animal models for empirical research (Lindell & Kidd, 2011). Brain lesion studies in humans offer insight into the potential for MNs to regulate

specific tasks.

However, the use of such a self-selecting sample presents the possibility of bias and limits statistical power within studies that adopt such designs (Coolican, 2014). The neuroplasticity of the human brain produces adaptations in brain lesion patients that are difficult to identify and control for in experimental research (Kays, Hurley, & Tabler, 2012). Animal models have been used to overcome this design problem; nonetheless, it worth to note that anatomical, biological, and cognitive differences between animals and humans potentially limit the validity of such research (Lindell & Kidd, 2011).

Despite these limitations, there appears to be evidence (e.g., Garrison et al., 2010; Arevalo et al., 2012) to support the hypothesis that lesions to MNs in the brain lead to poor performance on imitation tasks. Brain lesion studies (e.g., Garrison et al., 2010; Arevalo et al., 2012) have demonstrated this effect directly, while animal studies and reviews (e.g., Bonini, Maranesi, Livi, Fogassi, & Rizzolatti, 2012) have suggested this effect indirectly. Research has also progressed in terms of the understanding of MNs functioning and how these mechanisms operate in the case of simple imitation tasks, or more advanced interpretation of actions (Vivanti et al., 2011). Mapping analyses have identified precise regions responsible for processing of both neutral and effector-related stimuli, further illustrating the role of MNs in imitation and action understanding (Arevalo et al., 2012).

1.4 Methods of measuring MNs activity

A variety of methods for measuring MNs in humans have been established through a series of brain imaging, eye-tracking experiments, and brain structure studies. The majority of these studies were interested in identifying the corresponding area of the monkey F5 in humans (Rizzolatti et al., 1996), establishing a correlation between MNs dysfunction and clinical diagnoses that feature social deficits (Oberman et al., 2005; Martineau et al., 2008), and investigating the abnormality in MNs' function and related

brain structures (Nordahl et al., 2007).

Some of the major brain imaging evidence has come from Transcranial Magnetic Stimulation (TMS) experiments that have shown a pattern of muscle facilitation during action execution and action observation (see section 1.2) (Fadiga et al., 1995). Further studies have used Magnoencephalogram (MEG) (Hari et al., 1998; Salmelin & Hari, 1994) and EEG (Bernier, Dawson, Webb & Murias, 2007; Cochin et al., 1998) techniques to record electrical brain activity, and have observed changes in (de)synchronization in the mu rhythm band oscillations (8 to 13 Hz).

In addition, the number of eye-tracking studies that have attempted to investigate MNs is rising. In the study of Flanagan and Johansson (2003), researchers hypothesised that when participants observe a block stacking task, the coordination between their eye-gaze and the actor ś hand is typical of the gaze-hand coordination that they would produce if executing the task themselves. The pattern of eye gaze that individuals show while performing a hand action are found to be similar to the pattern of gaze while observing a human hand action. In accordance with direct matching hypothesis, this implies that participants have implemented eye-motor programs driven by motor representations of observed action. This appears to support direct matching hypothesis of MNs.

1.5 EEG and event-related desynchronization ERD

Neurons are charged by membrane transport proteins, which pump ions through their membranes. Excitatory ́action ’and inhibitory ́resting ’postsynaptic potential occurs. Ions of typical charge then pushed their neighbours in wavy movements. When these waves of ions reach the electrodes placed on the scalp, they either push or pull electrons on the electrodes and the outcome of this difference will determine the size of potential. This potential is measured by a voltmeter and is identified by EEG oscillations.

The healthy brain produces oscillations that can be recorded non-invasively from the scalp through EEG and MEG techniques **(**Kilner et al., 2003). EEG was primarily conducted on non-human species (Caton, 1875), and was first recorded in humans by

Hans Berger (1929), after a series of initial experiments on animals (e.g. measuring electrical activity in dogs by Pravdich-Neminsky in 1913; Swartz & Goldensohn, 1998). This critical move in the history of neurology led to the first evolutional attempt by Fisher and Lowenback (1934) to detect epileptic spikes, which was followed by subsequent studies that contributed to the clinical neurophysiology field. Some of this influential work will be discussed here.

EEG records oscillatory signals, which comprise a large number of different effects that can be categorized into three main types: spontaneous rhythms, induced effects, and evoked responses. Spontaneous rhythms generally refer to electrical potentials that are generated by the cerebral cortex nerve cells in response to stimulations. They are measured in microvolts (μV), within various frequencies of interest, through electrode sensors that are arranged across the participant's scalp. The voltage difference that occurs between two electrodes (i.e. each active electrode compared to a reference electrode) is identified and amplified, and then transmitted as a voltage potential to be digitised and displayed on a computer.

The method of electrode placement is determined by the clinical or research purposes (Martin, 2006), and there are two main electrode placement methods: the 10-10 international system that comprises 64 electrodes, and the 10-20 international system that includes 21 electrodes (Vespa, Nenov, & Nuwer, 1999). '10-10' and '10-20' refer to the distance between the electrode according to their placement on the cerebral cortex. Thus, the distance is either 10% or 20% of the total front-back or right-left distance of the skull. Each electrode has a unique letter referenced to the lobe, and a number to refer to the hemisphere (see figures 1.2 & 1.3).

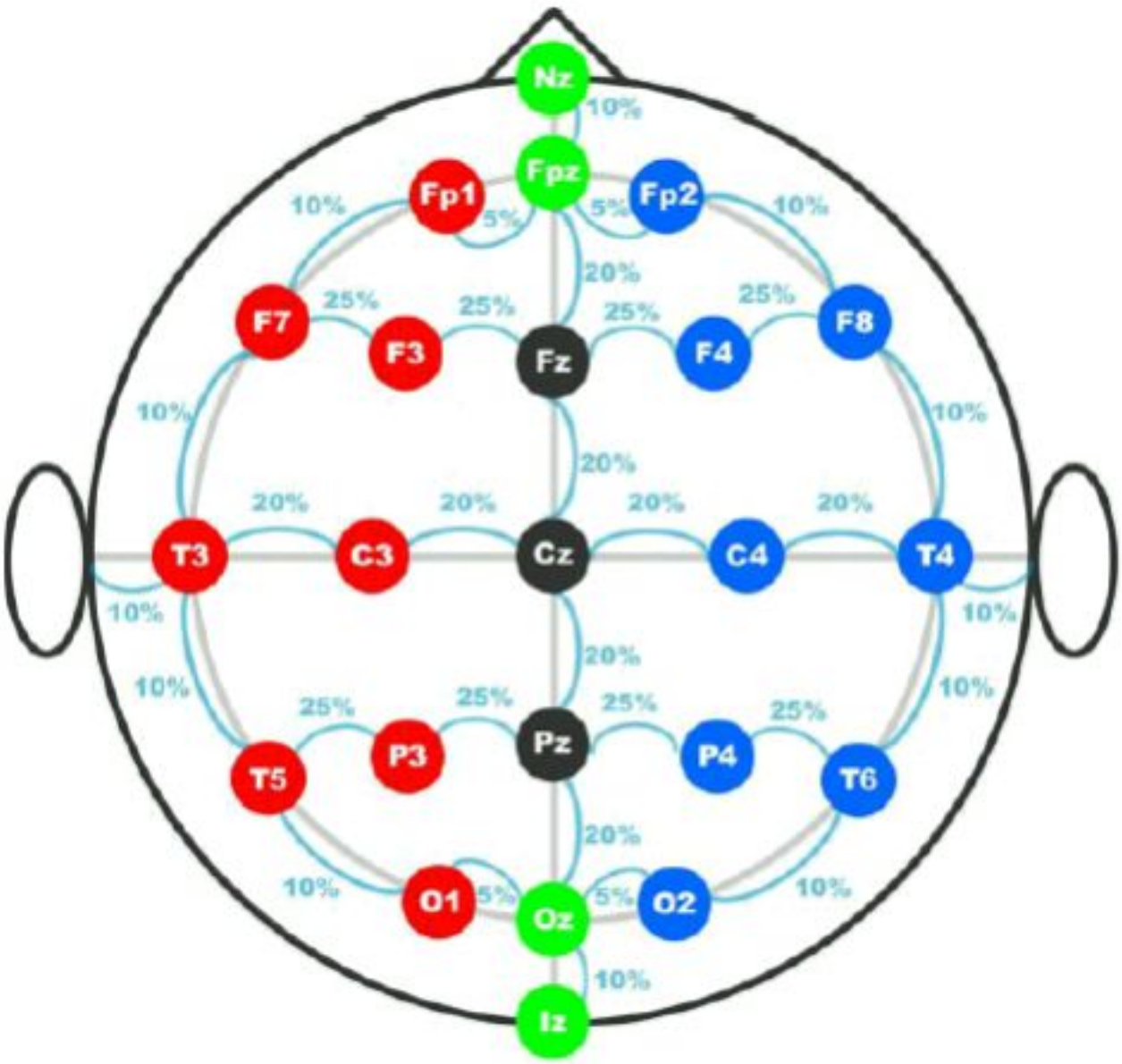


Figure 2 The figure shows the view from above the head: even numbers (2, 4, 6, and 8) refer to electrodes positioned on the right hemisphere, while odd numbers (1, 3, 5, and 7) refer to electrodes positioned on the left hemisphere (Trans Cranial Technologies, 20

Electrode	Lobe
F	Frontal
T	Temporal
C	Central *
P	Parietal
O	Occipital

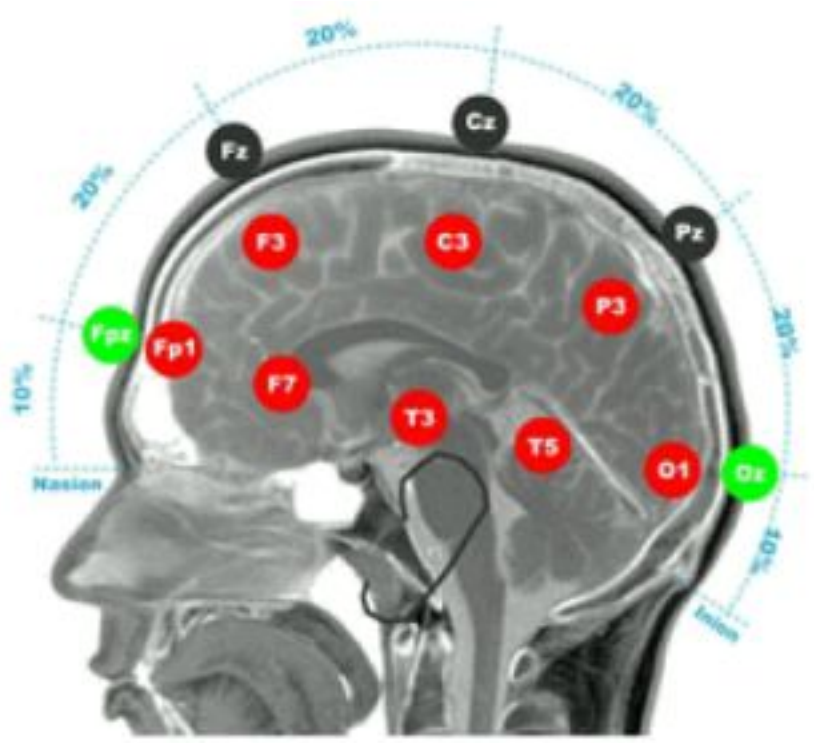


Figure 3 The table on the left displays letters and their identified lobes. The figure on the right shows electrode placement from the left profile: odd numbers (1, 3, 5, and 7) refer to electrodes positioned on the left hemisphere (Trans Cranial Technologies, 2

The experiments in this thesis used the 10-20 electrode placement system. EEG is a functional measuring approach, which means that its rhythmic activity mirrors the processing of sensory information, which is detected in a number of frequency bands. These frequency bands are categorised into five brain rhythms based on their frequency: Delta (3.5 Hz and lower), Theta (3 –7.5 Hz), Alpha (8 –12 Hz), Beta (12 –30 Hz) and Gama (30 Hz or higher) (see figure xx) (Niedermeyer, & Da Silva, 2004).

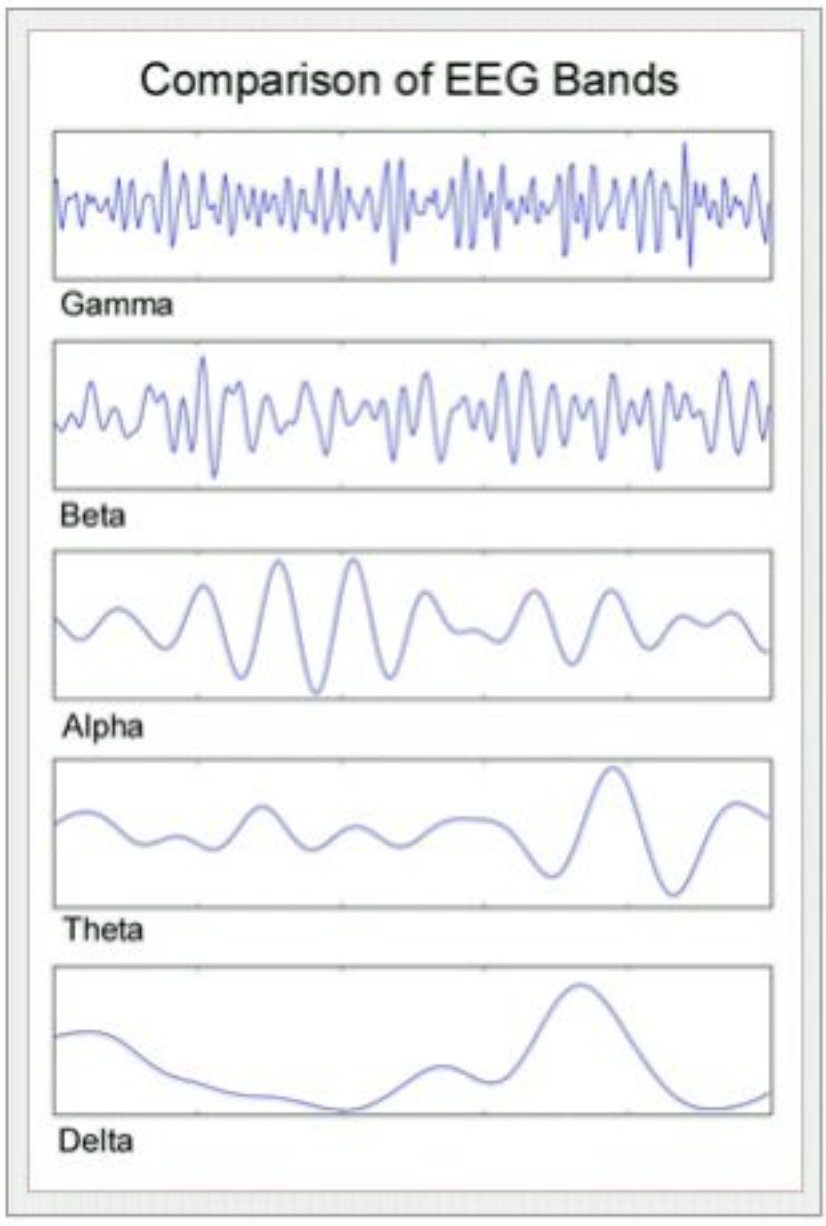


Figure 4 Comparisons of EEG oscillations which are determined by their frequencies (Kent, 2010).

Each frequency band is thought to correspond to a state of brain functioning (Steriade, Contreras, Amzica, & Timofeev, 1996) and neural network. For instance, the rhythmic activity in the alpha frequency band principally reflects relaxation and an attentive cognitive state, and predominantly increases when the eyes are closed. In addition, alpha activity is assumed to represent the thalamo-cortical interaction network (Steriade et al., 1996). Thus, besides being the key clinical method for seizure detection and monitoring (Smith, 2005), EEG remains the optimal experimental approach in brain

computer interface (BCI) research (Hochberg et al., 2006), neuroscience and cognitive psychology investigations, due to its high temporal sensitivity and tight correlations with ongoing neural activity.

When presenting a stimulus to the individual, this can induce modulations of neural activity in various frequency bands of ongoing rhythmic activity. This event-related change may consist of increases and decreases in power within a frequency band, and is often referred to as event-related synchronization (ERS) and event-related desynchronization (ERD) (Pfurtscheller, 1992; Pfurtscheller, 1977). Induced effects are time-locked to the event onset, but not phase-locked, thus the changes can be detected by frequency analysis, and not by simple linear methods.

The basic methodology for calculating induced effects comprises specific steps: all event -related trials are filtered; power is obtained and averaged across trials and participants to yield a grand average. To obtain the ERD/ ERS values, activity within this period of stimulation is compared to a ‘baseline’ reference period, and the power within specific frequency bands of interest is calculated (Pfurtscheller & Lopes da Silva, 1999). This calculation for ERD is summarised below, where ‘A’ refers to the stimulus event period, and ‘R’ refers to the reference period.

$$ERD\% = ((R - A) / R) * 100$$

Activity within the alpha range over sensorimotor areas is known to desynchronize during motor activity (Gastaut, 1952), and when imagining an action (Pfurtscheller et al., 2005). Further, Pfurtscheller and Berghold (1989) found that voluntary hand movements result in a desynchronization in the high alpha range (10-12Hz) and lower beta range (13-20Hz), which localizes close to the sensorimotor region, as well as synchronization of occipital alpha rhythms. Pfurtscheller proposed that the increased cellular excitability that occurs in the thalamo-cortical network leads to desynchronized EEG, and thus, this induced effect is reflective of activated cortical regions that are critical in processing sensory or cognitive information or producing motor behaviour.

The theta frequency band, along with alpha, has attracted attention within research on induced effects, in particular research relating to learning and working memory (Klimesch, 1999). This work was initiated by studies on humans in which the hippocampus, the key area of learning and memory, was found to mediate theta activity (Tesche & Karhu, 2000). In this thesis, we will initially focus our analyses on changes in the alpha (8-12Hz) and low beta (13-20Hz) bands, using ERD over sensorimotor areas to detect variations in MN activation. The higher bandwidth of theta (5.5-7.5Hz) will be additionally employed during the experiments in Chapters 4 and 5, and will be analysed alongside alpha and low beta data.

1.6 Recording an index of MNs using EEG

Since the discovery of so-called rolandic *"en arceau"* rhythm by Gastaut and Bert (1954), which is commonly referred to as mu rhythm, numerous studies have recorded physiological data to investigate the changes in brain wave patterns between frequencies of 8-13 Hz, to detect the activity of MNs (e.g., Pineda et al., 2000; Altschuler et al., 2000; McFarland et al., 2000; Muthukumaraswamy et al., 2004; Oberman et al., 2007; Pineda & Hecht, 2009). The attenuated power of the mu rhythm indicates increased neural activity (Kuhlman, 1978).

This attenuation has been reported to be an index of MNs activity in human adults (Pineda, 2005; Oberman et al., 2007; Perry & Bentin, 2009), and infants (van Elk et al., 2008). Studies that have recorded mu rhythm activity have substantially contributed to MNs literature. Discovering the characteristics of MNs granted explanations for various cognitive phenomena, in showing increased modulation when an agent is observing a target-directed action compared to when they are observing a non-target-directed action (Muthukumaraswamy & Johnson, 2004). Mu band activity has also revealed MNs' sensitivity to the degree of familiarity of the agent (Oberman et al., 2008), and the observer's experience with observed action (Marshall et al., 2009).

Further studies by Puzzo et al. (2010) have speculated that changes in low-beta band (12 –20 Hz) also reflect the neural activity of MNs. Indeed, one study reported that low beta is attenuated when an action is voluntarily executed, imagined or observed

(Babiloni et al., 2002). Therefore, a subsequent study has recorded mu and beta band EEG activity as an index of MNs, which revealed the role of a social hormone in allocating cortical resources that were modulated by MN activity (Perry et al., 2010).

1.7 Developing EEG recordings in childhood

It has been established early that the EEG frequencies change with age from infancy to at least 16 years (Somsen et al., 1997; Niedermeyer, 1997). The typical pattern of changes represents an increase in amplitude of the higher frequencies, and a decrease in amplitude of the lower frequencies (John et al., 1980). The appearance of 7 Hz rhythm at central sites was initially observed at around 4 months of age. The mean frequency of 'central alpha' notably remains around 7 Hz over the first year of life, but raises to 8 Hz by 18 months of age, then to 9 Hz by 4 years of age, and plateaus at around 10 Hz in mid-adolescence (Smith, 1941).

Further, in a longitudinal sample from 4 to 17 years, Benninger et al. (1984) found an increase in alpha between 7.5 and 12.5 Hz power, and a decrease in theta between 3.5 and 7.5 Hz power over central electrode sites. A similar longitudinal study assessed a sample of 29 participants, from early infancy to early childhood and examined the spectral analysis and peak frequency at central sites between 3 and 12 Hz. Results showed that peak frequency at central electrode sites increased from 8 Hz at 2 years to 9 Hz at 4 years of age, with a small number of children showing a dominant frequency of 10 Hz at 4 years of age (Marshal et al., 2002).

These speculations seem qualitatively consistent with findings from a recent study of the developmental course of mu suppression across age span, in which mu rhythm suppression appeared to increase with age, suggesting that mu frequency could be more sensitive to action observation in older childhood and adulthood relative to that in younger children (Oberman et al., 2012).

A wealth of research has focused on the recognition of biological motion in infancy and childhood (Hirai & Hiraki, 2005; Carter & Pelphrey, 2006; Reid et al., 2008; Kröger et al., 2013). In contrast, relatively small numbers of studies have speculated on the

similarities between the patterns of neural activity during the execution and the observation of motor gestures (Lepage & Théoret, 2007). Evidence of significant mu rhythm modulation by MN activity in infants has been shown in an action observation paradigm (van Elk et al., 2008), and more recently in an action observation and execution model in a sample of 9 month old infants (Southgate et al., 2009).

Lepage and Théoret (2006) examined the mu rhythm amplitude over central sites in a group of 15 children, aged between 4 and 11 years, during a task that involved execution and observation of hand grasping actions. The pattern of mu rhythm suppression during action execution was similarly present during observation and execution of the same action, which suggests the presence of an execution-observation matching system in children. In addition, two theta ranges were examined in this study, (3.5 –5.5Hz and 5.5 –7.5Hz), to investigate the neural activity during execution and observing conditions in young children. Results showed that theta modulations while executing an action did not show qualitatively different neural activity compared to three observing conditions.

1.8 Effect of stimulus presentation modality

There has been a strong predisposition to suggest that watching videos or television increases motivation in children with ASD (Shane & Albert, 2008), and assists them to focus on the relevant properties of incoming information by restricting their field of vision to the targets, and ensuring a minimal degree of social interaction (Corbett 2003). Elsewhere, Corbett and Abdullah (2005) reemphasised what has been suggested by Dorwick and associates (1991), that video modelling provides the essential elements (i.e. attention, retention, motivation and production) for ‘observational learning’ to occur.

This proposal was based on a number of significant findings that were revealed by video modelling interventions. One such study was conducted by Charlop-Christy et al. (2000), who investigated how children with ASD acquire new skills; they found that the rapidity of skills acquisition, such as greetings, imitation and self-help, was evidently faster when children learned from video stimuli, compared to live stimuli. Subsequent work by Ayres and Langone (2005) revealed the effectiveness of videos as a teaching method for some

behaviours in a recent review. Researchers measured the total time children spent attending to visual stimuli, and found that the visual attention of children with ASD can be enhanced to a greater degree using video modelling compared to live modelling (Cardon & Azuma, 2012).

1.9 Anatomical and functional abnormalities of MNs in ASD: a debatable view

Though key lines of published empirical evidence have significantly contributed to the 'broken mirror neurons theory' in ASD, evidence opposing this theory also exists. It was discussed earlier that the anatomical co-localisation of areas related to MNs and the brain regions involved in social cognition suggest that, in ASD, abnormality in MNs' function and related brain structures (including the inferior frontal gyrus (IFG) and the right superior parietal lobule (SPL)) would show equivalent defects in social cognition.

The development of the connections for the social brain can be abnormal in ASD (Johnson et al., 2005) such that ASD participants have a different pattern of neural activity in social brain areas to typical participants during a task involving fearful expression perception (Ashwin et al., 2007). ASD adults also show little activation of the IFG and premotor cortex when observing fear, suggesting a link between these locations and an ability to appreciate emotion in others (Grezes et al., 2009). Normal controls exhibit strong modulations of the social brain and MNs in emotional conditions but ASD participants do not show the normal increased activation (Hadjikhani, 2010).

Researchers investigated the activity of the pars opercularis of the IFG in typically developing (TD) children and children with ASD while they were observing and imitating facial expressions. Their data revealed two major findings: firstly, no statistical difference was found between the groups in their ability to imitate facial expression. However, the pars opercularis failed to show activation during imitation in the ASD group, while the control group showed bilateral activation even in the observation condition; the level of activation that was established by the control children was significantly higher than the children with ASD. Secondly, neural activity in the pars opercularis, while imitating facial expression, was negatively correlated with the

severity of individuals 'assessment scores on tests in the social domain (Dapretto et al., 2006).

In a subsequent study, individuals with ASD showed a significantly reduced volume of right pars opercularis of IFG, a site of structural abnormalities in ASD (Nordahl et al., 2007), compared to a control group (Yamasaki et al., 2010).The pars opercularis and sub -regions of the IFG, which mainly consists of Brodmann area 44, could principally be involved in the human MN system (Molnar-Szakacs et al., 2005). This suggestion is supported by the association found between the reduced size of the pars opercularis volume in the ASD group and social communication and reciprocity deficits (Yamasaki et al., 2010).

Aberrant cortical thickness was reported in ASD individuals (Hadjikhani et al., 2006), particularly thinning in a number of brain areas, which included the IPL, STS, IFG, prefrontal cortex, superior parietal lobule, and inferior temporal gyrus. Apparently, these areas involved the MNs network. In a recent study, though, there was no group difference in surface area (Wallace et al., 2013), The FreeSurfer image analysis method showed that the gyrification increases in the individual with ASD relative to the adolescent and young adult matching control.

The superior temporal sulcus (STS) and amygdala are part of the broader network of face-processing modules (Hadjikhani et al., 2007) that enable the individual to recognise others and evaluate their mental state (Pelphrey et al., 2011) and these have a strong overlap with the MNs in terms of the orbitofrontal cortex and STS (Pelphrey et al., 2011). Individuals with ASD show little difference in recruitment of MNs areas during emotional state tasks, although a deficiency in frontal areas with a neurofunctional segregation within the medial prefrontal cortex is demonstrated (Schulte-Rüther et al., 2011). Adult high-functioning ASD participants, compared to a matched control group, have subtle deficits observable in the area of the social brain. The areas involved were MNs (IFC), (STS) and the amygdala through modulation of face processing (Hadjikhani et al., 2006; Hadjikhani et al., 2007).

Altered connectivity exists between MNs regions, such as inferior frontal areas, and

emotion processing regions including the fusiform face area in ASD participants (Kleinhans et al., 2008), with Sun et al. (2012) showing altered activity in MNs regions and increased MNs connectivity which has also been found to be correlated with symptomatology (Fishman et al., 2014). Connectivity changes have been reviewed in more detail by (Kana et al., 2011a) who later describes the multidimensional nature of the MN system and how coordination within this system affects individuals with ASD (Kana et al., 2011b).

Brain activity was measured during the observation of facial expressions in a group of adults with ASD and typically matched control subjects (Bastiaansen et al., 2011), and although their findings were not consistent with the findings of three previous investigations with children with ASD in establishing hypoactivity of the IFG (Dapretto et al., 2006; Bookheimer et al., 2008; Uddin et al., 2008), the study was the first to demonstrate age-related increased activity in the IFG during the perception of facial expressions in autism, and this was associated with improved social functioning. Bastiaansen and colleagues argued further that their speculation synchronised the results of Ashwin et al. (2007) and Pierce et al. (2004), in which there was no group difference - related to IFG - in adult and adolescent participants.

The second line of evidence mainly originates from studies that were concerned with imitation, and was primarily determined by the findings from studies that have made observations of adults ' ability to imitate hand actions which would be expected to stimulate MNs activity (Iacoboni, 1999), but which showed up problems with imitation in those with impaired MNs (Heilman et al., 1982). Similar difficulties are manifest in children with ASD whose ability to imitate has been found to be impaired (Williams et al., 2004).

In a further study (Williams et al., 2006), the overall activation of MNs areas in adults with ASD and matched control during a finger movement imitation task seemed to be reduced, though the activity was shown to engage IFG in the control adults in a previous study (Iacoboni et al., 1999). Greater ASD activity in the dorsal premotor and prefrontal areas is taken to mean that alternative strategies and processing areas are adopted by

ASD in place of MNs processing. This latter idea is consistent with the information available on the social brain, in that individuals with ASD require more conscious effort to decipher social situations which may explain the altered activity in the social brain (Ashwin et al., 2007).

ASD subjects experience difficulty in transforming their original intentions to motor intentions (Rizzolatti and Fabbri-Destro, 2010) due to aberrant MNs mechanisms that underlie this purpose. Relative to the matched control, some participants with ASD failed to activate Broca ś area during an orofacial imitation task, and that activation was further delayed and weaker (Nishitani et al., 2004).

A series of neuroimaging studies demonstrated a significant variation in the mu wave suppression, the primary band proposed to reflect the underlying MNs activity (Hari, 2006), between execution and observation of hand actions in individuals with ASD. The neural oscillation in the mu frequency band (8–13Hz) was diminished during observation of a hand action, relative to the equivalent response when executing an action in individuals with ASD (Oberman et al., 2005) and it was linked with poor imitation performance (Bernier et al., 2007).

Abnormalities in suppression were reflected through other EEG frequency bands, theta 1 (3–5.5Hz), theta 2 (5.5–7.5Hz) and alpha 1(7.5–10.5 Hz), during observation conditions (Martineau et al., 2008). Using the post-movement beta rebound (PMBR) method, which focuses on power increase in the beta frequency band after observation and execution of movements PMBR was exclusively reduced during observation of individuals with ASD compared to the matched control. Reduction was evident in cortical regions within the MNs, the sensorimotor area, premotor cortex, and superior temporal gyrus (Honaga et al., 2010).

Abnormal modulation of the motor cortex during observation of finger movements was evident through TMS studies, as it was significantly reduced in the ASD group compared to the matched control (Théoret et al., 2005), and it was negatively associated with MNs activity (Enticott et al., 2012). Significant abnormal function of extended MNs regions

(pMCC/ SMA) can be observed in adults with ASD during tasks involving understanding hand actions.

The behavioural data on action understanding demonstrated variation in error rates and reaction times between ASD and typically matched groups in an action comprehension task (Marsh and Hamilton, 2011). Compared to TD children, failure of predictive muscle activation was demonstrated in children with ASD in which the muscles responsible for the action's final goal increase their activity as soon as the action starts (Cattaneo et al., 2007).

The first study seeking to link MNs deficiency and ASD using functional imaging (MEG) reported no significant results, although follow up studies were able to demonstrate weak links (Hadjikhani 2010). Incongruously, a young adult with ASD demonstrates hyperactivation of the pars opercularis during observation of human movement (Ecker et al., 2010). A relatively early review of MNs and ASD indicated that MNs dysfunction might, at best, be only part of the explanation due to the heterogeneity of ASD (Williams et al., 2001); this is something which has been echoed more recently (Schroeder et al., 2010). Critics of the broken MNs hypothesis point out that experimental tasks may not fully represent mirror activities and therefore may not adequately test motor functions (Agnew et al., 2007) with such methodological problems being highlighted elsewhere (Vanderwert et al., 2013).

Many of the studies referred to above utilise male participants, yet it is noted that the recruitment of MNs varies between sexes, with females being recruited more in empathic face-to-face interactions (Cheng et al., 2009). Other conclusive results showing that impaired imitation and mindreading were not secondary to the MNs mechanism of action execution/ observation was driven by a small groups of participants as the motor cortex was activated in both groups equally (Avikainen et al., 1999). Likewise, high functioning ASD are used to aid procedural issues such as with fMRI but this may affect the generalizability of results (Fishman et al., 2014).

With regard to the imitation literature, the absence of any imitation deficit has been

demonstrated in children with ASD, along with a lack of global MNs deficit (Hamilton et al., 2007) and no observable differences in mu activity and suppression (Raymaekers et al., 2009). There may be an age related link between MNs deficits and ASD as indicated earlier (Bastiaansen et al., 2011), although the converse has been shown in some research (Hamilton et al., 2007), casting doubt on the veracity of the research.

A modulation deficit rather than MNs deficit could occur in ASD (Hamilton, 2013). The key criticism of the evidence for the broken MNs hypothesis relates to the choice of tasks to demonstrate function of the MNs system, as these may only reflect possible function (Hamilton, 2013) or be indirect measures (Southgate and Hamilton, 2008). Confining the cause of ASD to a localised region within the brain is too simplistic and fails to take into account other causes (Southgate and Hamilton, 2008). This narrow focus of investigation has also been noted by (Hickok, 2009) with the heterogeneity of ASD and its biological basis an issue (Ecker et al., 2010), since multiple neural systems are affected (Anagnostou and Taylor, 2011). Evidence exists for a wider action sequence impairment (Zalla et al., 2010), rather than MNs dysfunction directly (Fabbri-Destro et al., 2009).

Alternate explanations for ASD cast doubt on the impaired MNs hypothesis, in that they provide evidence which either discounts the role of MNs directly or negates the need to consider them. In a voxel-based morphometry study, which investigates focal differences in brain anatomy, adults with ASD revealed reduction in grey-matter volume in medial temporal, fusiform and cerebellar regions, and in white matter of the brainstem and cerebellar regions, but not in IFG or IPL (Toal et al., 2010).

Young adult ASD subjects show no impairment in discriminating human face identity or emotion - in itself contrary to the idea that all individuals with ASD have impairments in facial recognition (Behrmann et al., 2006, Boucher and Lewis, 1992), and trustworthiness ratings were akin to patients with amygdala damage, suggesting amygdala dysfunction could underlie some ASD symptoms (Adolphs et al., 2001; Schultz, 2005). Amygdala activity is greater in ASD during imitation (Williams et al., 2006) although there is a differential effect depending on ASD functional level (Ashwin et al.,

2007). Frontal lobe dysfunction in ASD is demonstrated through specific growth abnormalities in the PFC related to neuron numbers (Courchesne and Pierce, 2005), although this change in neuronal numbers doesn't necessarily correlate with ASD symptomatology (Uppal and Hof, 2013).

1.10 How the deficit in MNs relates to deficits in social competence

According to 'Theory Theory' which derives from Adam Morton (1980), attributing mental states to others requires theoretical reasoning involving causal laws. In contrast, 'Simulation Theory' suggests that, in order for simulators to resonate the mental state of others, they must use their own mental mechanism by incorporating 'pretend states' to replicate and mimic the agent's mental states (Gordon, 2005). Thus, whereas mimicking the mental activity of an agent seems to be a major principle in 'Simulation Theory,' it has no role in mindreading according to 'Theory Theory.'

This core difference leads to the proposition that if both simulator and agent appear to experience matching mental activity, mindreading would correspond with 'Simulation Theory' (Gallese & Goldman, 1998). MN theory seems to underlie the process of generated mental states in the observer, similar to that of the agent, by mirroring his/her emotions, actions and mental states. As such, the capacity to understand others' mental states and actions seems qualitatively equivalent to the capacity of the observer to simulate the mental states of others (Oberman & Ramachandran, 2007).

The development of automatic embodied simulation seems to emerge from infancy towards the end of the first year of life (Wan et al., 2010), something which is reflected in the synchronicity of the facial and voice interactions between a mother and her child (Reddy et al., 1997). The ability to attribute mental states to others seems to emerge from the second year onwards (Shultz & Cloghesy, 1981; MacNamara et al., 1976). The critical function of this embodiment mechanism lies in its vital role in social development, reciprocity and communication. Any failure in MNs in conceiving the use of mental state concepts, predicting and retrodicting an agent's action would manifest in a similar profile of symptoms to individuals with ASD (Baron-Cohen et al., 1985; Colle et al., 2007; Dapretto & Iacoboni, 2006). Specifically, impaired MN activity would be

represented by poor communication, a lack of understanding of other's intentions and actions, poor imitation ability and delayed or impaired language acquisition (Welsh et al., 2009).

1.11 Effect of familiarity and similarity

Familiarity is perceived in terms of the actual frequency with which individuals are exposed to certain people, while similarity refers to the match between two individuals, which can be found in values, demographics and attitudes (Adams-Webber, 1977). Familiarity cannot be altered by similarity, since familiarity is determined by prior exposure. However, it is plausible, in some circumstances, that the perceived familiarity of others is likely to be driven by their similarity to ourselves. For instance, it has been shown that we tend to feel attracted towards people who are similar to us; feelings of attraction could result in an enhancement of the perceived familiarity of others, leading to overestimations of the frequency of exposure to a given individual (Matlin & Stang, 1978).

1.12 Effect of familiarity

There is large number of neuroimaging studies that have suggested variations in the neural activation of MNs depending on the nature of the observed action (Cattaneo & Rizzolatti, 2009). For instance, actions that do not belong to the observer's known motor repertoire are unlikely to trigger MNs (e.g. barking) (Buccino et al., 2004). A similar pattern is seen when observing biologically impossible actions (e.g. flying), as MNs show less activation than when observing achievable actions (Stevens et al., 2000).

The observer's experience with observed motor actions, however, plays another major role in affecting the amount of neural activation. Marshall et al. (2009) provided support for this view by showing that EEG desynchronization over central sites increased after exposing healthy individuals to stimuli that they had already experienced during a brief training period, compared to the equivalent response to novel actions. This finding is consistent with other work (e.g. Calvo-Merino et al., 2005) in validating the role of the observer's motor expertise in MNs modulation; data show that expert ballet dancers demonstrate greater MNs activation when watching ballet moves than when watching

capoeira moves. The opposite pattern was found for capoeira dancers. In addition, the data reported no difference between the two types of action for 'control' participants who did not have any specific motor expertise.

These findings relating to action familiarity are in agreement with those of Wolff and Barlow (1979) and Oberman et al. (2008) which showed that familiarity of the actor performing a given action was found to modulate the neural suppression of both TD and ASD children. In particular, both groups of participants illustrated greatest suppression to the stimuli that elicited the greatest familiarity - 'observing their own hand.' The second greatest level of suppression was seen when participants were 'observing their parent's hand,' and the least suppression was observed when 'observing a stranger's hand.' These findings clearly distinguish how familiar action reasoning operates in relation to the degree of the actor familiarity between the two groups. These effects seem to match the proposal about a familiar bias put forward by Sai (2005) who showed that, since birth, children show a preference for looking at their mother's face compared to a stranger, which reflects the fact that the majority of the child's time is spent in their parent's presence. Thus, the effect of early exposure and experience could result in children showing better simulations of their parent compared to a stranger. This view is supported by the argument that people tend to sympathize more with known, and in-group people, compared to unknown and out-group people (Hornstein, 1976).

In this thesis, across four experiments, we will examine the effect of a number of culturally familiar communicative gestures, and also their interactive effect with person familiarity, age similarity and ethnic similarity. In chapter 6, apart from replicating the findings of previous works, we will infer how our design allowed us to investigate the effect of action familiarity and person familiarity independently.

1.13 Effect of ethnic similarity

Losin et al. (2010) highlight the importance of researchers defining what is meant by 'culture' and not confusing it with 'ethnicity.' 'Culture' refers to shared social experience including social practices, values, geography, religion and language (Chiao & Ambady, 2007), whereas the term 'ethnicity' refers to an inherited racial background (Chiao &

Ambady, 2007; Losin et al., 2010). It became apparent during this review that many authors had not defined culture, but were really referring to 'ethnicity' – more specifically, 'race' (Bacal, 1991).

Same ethnicity effect, or 'in-group bias', rationally, would require reasonable ability to distinguish this particular ethnicity from other ethnicities. This seems in agreement with recent findings that have shown that 3 and 6 month old infants have the capability to distinguish between individuals from the same ethnic group and individuals from different ethnicities (Katz & Downey, 2002; Sangrigoli & DeSchonen, 2004). Subsequent evidence has been demonstrated by Kelly et al. (2005) and Bar-Haim et al. (2006) who validated the ability of three month old infants to distinguish same and different ethnic members.

In this thesis, in Chapter 5, we will be investigating the effect of ethnic-person similarity and its interaction with action familiarity, which will be measured by MNs activation and imitative performance in preschool TD and ASD children. This has never been investigated to our knowledge within the ASD population.

1.14 Effect of age similarity

Recent research has demonstrated support for the facilitatory effect of similar-aged peers in supporting academic achievement (Utley et al., 1997), behaviour change, and social skills (McConnell, 2002). For example, introducing peers in a therapeutic intervention has been shown to lead to critical improvements of the functional skills of their siblings with disabilities (Cash & Evans, 1975) and siblings with ASD (Colletti & Harris, 1977). It is further seen that improvement of play and level of activity correlates with the level of peers' involvement (Kern & Aldridge, 2006). Studies such as this notably endorse the influence of same-aged peer models (e.g., Peer-mediated instruction) which could offer the most potential as part of an intervention strategy to prompt the use of motor and social skills in children with ASD (Chan et al., 2009).

This tendency that children demonstrated towards observing peers mirrors the notion that not all presented models will be imitated equally; the observer, therefore, will be

more influenced by those who symbolise engaging qualities (Bandura, 1977). This is convergent with the proposal of Epstein (1983) in showing that the characteristics of an observed model (e.g., age, ethnic status) influence the degree to which social attitudes and behaviours will be produced by others. Indeed, research evidence has shown that upon perceiving others as potential social partners, children start to increasingly integrate their activities with unfamiliar peers and imitate their actions (Maudry & Nekula, 1939; Harlow, 1969), which allows them to elaborate on a social engagement with an unfamiliar 'peer,' compared to a familiar 'mother' (Eckerman et al., 1975). According to Meltzoff (1990), this synchronises the 'like-me' view, in which peer preference stems from childrens' ability to recognize others as being similar to the self, which in turn requires linking an observed action to the self to understand simulations of others' actions and mental states.

In this thesis, in Chapter 4, we will be investigating the effect of the similar aged person, and its interaction with action familiarity, which will be measured by MNs activation and imitative performance in preschool TD and ASD children. This, to our knowledge, has never been investigated with MNs methodology within the ASD population.

1.15 When do children develop awareness about similarities?

The argument inferring a 'similar preference,' relating to the sensitivity to similarity to oneself and other children, correlates with research showing the development of children's awareness of similarities. Seehagen and Herbert (2011) found that early in the second year of life, infants appear to develop awareness about similarities between themselves and others. Other researchers set the age of similarity awareness even earlier, at 9-months, based on when infants are able to detect individuals who are similar to themselves (Sanefuji et al., 2006). However, perceiving other people as similar or dissimilar to oneself primarily requires a plausible level of self-recognition; recognizing the self as a familiar perceptual stimulus seems to emerge during the first 6 months of life (Bahrick et al., 1996).

Nonetheless, assuming that a 'similar preference' is derived from physical similarity does not mean that the preference is limited to physical appearance, which suggests that

physical appearance could be just one component of similarity judgements. This observation is based on a study that was conducted by Sanefuji and colleagues (Sanefuji et al., 2008), which sought to control the effect of physical similarity and to measure the effect of other possible contributions, specifically relating to variability in 'locomotion'.

In their study, crawling and walking infants observed stimuli that depicted crawling and walking infants, presented in the form of point-light displays. Remarkably, results from the total looking time revealed that infants were better at detecting the point-light 'locomotion' that corresponded to their own current locomotion development. Indeed, this finding seems consistent with the assumption of Meltzoff (1990), since it suggests that physical movements might inform preference to similarity.

A question arises regarding how these findings might be explained in the light of other studies, in which the age of recruited models (children and adults) is dissimilar to the participants (infants). These behavioural studies have revealed an agreement that infants treat unfamiliar adults and unfamiliar children differently. Specifically, 'fear' behaviours were exhibited, but only as a reaction to adult strangers and not to child strangers (Lewis & Brooks 1974). Although the models used in both conditions were dissimilar in age to the infant participants (i.e. older age children vs. adults), the infants showed a more positive affect towards unfamiliar children. Thus, age similarity seems to minimize the effect of 'strangeness' which can elicit a fear response towards an unfamiliar person. It can then be anticipated that fear levels relate to some degree on 'size', as related research shows that fear behaviours are more evident towards an adult male compared to an adult female stranger (Benjamin, 1961). This suggests that the larger the physical status of a person, the more threat-provoking they are perceived to be. This is evidenced by the fact that the effect of gender was not significant when the heights of the male and female adult strangers were matched. Indeed, subsequent evidence lends further support to this proposal; Brooks and Lewis (1976) showed that an unknown child-sized adult also elicited negative affect in infants.

These findings first highlight the ability of infants to recognize, independent of the size, the configural features of children's versus adults' faces. This leads one to suggest that children employ the available knowledge about the self to evaluate others as 'like' or 'unlike'

unlike ’and react accordingly. In this regard, in the absence of a similar aged model, infants evaluate older children as ‘like ’me, and display positive behaviours toward them, and evaluate adults as ‘unlike .’ Accordingly, infants have been shown to reach towards approaching children, regardless of their similar or dissimilar gender (Greenberg et al., 1973).

1.16 Imitation

Imitation is a cognitive learning mechanism whereby an individual observes others ’ behaviours, recognizes the goals that underlie them, and reproduces them through an exact observed plan of movements (Tomasello, 1999). Animal and human behavioural literatures present a wide range of definitions for imitation concerning what can be considered as true imitation or other forms of social learning (Nehaniv & Dautenhahn, 2002). In human psychology literature, there has been further variance among researchers on the definition of imitation, particularly with regards to the degree to which it requires an aspect of novelty in some cases, or with no clear boundaries (Billard & Dautenhahn, 1999).

Classically, replicating an observed behaviour performed by another person is largely described as ‘imitation ,’ which narrows the chance of examining each type of ‘ replication ’and the context in which it was evoked (Hanna & Meltzoff, 1993; Meltzoff, 1995; Herbert, & Simcock, 2003). According to Byrne and Russon (1998) imitation, within the context of tools and object use, requires differentiation into three different forms of ‘copying :’ *enhancement*, *response facilitation*, and *emulation*. In contrast to object/ goal related mechanisms, children might learn and copy the action to reproduce the same observed plan of movements, without understanding the goal (Tomasello, Kruger, & Ratner 1993). This *mimicry* action is a commonly used term in nonhuman social learning studies, but it is rarely applied in corresponding human studies.

Gestures are ‘intransitive actions ’that comprise various categories: (i) meaningless, pantomime, or communicative gestures which are used in the absence of speech, (ii) co-speech gestures whereby the message is conveyed through shared speech and hand/ mouth articulations, and finally (iii) emblems which are culturally determinant

gestures (McNeill, 2000). Stefanini et al. (2007) proposed that in early language development, TD children tend to use gestures as lexical fillers, which have various functions until they are able to effectively acquire spoken words, when these ‘lexical filler’ gestures are gradually replaced with expressed semantic linguistic forms (Bates, 1979).

This proposal was recently supported by Botting et al. (2010), who showed that 4 to 7 year-old TD children recruited gestures as a compensatory communicative method. In that regard, a question may arise concerning whether imitating ‘communicative gestures’ differs from those of other intransitive gestures. Empirical data have led to postulation on connections between gesture imitation and language in TD children (Snow, 1989) and children with ASD (McDuffie et al., 2005). This link reflects the fact that some of the key language areas, Broca and Wernicke, overlap with the critical areas for imitation (Rizzolatti & Arbib, 1998). In addition, the motor area for speech seems to match the linguistic contents with motor movements.

Despite MNs’ evident role in action understanding as described in section 1.1 (see also Gallese & Goldman, 1998), research has shown that monkeys, who do show MNs activity for actions, are unable to imitate others’ actions (Visalberghi & Fragaszy, 1990), which raises a question about MNs’ role in imitation. In addition, the corresponding connection of MNs in both the sensory and motor areas raises many questions about their connectivity, in particular, how visual information is transmitted and translated into actions, specifically in the case of observing a novel action.

Studies in humans using research methods such as TMS, EEG and EMG, have revealed a mechanism whereby motor areas resonate during action observation (Cochin et al., 1998; Fadiga et al., 1995; Iacoboni et al., 1999), thereby, reproducing an observed action from one of the underlying mechanisms of imitative ability, a “resonance mechanism” (Gallese et al., 1996). Meltzoff and Prinz (2002) argue that this mechanism, which is arguably driven by the MNs, should produce a neural code to perception and execution. This mechanism with a common sensory motor representation is fundamental to behaviour commonly described as ‘imitation.’

Empirical evidence has shown that MNs are recruited during imitation of simple finger movements (Iacoboni et al. 1999), and in learning complex motor acts without practice (Buccino, et al. 2004). The same principle seems to appear through observational learning as an aid to motor skill acquisition, in which procedural knowledge is required. Research findings have demonstrated equivalent improvements in skills acquisition in individuals who observed a skilled model perform an action 60 times, and individuals who physically practiced that same action (Vogt, 1996a).

This view seems to be in line with the proposal of 'motor theory of speech perception', a theory of language acquisition (Wan et al., 2010) that was presented in section 1.13. In principle, 'motor theory of speech perception' argues that the observation of articulatory gestures, like the movements of the mouth and lips, is an essential part of speech perception. The observed articulatory gestures are mapped onto motor schemata, and thus associated speech production brain regions become activated (Liberman & Mattingly, 1985). In this capacity, it could be argued that there is reciprocal theoretical support between the 'motor theory of speech perception' and the MN theory, as well as other theories of language acquisition that correlate impaired speech perception with the absence of visual modalities (Dodd, 1977; Munhall et al., 2004).

In this context, Rizzolatti and Sinigaglia (2006) argue that functional MNs are a necessary condition for imitation, but not sufficient. An individual still requires a control system to govern the mirror mechanism, in which the detectable actions are properly reproduced as a performance. Without a control system, an individual would compulsively replicate every single possible motor action.

For this reason, the frontal lobe, which involves inhibition and executive function skills, is largely assumed to be involved in any actions in which the individual makes a decision about imitating or disregarding the action. Therefore, there seems to be a complex system underlying the capacity to imitate, and MNs have a role in decoding sensory-motor information into a shared code.

It has been established that children with ASD have difficulty with imitation, which becomes evident from a young age (Charman et al. 1997; Perra et al., 2008; Vanvuchelen

et al., 2007). Moreover, children who are found to be poor imitators are more likely to manifest severe symptoms of ASD compared to children who are good imitators (Rogers et al., 2003). However, a clear variability has been revealed in the empirical data concerning the explicit performance ability and related brain activation.

For example, Bernier et al. (2007) showed that adults with ASD exhibit significantly poorer performance in all domains of imitation ability compared to control adults. Critically, in subsequent EEG investigation, Bernier and colleagues found no significant difference between mu suppression for imitation conditions of children with ASD and a control group of TD children (Bernier et al., 2013). In another contradictory example, Press et al. (2010) demonstrated intact automatic imitation of emotional facial actions in adults with and without ASD, whereas an EMG study by McIntosh et al. (2006) showed that adults with ASD demonstrated less expression-compatible muscular activation compared with TD adults.

In their study, they examined automatic and voluntary mimicry of emotional facial expression in adolescents and adults with ASD, with matched typical participants. Participants were presented with pictures of both happy and angry facial expressions and during that time, the muscle activity over their cheek and brow muscles was monitored with electromyography (EMG). Results revealed that the ASD group did not automatically mimic facial expressions, while the control group did. As for voluntary mimicry, both groups demonstrated successful mimicry. This suggests that the basic automatic social-emotional process is impaired in ASD.

There are three vital perspectives to consider when inferring reasons for the conflicting data from studies that have analysed explicit imitation performance versus implicit EEG and EMG. The first point was raised by Hamilton et al. (2007) in which both the category and the property of the imitation behaviour determine the involved cognitive and neural systems. This argument will be extensively discussed in the General Discussion for Chapter 6 in relation to our findings.

The second speculation was proposed by Bernier et al. (2013) who argued that ‘imitation’ is not clinically a diagnostic criterion for ASD, and therefore a great deal of

variability in performance occurs among individuals with ASD. The third argument that has been proposed by Jones and Klin (2009) is built on the inconsistency between sample characterizations (e.g. age range) which will be linked to our findings in Chapter 6.

The empirical work in this thesis (Chapters 2, 3, 4, and 5) inspects imitation in children with and without ASD. Several factors motivated the decision to examine imitation performance. Firstly, we sought to examine its interplay with other cognitive mechanisms. Secondly, given that imitation is known to involve various cognitive processes, we aim to evaluate if a motor resonance mechanism is enough to evoke correct imitation performance in children with ASD. Thirdly, it has been speculated that there is another motor reasoning route out of the motor cortex. Thus, we aim to examine the correspondence between central MNs activation and the rate of action reproduction.

1.17 Priming

Visual properties are usually perceived more quickly and easily by a person if they have previously been presented to him/her, in spite of whether the individual remembers seeing them or not. This phenomenon, called 'priming,' implies that previous exposure to an item/object has a persistent effect on later task performance, by changing the representation of the observed item (Gauthier, 2000). The characteristic of the priming task would determine the repetition; for instance, in memory, a priming task is known to last over various intervals of periods (e.g. a few seconds, minutes, or hours), which differs from other types of semantic priming that last for a few seconds (Neely, 1991).

There have been many speculations formed by scholars about the concept of 'priming,' and how it occurs; for instance, Byrne and Russon (1998) hypothesised that observing an action that has already been observed before, in which this action became known or familiar, would act as a priming stimulus. Therefore, any imitation task that was not built on a novel action is considered to be a priming stimulus. Other views were formed by Neurophysiological studies like that of Miller & Desimone (1994) which demonstrated that object repetition was accounted for in brain activity reduction in the infero-temporal and frontal cortex. It has been suggested that this attenuation is

reflective of a sharpening process that occurs in the neural networks representing the priming objects.

This view hypothesized that, during primary observation of an object, a group of neurons are tuned, and with repetition of visual observation of that object, there will be a decrease in the responsiveness of neurons that carry little information; concurrently, there will be an increase in the responsiveness of a population of informative neurons, which become more efficient.

The priming varies depending on sensory modality, and thus, for instance, there will be tactile, auditory, and visual priming (Van Beilen, 2011), a category that is being used in the current thesis. Observational priming seems to depend on perceptual to motor transformation, in which the internal representations correspond to the observed phenomenon.

Based on this, whenever an individual observes an action or item that has already been seen by him, the ‘observing’ will trigger the internal representations that exist in the individual’s own repertoire. Through growing research and studies into priming, a concern has been raised questioning the causes which underlie the suppression and enhancement that occurs in visual priming. In an fMRI study (Henson et al., 2000), researchers found that priming effects are largely dependent on the familiarity of the observed stimulus in that enhancement of neural activity was observed solely for familiar objects linked to names or semantic information, while a decrease in neural activity occurs when repeatedly observing novel objects.

When the same neuroimaging technique, fMRI, was used to investigate visual priming with familiar and unfamiliar faces, suppression occurred to the fusiform gyrus for repeated familiar faces; however, there was enhancement in response to repeated unfamiliar faces. Researchers have taken these observations to build an understanding of the period of time that an unfamiliar stimulus would require to be seen in order to become familiar and they found that merely observing a recent stimulus was not sufficient to make it familiar.

Within this thesis, we will utilise a visual priming paradigm, to investigate the effects of

three main perceptual properties of familiarity and similarity on action understanding and imitation; in Chapter 3, we examine the priming effect of observing a familiar person. In Chapter, 4, we aim to investigate the priming effect of observing a similar aged person, and lastly, in Chapter 5, we will examine the priming effect of observing a similar ethnic model.

1.18 Summary and current thesis

The literature that has been discussed could be considered to comprise a possible neural model of MNs and how this model overlaps anatomically with 'social cognition', which leads to a growing body of neuroimaging and neurophysiological experiments aiming to understand its functions and its role in the course of social development. With the emergence of initial empirical data, suggesting an effect to MNs abnormality in the manifestation of social deficits, a plethora of hypotheses have been postulated either to support or challenge the existing literature proposing that the failure of ASD in performing social cognitive tasks (e.g. understanding others' actions) is accounted for by failure in MNs activation, referred to broadly as 'broken mirror neurons.'

This was synchronised with another line of investigations, with a variety of experimental techniques and methodologies to examine the circumstances, and properties (e.g. familiarity) that would trigger these MNs in this clinical population. In this current thesis, we have adopted Oberman et al.'s (2005) EEG methodological investigation paradigm. Therefore, the interested frequency bands, alpha (8-12Hz), low beta (13-20Hz), and high theta (5.5-7.5Hz), were obtained from central electrode sites (C3, Cz, and C4) during both observation and imitation conditions. Besides the neural activity recording, all imitation conditions were recorded for off-line analysis. Both TD and ASD children were within pre-school grades. Prior to their EEG session recording, they were evaluated using the Wechsler Intelligence Scale for Children (WISC) (Ismail & Malika, 1974), as well as the Autism Diagnostic Interview –Revised (ADI-R; Lord et al., 1994).

This led to the aim in **Chapter 2 - Experiment 1:** to investigate *if pre-school children with ASD would show greater MNs activity compared to TD children, during observation*

of a familiar model (parent) compared to an unfamiliar model, while this model is performing familiar communicative hand gestures or meaningless gestures. Upon demonstrating increased alpha and low beta suppression for observing familiar models, this effect of person familiarity will be employed in **Chapter 3 - Experiment 2:** where we utilise a visual priming paradigm to see *whether incorporating a prime familiar model (parent) would lately facilitate action understanding and imitation of an unfamiliar model executing the same communicative hand gestures, in preschool children with ASD.*

In **Chapter 4 - Experiment 3:** we will utilise the same priming paradigm, attempting to investigate two unanswered questions: firstly, *do children with or without ASD show evidence of enhanced action understanding when observing a similar-aged person (child) performing a hand gesture, compared to observing a dissimilar-aged person (adult) performing the same gesture?* Secondly, *if the effect of age similarity is present in children with ASD, can this similar-age facilitation effect be used to prime qualitative changes in behaviour when observing a dissimilar-aged person performing a hand gesture?*

In **Chapter 5 - Experiment 4:** we will utilise the same priming paradigm, attempting to investigate two unanswered questions: firstly, *do children with or without ASD during observing a similar-ethnic model (Saudi) performing familiar communicative gestures, elicit greater neural activity of MNs compared to observing a dissimilar-ethnic model (European) performing the same gesture.* Secondly, *does incorporating a prime similar-ethnic model subsequently elicit qualitative MNs neural activation for observing a dissimilar-ethnic model performing communicative hand gestures, in ASD children?*

In **Chapter 6 - Discussion:** we aim to incorporate our findings and previous data, with a view to showing the contribution of current findings to the literature, and to discussing plausible elements of divergence or agreement between our work and previous studies.

2 Person familiarity facilitates action understanding

2.1 EXPERIMENT 1

Individuals with ASD are known to have remarkable difficulty in social reciprocity, and in expressively and receptively communicating with others (American Psychiatric Association, 1995). Techniques of teaching and training children with ASD are many; nonetheless, they are challenging. One highlighted method, which is known to hold the curiosity of many researchers, is modelling (Charlop et al., 1983), which requires the person to observe another individual performing target behaviour.

Although this type of observational learning has been found to be effective with normally developing children (see Bandura et al., 1961), it is particularly successful with the ASD population for various reasons (see chapter 1, section 1.7), for example, because these individuals demonstrate better processing for visual rather than auditory stimuli) (Hodgdon, 1995).

While individual learning is controlled largely by the interaction between the individual and its environment, imitative learning is widely influenced by the state (e.g. age, social status, and race) of others in the individual's population (Cavalli-Sforza & Feldman, 1981). Through this remarkable 'social learning theory', Bandura (1977) urged the literature of modelling with the conceivable impact of embedded social aspects of models, illustrating how an individual acquired a new behaviour through observing behaviour, and its consequences on the performer. There are different types of modelling that comprise: 'direct modelling' (i.e. copying the model's behaviour), 'synthesized modelling' (i.e. merging a number of observations to build a new behaviour), and 'symbolic modelling' (i.e. copying fictional characters from a book) (Goetz et al., 1992).

In relation to the present experiment, in which visual video modelling that depicts social/ communicative content, is used with children with ASD, the primary contribution was voiced in Oberman et al.'s work (2008); their EEG study, merged the methodology of utilising the social aspect of parental familiarity and the effectiveness of visual video modelling in facilitating MNs activation in 8-12 year individuals with ASD.

The effect of person familiarity seems to match the proposal about a familiar bias put forward by Sai (2005) who showed that since birth, children show a preference for looking at their mother's face compared to a stranger, which reflects the fact that the majority of the child's time is spent in their parent's presence. Thus, the effect of early exposure and experience could have resulted in children showing better simulations of their parent compared to a stranger. This is supported by the argument that people tend to sympathize more with known, and in-group people, compared to unknown and out-group people (Hornstein, 1976).

Although the majority of behavioural studies were consistent in reporting significant evidence of poor performance on imitation, the inconsistency was found in inferring the attribution, even in the case of intact performance. Affected imitation could be caused by malfunction of mirror neurons, or by failure of other brain systems. Group differences in regions that do not literally correspond with MNs areas were established by evidences of a number of brain structure studies.

These regions include the cerebellum (Toal et al., 2010; Brambilla et al., 2003), the fusiform (Toal et al., 2010; Duerden et al., 2012), the cingulate and insula (Duerden et al., 2012), but not in IFG or IPL. The fact that the difference between ASD and control group brain structure was not limited on related MNs areas raised the debate about the possibility of the involvement of other brain structures. The suggestion of Oberman and Ramachandran (2007) of the possibility of existing broken mirror neurons in the cerebellum seems to be plausible, as it appears to support its disruption in autism, and its involvement in shifting attention and movements (Iarocci & McDonald, 2006), which could be linked to repetitive behaviour and difficulty in shifting from one activity to another (DiCicco-Bloom et al., 2006).

Despite the debatable causes of defected imitative performance in the ASD population, in this thesis, we extend our interest in examining the effect of familiarity, through video modelling, on the immediate behavioural performance of ASD children with matched TD children.

In the current experiment, observing and executing paradigm is used in an attempt to replicate:

1) *If observing a familiar action performed by a familiar model (parent) would elicit greater MNs activation relative to observing an unfamiliar model (adult) performing the same hand gestures, in ASD children.*

Accordingly, the current experiment adopts a simple observing/ executing paradigm, where two groups of children, one with ASD and one control, watched 4 video clips depicting actors performing a simple hand action. The participant was then required to imitate the action after each video. In this experiment, we crossed a familiar actor (i.e. a parent) with an unfamiliar actor (i.e. a stranger), with action familiarity (i.e. a familiar action *vs*. an unfamiliar one). Video recordings of the children's imitation performance were collected for later coding and analysis. Given the existing literature discussed earlier, which has suggested a great influence by a familiar person using a familiar action, we hypothesized that:

1) Children with ASD will demonstrate decreased levels of MNs activation relative to that in TD children, as reflected in alpha and low beta suppression.
2) Children with ASD will demonstrate fewer correct imitations of the hand action relative to TD children.
3) Both children with ASD and TD children will demonstrate decreased levels of MNs activation when observing an unfamiliar action relative to observing a familiar action.
4) Children with ASD and TD children will demonstrate increased levels of MNs activation when observing a familiar model, relative to an unfamiliar model.
5) The level of MNs activation when observing a familiar person performing a familiar action will be higher than when observing an unfamiliar action in TD and ASD children.

2.2 METHODS

2.2.1 Participants

The participants originally comprised 22 control children and 16 children diagnosed

with ASD; they ranged in age from 3 years/5 months to 5 years/1 month. The ASD participants were recruited from the Autism Research and Treatment Center at King Khaled Hospital in Riyadh, whereas the control participants were recruited from selected nurseries in Riyadh, Saudi Arabia. All participants had normal or corrected-to-normal vision prior to testing. Autistic participants who had comorbid neurological conditions or full scale IQ<80 were excluded.

	Control group	**ASD group**	
Full sample	(*n*= 15)	(*n*= 14)	
Age (*M*/*SD*)	4.2 ± 0.2	4.3 ± 0.3	$t(27) = -.11, p < .90$
Gender	6M/9F	6M/8F	
Handedness	11RH/4LH	10RH/4LH	
Ethnicity (% Saudi)	100%	100%	
Sub-sample	(*n*= 10)	(*n*= 11)	
Age (*M*/*SD*)	4.2 ± 0.3	4.3 ± 0.2	$t(19) = .14, p < .88$
Gender	3M/7F	3M/8F	
Handedness	6RH/4LH	8RH/3LH	
Ethnicity (% Saudi)	100%	100%	

ASD, autism spectrum disorder; F, female; M, male; RH, right-handed; LH, left-hande

Table 1 Descriptive characteristics of the full and sub-sample

Three participants from the control group and one from the ASD group were excluded due to an excessively noisy EEG recording. 4 participants from the ASD group and one from the control were also excluded as their parents did not comply with the instructions during hand stimuli preparation. Descriptive characteristics of the current sample are summarised in table 2.1.

Participants were either reimbursed for their participation at a rate of £20 per 30 minutes or rewarded with toys of a similar value. All participants 'parents or legal guardians gave informed, signed consent. Permission to conduct the current study was granted by the Research Ethics Committee of the School of Psychology at the University of Kent, in collaboration with the Autism Research and Treatment Center at King Khaled Hospital. The research was conducted in accordance with the ethical standards of the British Psychological Society.

2.2.2 Visual stimuli

Four critical types of visual stimuli were created for the current experiment: (I) a familiar person performing a familiar action, (II) a familiar person performing an unfamiliar action, (III) an unfamiliar person performing a familiar action, and (IV) an unfamiliar person performing an unfamiliar action. 'Familiar person' stimuli depicted one of the subject's parents performing an action, while 'unfamiliar person' stimuli depicted different unknown middle-aged males performing an action.

Familiar actions involved the performance of one of two repetitive culturally familiar communicative gestures (Nydell, 2002, p-57), either a 'no' sign (by moving the index finger from side to side), or a 'come here' sign (by moving four fingers together in a beckoning action). Unfamiliar actions involved the performance of one of two repetitive unfamiliar (meaningless) hand gestures, either moving the hand in a rotating movement, or making a fist with the four fingers and moving the hand horizontally, as seen in figure 2.1.

Each video clip lasted for 80 seconds and all were silent, coloured video clips depicting the actor against a plain white background. Thus, person familiarity and action familiarity were manipulated in a fully crossed design, such that each familiar actor, each unfamiliar actor, and each action (familiar and unfamiliar) was seen only once during the entire experimental session.

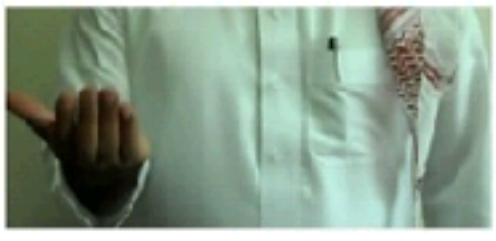
Observation

Imitation

Familiar Person/Familiar Action

Familiar Person/Unfamiliar Action

Unfamiliar Person/Familiar Action



Unfamiliar Person/Unfamiliar Action

Figure 5 Four example experimental trials showing the four video stimuli combinations. Each trial consists of one observing period followed by one imitation period, each lasting 80 seconds. The video clip of each trial depicted one of the four conditions described above, crossing both person familiarity and action familiarity.

Each of these experimental stimuli was analysed in comparison to a baseline visual stimulus condition: white visual noise. This white visual noise depicted a unified, silent, white visual noise video clip that lasted for 30 seconds.

Figure 6 Still image of white visual noise used in the 'baseline condition'.

2.2.3 Procedures

Clinical assessment

Initially, participants in the control group were assessed with the Egyptian version of the Wechsler Intelligence Scale for Children (WISC) (Ismail & Malika, 1974). ASD participants' diagnoses were confirmed by clinical evaluations based on DSM-IV criteria as well as the Autism Diagnostic Interview –Revised (ADI-R; Lord et al., 1994).

	Control group	ASD group	*t* *p* Cohen's *d*
Full sample			
VIQ	116.4 ± 6.15	115.57 ± 9.38	$t(27) = .28, p = .77, d = .10$
PIQ	109.33 ± 9.02	106.85 ± 10.51	$t(27) = .68, p = .50, d = .25$
FSIQ	112.8 ± 6.6	110 ± 8.1	$t(27) = 1.01, p = .32, d = .38$
ADI-R:			
Reciprocal Social Interaction	6.2 ± 1.78	19.2 ± 1.18	$t(27) = -22.97, p < .001, d = -8.84$
Communication	4.4 ± 1.50	15.5 ± .94	$t(27) = -23.64, p < .001, d = -9.09$
Restricted, Repetitive, and Stereotyped Patterns of Behavior	0.34 ± .15	4.2 ± .69	$t(27) = -20.94, p < .001, d = -8.05$
Sub-sample			
VIQ	116.20 ± 6.76	116.54 ± 9.08	$t(19) = .09, p = .92, d = .04$
PIQ	108.40 ± 9.96	110.36 ± 11.58	$t(19) = .41, p = .68, d = .18$
FSIQ	112.30 ± 7.74	110.81 ± 8.54	$t(19) = .41, p = .68, d = .16$
ADI-R:			
Reciprocal Social Interaction	6.60 ± 2.01	19 ± 1.26	$t(19) = -17.08, p < .001, d = -7.83$
Communication	3.90 ± 1.19	15.63 ± .92	$t(19) = -25.28, p < .001, d = -11.59$
Restricted, Repetitive, and Stereotyped Patterns of Behavior	0.35 ± .15	4.18 ± .75	$t(19) = -16.52, p < .001, d = -7.57$

ADI-R, Autism Diagnostic Interview- Revised (ADI-R; Lord, Rutter, & Le Couteur, 1994)
FSIQ, full-scale intelligence quotient; average subtests: information,comprehension,arithmetic, similaritie, ,vocabulary,digit span, picture completion, picture arrangement, sblock design, object assembly, and coding (WISC; Ismail & Malika, 1974)

Table 2 Descriptive characteristics of the clinical assessments for the sample & sub-sample

EEG data acquisition

During the main experiment, the experimenter prepared and tested each participant individually. Preparation for the EEG recording involved preparing the scalp area by cleaning and gently abrading the areas over which electrodes would be placed to reduce the impedance. The electrode cap was then fitted to the participant s̓ head, and the electrodes were filled with electro-conductive gel. During this procedure and the experimental tasks, participants sat comfortably either in a chair or on their parent s̓ lap, and were instructed to avoid excessive eye movements while watching the video clips.

Videos were presented on a 16 inch computer screen within comfortable viewing distance. Participants initially viewed the video of white visual noise (baseline condition), which lasted for 30 seconds. Then, they viewed each of the four video clips, as described above, in a counterbalanced order. Participants observed each video individually for 80 seconds, then immediately after were instructed to imitate the observed hand action for 80 seconds. With prior permission from each child s̓ parent, participants ʼhand actions were recorded throughout the imitation period for later analysis of behavioural performance.

Participants were invited to take short breaks between experimental videos, to ensure that they were alert and prepared for each recording phase. In total, the entire EEG recording period for Experiment 1 lasted approximately 20 minutes.

EEG data were collected from three electrodes over the sensorimotor cortex, from C3, Cz and C4 electrodes, and from the left and right mastoids, positioned according to the international 10-20 system. Impedance levels were lowered to at least 10 kΩ in all electrodes. The EEG signal was acquired using BIOPAC system (MP150), and Acknowledge software. EEG data were recorded against a linked mastoids reference, at a sampling rate of 1000 Hz. EEG data were collected for all observation and imitation periods.

Figure 7 BIOPAC system (MP150)

2.2.4 EEG data preparation and statistical analysis:

EEG data were analysed using Brain Vision Analyzer 2 (Brain Products). Firstly, the continuous EEG signal for each participant was filtered using a 40Hz low-pass cutoff and a 0.5Hz high-pass cutoff. Each 80 second period of continuous EEG for each condition was then divided into epochs of 2 seconds, with 50% overlap. Using a semi-automatic

artifact rejection method, any segments containing artifacts, such as muscle movement or drift, were identified and removed. Fast Fourier transform was then performed on the data using a 10% Hanning window. Averaged power data were obtained for each participant and condition, at each of the 3 electrode sites (C3, Cz, and C4) in each of the frequency bands of interest, and ERD was calculated, as proposed by Pfurtscheller and Aranibar (1977). ERD is defined as the percentage of decrease or increase in band power during a specific interval as compared to a reference interval. This ERD is calculated using the following formula:

*ERD% = ((Reference power activity –Test power activity) / (Reference power activity)) *100*

Thus, alpha/ Mu (8 –12Hz) and low beta (12 –20Hz) frequency desynchronization over the primary motor cortex (C4, CZ, and C3) was calculated as the percentage change in power for each of the test intervals (i.e. activity in each experimental condition), compared to the baseline reference interval (white visual noise) [1].

IBM SPSS version 20 software was used to perform ANOVAs on the EEG data comparing the between-participants factor, group (control/ ASD) with the repeated-measures factors, person (familiar/ unfamiliar), action (familiar/ unfamiliar) and task (observation/ imitation), averaged across electrodes (C3, Cz, C4). Given that corresponding behavioural data were not available for the full sample, EEG data were analysed primarily on a full sample and a sub-sample who only completed the behavioural and EEG measures. Note that degrees of freedom were corrected using Greenhouse-Geisser when Mauchly's test of Sphericity indicated that sphericity had been violated.

2.3 RESULTS

2.3.1 Behavioural results

Behavioural analyses were conducted to examine children's explicit ability to imitate observed actions in each condition. These analyses were conducted by hand-coding the

video recordings of each participant performing the imitation actions, in each condition. The number of times that children performed the correct action during each 80-second imitation period was counted, along with the number of incorrect actions performed in the same period.

Eight participants from the total experimental population did not consent to the use of video recording during the task. Therefore, the behavioural analyses were conducted on a smaller set of ten participants in the control group and eleven participants in the ASD group.

Correct imitations

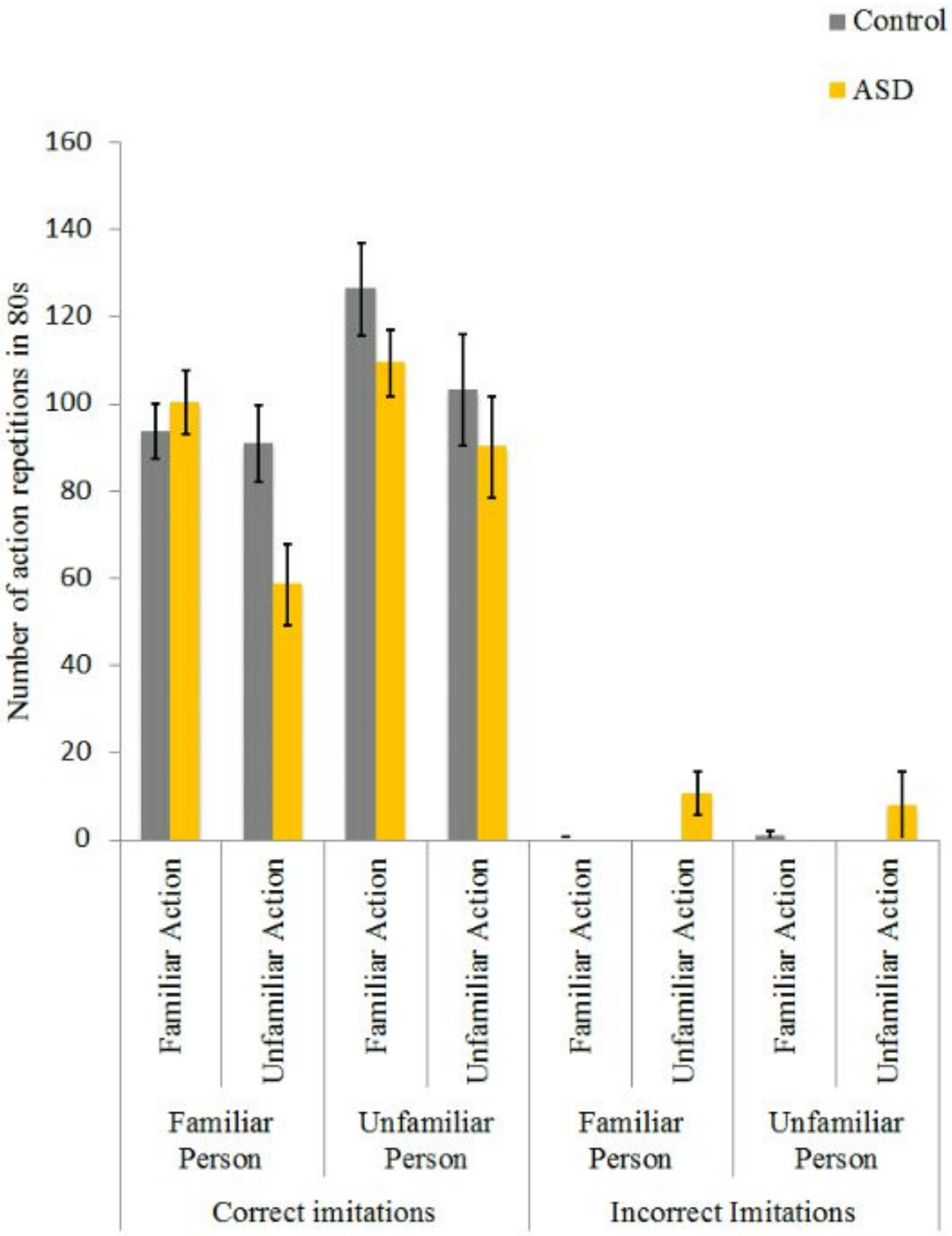


Table 3 Mean number of correct and incorrect imitations for control and ASD groups for each of the four imitation conditions. Error bars represent the standard error of the mean.

Is imitating communicative gestures impaired in ASD?

A three-way mixed 2 x 2 x 2 ANOVA was conducted to examine the effects of person (familiar/ unfamiliar) and action (familiar/ unfamiliar) as within-subject factors, and group (control/ ASD) as a between-subject factor on the number of correct action

imitations. Results revealed a number of significant main effects and interactions. The main effect of the group was not significant ($F < 2.49$, $p = .13$), but showed a trend towards an increased number of correct action imitations in the control ($M = 103.50$) versus the ASD group ($M = 89.56$).

Does person and action familiarity facilitate imitation performance?

A main effect of person, $F(1,19) = 26.96$, $p < .001$, $\eta_p^2 = .58$, demonstrated an increased number of correct imitations when participants were imitating an unfamiliar person ($M = 107.21$) compared to when they were imitating a familiar person ($M = 85.9$). The main effect of action was also significant, $F(1, 19) = 9.11$, $p = .007$, $\eta_p^2 = .32$, demonstrating an increased number of correct imitations for imitating a familiar action ($M = 107.40$) compared to an unfamiliar action ($M = 85.65$). None of the interactions reached significance ($Fs < 3.49$, $ps > .07$).

Incorrect imitations

A three-way mixed 2 x 2 x 2 ANOVA was conducted to examine the effects of person (familiar/ unfamiliar), and action (familiar/ unfamiliar) as within-subject factors, and group (control/ ASD) as a between-subject factor on the number of incorrect action imitations. Results showed that none of the main effects or interactions reached significance ($Fs < 2.84$, all $ps > .10$).

2.3.2 Electroencephalographic results

ERD was calculated in two frequency bands; alpha (8-12Hz) and low beta (12-20Hz).

Alpha frequency band (8-12 Hz)

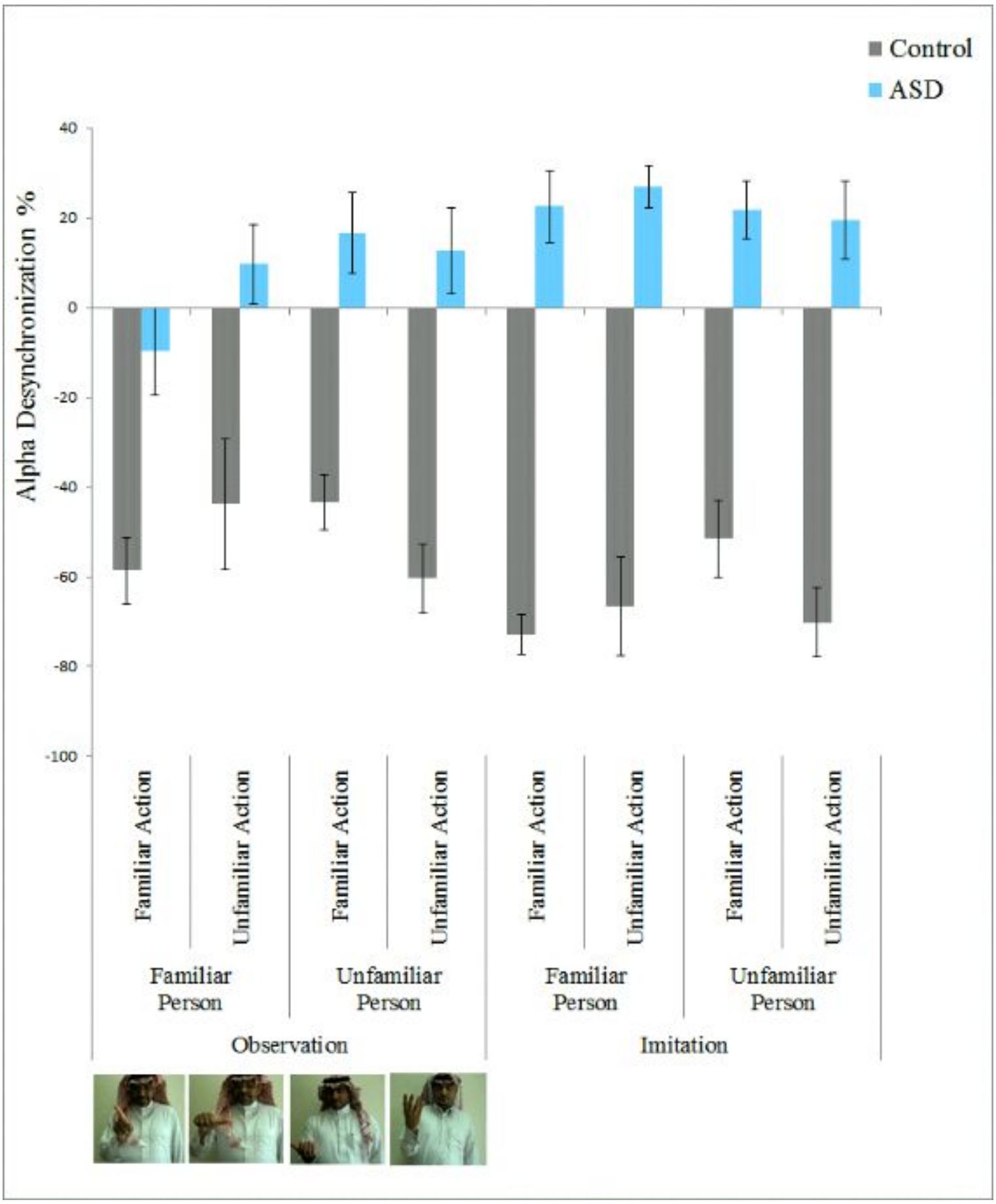


Figure 8 The percentage of event-related changes in alpha power for control and ASD groups. Error bars represent the standard error of the mean.

A four-way mixed 2 x 2 x 2 x 2 ANOVA, was conducted to examine the effects of person (familiar/ unfamiliar), action (familiar/ unfamiliar) and task (observing/ imitation), as within-subject factors, and group (control/ ASD) as a between-subject factor on alpha suppression[2]. Results revealed a number of significant effects and interactions which are displayed in table 2.3.

	All data			Control group			ASD group		
	df	F-value	p-value	df	F-value	p-value	df	F-value	p-value
Group	1,27	97.60	0.001***	-	-	-	-	-	-
Person	1,27	12.56	0.001***	1,14	93.67	0.001***	1,13	3.84	0.072
Action	1,27	0.01	0.93	1,14	0.14	0.71	1,13	5.40	0.037
Task	1,27	0.03	0.85	1,14	2.86	0.11	1,13	13.04	0.003**
Group*Person	1,27	0.21	0.64	-	-	-	-	-	-
Group*Action	1,27	0.63	0.43	-	-	-	-	-	-
Group*Task	1,27	9.68	.004**	-	-	-	-	-	-
Person*Action	1,27	29.19	0.001***	1,14	56.70	0.001***	1,13	4.27	0.059
Person*Task	1,27	1.53	0.22	1,14	2.25	0.15	1,13	27.22	0.001***
Action*Task	1,27	0.45	0.50	1,14	0.09	0.76	1,13	3.08	0.10
Group*Person*Action	1,27	2.75	0.10	-	-	-	-	-	-
Group*Person*Task	1,27	14.4	0.001***	-	-	-	-	-	-
Group*Action*Task	1,27	0.008	0.93	-	-	-	-	-	-
Person*Action*Task	1,27	0.81	0.37	1,14	0.08	0.78	1,13	4.01	0.067
Group*Person*Action*Task	1,27	0.14	0.70	-	-	-	-	-	-

* for p<.05, ** for p<.01 and *** for p<.001.

Table 4 Mixed & separate ANOVAs for alpha suppression cross person, action, and task

Is the MNs activity impaired in ASD?

A main effect of group, $F(1, 27) = 97.60$, $p < .001$, $_p\eta^2 = .56$, demonstrated an increased level of alpha suppression for the control group (M= -58.42%) relative to the ASD group (M= 15.08%).

Is MNs mediated by person and action familiarity?

A main effect of person, $F(1, 27) = 12.56$, $p = .001$, $_p\eta^2 = .32$, demonstrated an increased level of alpha suppression for a familiar person (M = 25.7%) relative to an unfamiliar person (M = 32.7%). Analysis of the control group showed a main effect of person, $F(1, 14) = 93.67$, $p < .001$, $_p\eta^2 = .87$, which demonstrated an increased level of alpha suppression for a familiar person (M = -60.45%) compared to an unfamiliar person (M = -56.38%).

In comparison, analysis of the ASD group showed that although there was greater alpha suppression for a familiar person (M = 12.43) compared to an unfamiliar person (M = 17.74%), the main effect of person was marginal, $F(1, 13) = 3.84$, $p > .07$, $_p\eta^2 = .22$. A main effect of action, $F(1, 13) = 5.40$, $p = .037$, $_p\eta^2 = .29$, demonstrated an increased level of alpha suppression for a familiar action (M = 12.88%) compared to an unfamiliar action (M= 17.28%).

In addition, planned comparisons (paired-samples t-tests), collapsed across task were conducted to explore the two-way interaction between person and action for the control group. For a familiar person, comparisons revealed that the alpha suppression of a familiar action, compared to an unfamiliar action, did not elicit a significant difference ($t < -1.37$, $p > .19$) - although the mean of familiar action was higher (M = -65.7%) relative to the mean of an unfamiliar action (M = -55.2%).

Similarly, for an unfamiliar person, comparisons revealed that the alpha suppression of a familiar action did not elicit a significant difference in comparison to an unfamiliar action ($t <1.6$, $p > .14$). Furthermore, when the action was familiar, comparison revealed that the alpha suppression of a familiar person was significantly higher (M = -65.7%), relative to an unfamiliar person (M = -47.5%), $t(14) = -12.4$, $p < .001$, d = -3.2. However,

when the action was unfamiliar, comparison revealed that the alpha suppression of a familiar person was significantly lower (M = -55.2%) relative to an unfamiliar person (M = -65.3%), $t(14) = 4.4$, $p < .001$, $d = 1.1$.

As for the ASD group, planned comparisons (paired-samples t-tests), collapsed across task were conducted to explore the two-way interaction between person and action for the ASD group. For a familiar person, comparisons revealed that the alpha suppression of a familiar action was significantly higher (M = 6.47%) relative to an unfamiliar action (M = 18.39%), $t(13) = -2.57$, $p = .023$, d= -1. Similarly, when the action was familiar, comparison revealed that the alpha suppression of a familiar person was significantly higher (M = 6.47%), relative to an unfamiliar person (M = 19.29%), $t(14) = -2.52$, $p = .025$, d = -3.2. For unfamiliarity, none of the comparisons elicited an acceptable level of significance (ts < .89, ps >.38).

Independent sample t-tests were run to explore the differences between the group effect of group*person*action interaction.

Comparisons of familiar person performing familiar action demonstrated that alpha suppression of the control group (M = -65.73%) was significantly higher compared to the ASD group (M = 6.47%), $t(27) = -6.97$, $p < .001$, $d = 2.57$. Similarly, comparisons of familiar person performing unfamiliar action demonstrated that alpha suppression of the control group (M = -55.18%) was significantly higher compared to the ASD group (M = 18.39%), $t(27) = -11.22$, $p < .001$, $d = 4.10$. In addition, comparisons of unfamiliar person performing familiar action demonstrated that alpha suppression of the control group (M = -47.48%) was significantly higher compared to the ASD group (M = 19.29%), $t(27) = -6.34$, $p < .001$, $d = 2.35$. Similarly, comparison of unfamiliar person performing unfamiliar action demonstrated that alpha suppression of the control group (M = -65.28%) was significantly higher compared to the ASD group (M = 16.18%), $t(27) = -8.84$, $p < .001$, $d = 3.24$.

Was MNs functioning influenced by IQ and hand imitation skills?

Correlational analyses revealed insignificant association between alpha suppression and intelligence quotient, *rs* < -.019, *ps* > .69. Similarly, there was no significant association between alpha suppression and behavioural imitation, *rs* < -.23, *ps* > .30.

Low beta band (12-20Hz)

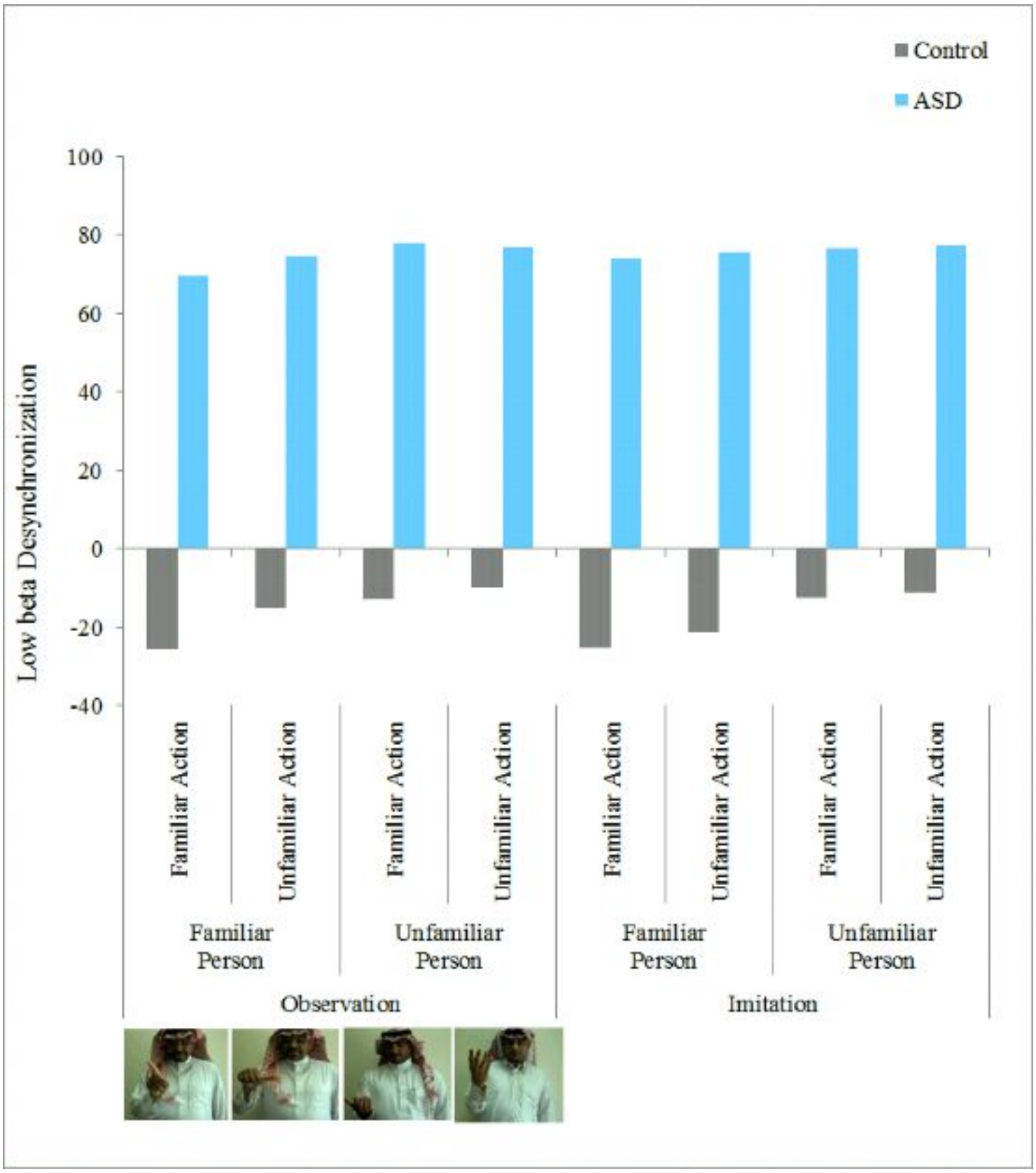


Figure 9 The percentage of event-related changes in low beta power for control and ASD groups. Error bars represent the standard error of the mean.

As in alpha band, a four-way mixed 2 x 2 x 2 x 2 ANOVA, was conducted to examine the effects of person (familiar/ unfamiliar), action (familiar/ unfamiliar), and task

(observing/ imitation), as within-subject factors, and group (control/ ASD) as a between -subject factor on low beta suppression[3]. Results revealed a number of significant main effects and interactions which are displayed in table 2.4.

	All data			Control group			ASD group		
	df	F-value	p-value	df	F-value	p-value	df	F-value	p-value
Group	1,27	230.7	0.001***	-	-	-	-	-	-
Person	1,27	58.03	0.001***	1,14	43.57	0.001***	1,13	16.61	0.001***
Action	1,27	39.3	0.001***	1,14	43.43	0.001***	1,13	4.98	0.044*
Task	1,27	0.22	0.63	1,14	98.28	0.001***	1,13	1.05	0.32
Group*Person	1,27	12.47	.002**	-	-	-	-	-	-
Group*Action	1,27	9.96	.004**	-	-	-	-	-	-
Group*Task	1,27	6.65	.016**	-	-	-	-	-	-
Person*Action	1,27	30.89	0.001***	1,14	17.40	0.001***	1,13	14.44	.002**
Person*Task	1,27	0.73	0.39	1,14	102.67	0.001***	1,13	8.11	.014**
Action*Task	1,27	5.32	0.029*	1,14	16.88	0.001***	1,13	0.18	0.67
Group*Person*Action	1,27	1.06	0.31	-	-	-	-	-	-
Group*Person*Task	1,27	24.16	0.001***	-	-	-	-	-	-
Group*Action*Task	1,27	2.32	0.13	-	-	-	-	-	-
Person*Action*Task	1,27	27.43	0.001***	1,14	20.26	0.001***	1,13	17.17	.007**
Group*Person*Action*Task	1,27	0.01	0.93	-	-	-	-	-	-

* for p<.05, ** for p<.01 and *** for p<.001.

Table 5 Mixed & separate ANOVAs for low beta suppression across person, action, and task

Is the MNs activity impaired in ASD?

A main effect of group, $F(1, 27) = 230.73$, $p < .001$, $_p\eta^2 = .97$, demonstrated an increased level of low beta suppression for the control group ($M = -16.70\%$) relative to the ASD group ($M = 75.17\%$).

Is MNs activity mediated by person and action familiarity?

A main effect of person, $F(1, 27) = 58.03$, $p < .001$, $_p\eta^2 = .68$, demonstrated an increased level of low beta suppression for the familiar person ($M = 25.74\%$) compared to the unfamiliar person ($M = 32.72\%$). Further, a main effect of action, $F(1, 27) = 39.25$, $p < .001$, $_p\eta^2 = .59$, demonstrated an increased level of low beta suppression for familiar action ($M = 27.65\%$) compared to unfamiliar action ($M = 30.80\%$).

In addition, analysis of the control group revealed a main effect of person, $F(1, 14) = 43.57$, $p < .001$, $_p\eta^2 = .78$, demonstrating an increased level of low beta suppression for the familiar person ($M = -21.81\%$) compared to an unfamiliar person ($M = -11.59\%$). A main effect of action, $F(1, 14) = 43.43$, $p < .001$, $_p\eta^2 = .76$, demonstrated an increased level of low beta suppression for a familiar action ($M = -19.07\%$) compared to an unfamiliar action ($M = -14.34\%$).

In comparison to the control group, analysis of the ASD group showed a main effect of person, $F(1, 13) = 16.61$, $p = .001$, $_p\eta^2 = .56$, which demonstrated an increased level of low beta suppression for a familiar person ($M = 73.29\%$) compared to an unfamiliar person ($M = 77.04\%$). A main effect of action, $F(1, 13) = 4.98$, $p < .044$, $_p\eta^2 = .28$, demonstrated an increased level of low beta suppression for a familiar action ($M = 74.39\%$) compared to an unfamiliar action ($M = 75.95\%$).

Is familiarity of person necessary for motor resonance in control and ASD?

Planned comparisons (paired-samples t-tests), were conducted to explore the three-way interaction between person, action, and task for the control group. During observation, for a familiar person, comparison revealed that the low beta suppression of a familiar action was significantly higher ($M = -25.75\%$) relative to an unfamiliar action ($M = -15.08\%$), $t(14) = -5.35$, $p < .001$, $d = -1.3$. Similarly, when action was familiar, the low

beta suppression of a familiar person was significantly higher (M = -15.08%) relative to an unfamiliar person (M = -9.98%), $t(14)$ = -7.01, p < .001, d = -1.8. Furthermore, for an unfamiliar person, comparison revealed that the low beta suppression of a familiar action was significantly higher (M = -12.79) relative to an unfamiliar action (M = -9.98), $t(14)$ = -4.84, p < .001. Similarly, when the action was unfamiliar, comparison revealed that the low beta suppression of a familiar person was significantly higher (M = -25.75%) relative to an unfamiliar person (M = -12.79%), $t(14)$ = -5.38, p < .001, d= -1.4)., d= -1.2.

A similar trend of significance was revealed by the comparisons during imitation; for a familiar person, comparison revealed that the low beta suppression of imitating a familiar action was significantly higher (M = -25.26%) relative to an unfamiliar action (M = -21.16%), $t(14)$ = -5.31, p < .001, d= -1.4). Similarly, when the action was familiar, comparison revealed that the low beta suppression of imitating a familiar person was significantly higher (M = -25.26%) relative to an unfamiliar person (M = -12.48%), $t(14)$ = -6.62, p < .001, d= -1.7.

Furthermore, for an unfamiliar person, comparison revealed that the low beta suppression of imitating a familiar action was significantly higher (M = -12.48%) relative to an unfamiliar action (M = -11.12%), $t(14)$ = -5.65, p < .001, d= -1.4). Similarly, when the action was unfamiliar, comparison revealed that the low beta suppression of imitating a familiar person was significantly higher (M = -21.16%) relative to an unfamiliar person (M = -11.12%), $t(14)$ = -8.10, p < .001, d = -2.

In addition, comparison of each observation condition with the corresponding imitation condition revealed that the imitation condition was significantly higher relative to observation in two comparisons: in a familiar person performing an unfamiliar action (observation: M = 15.08% ,imitation: M = 21.16%), $t(14)$ = -9.15, p < .001, d = 2.36, and in an unfamiliar person performing an unfamiliar action (observation: M = -9.98% ,imitation: M = -11.12%), $t(14)$ = 4.33, p = .001, d = 1.11. None of the remaining comparisons reached significance (ts < -.60, ps >.55).

As for the ASD group, planned comparisons (paired-samples t-tests) were conducted to

explore the three-way interaction between person, action, and task for the ASD group. During observation, for a familiar person, comparison revealed that the low beta suppression of a familiar action was significantly higher (M = 69.34%) relative to an unfamiliar action (M = 74.45%), $t(13) = -4.13$, $p = .001$, $d = -1.1$. Similarly, when the action was familiar, comparison revealed that the low beta suppression of a familiar person was significantly higher (M= 69.34%) relative to an unfamiliar person (M= 77.92%), $t(13) = -7.87$, $p < .001$, d= -2.1. In contrast, unfamiliarity did not elicit significance (ts < -2.03, ps > .06).

During imitation, for a familiar person, comparison revealed that the low beta suppression of a familiar action was higher (M = 73.87%) relative to an unfamiliar action (M = 75.52%), but it did not reach significance ($t < -.96$, $p > .35$). Furthermore, when the action was familiar, comparison revealed that the low beta suppression of a familiar person was marginally higher (M = 73.87%) relative to an unfamiliar person (M = 76.42%), $t(13) = -2.06$, $p = .059$, d= -0.5. In contrast, unfamiliarity did not elicit significance (ts < -1, ps > .3).

In addition, comparing each observation condition with the corresponding imitation condition revealed that none of the comparisons reached significance (ts < -.60, ps >.55).

Independent sample t-tests were run to explore the differences between the group effect of group*person*action*task interaction.

Comparisons of observation of familiar person performing familiar action demonstrated that low beta suppression of the control group (M = -25.75%) was significantly higher compared to the ASD group (M = 69.34%), $t(27) = -33.49$, $p < .001$, $d = 12.51$. Similarly, comparisons of imitation of familiar person performing familiar action demonstrated that low beta suppression of the control group (M = -25.26%) was significantly higher compared to the ASD group (M = 73.87%), $t(27) = -29.87$, $p < .001$, $d = 10.98$. Comparisons of observation of familiar person performing unfamiliar action demonstrated that low beta suppression of the control group (M = -15.08%) was significantly higher compared to the ASD group (M = 74.45%), $t(27) = -39.76$, $p < .001$, d = 14.53. Similarly, comparisons of imitation of familiar person performing unfamiliar

action demonstrated that low beta suppression of the control group (M = -21.16%) was significantly higher compared to the ASD group (M = 75.52%), $t(27) = -41.89$, $p < .001$, $d = 15.37$.

In addition, comparisons of observation of unfamiliar person performing familiar action demonstrated that low beta suppression of the control group (M = -12.79%) was significantly higher compared to the ASD group (M = 77.92%), $t(27) = -54.24$, $p < .001$, $d = 19.86$. Similarly, comparisons of imitation of unfamiliar person performing familiar action demonstrated that low beta suppression of the control group (M = -12.48%) was significantly higher compared to the ASD group (M = 76.42%), $t(27) = -33.78$, $p < .001$, $d = 12.33$. Comparisons of observation of unfamiliar person performing unfamiliar action demonstrated that low beta suppression of the control group (M = -9.98.66%) was significantly higher compared to the ASD group (M = 76.75%), $t(27) = -53.17$, $p < .001$, $d = 19.48$. Similarly, comparisons of imitation of unfamiliar person performing unfamiliar action demonstrated that low beta suppression of the control group (M = -11.12%) was significantly higher compared to the ASD group (M = 77.07%), $t(27) = -50.75$, $p < .001$, $d = 18.56$.

Was MNs functioning influenced by IQ and hand imitation skills?

Correlational analyses revealed insignificant association between low beta suppression and intelligence quotient, $rs < -.22$, $ps > .32$. Similarly, there was no significant association between low beta suppression and behavioural imitation, $rs < -.34$, $ps > .12$.

2.4 DISCUSSION

The main aim of the current experiment is to replicate what has been established in previous literature (see Oberman et al., 2008), that both TD and ASD children will show greater capacity to simulate familiar actors, reflected by greater neural suppression. In addition, this simulation will be greater while the familiar actor is performing a familiar action. As predicted, EEG data demonstrated a number of important findings; firstly, it showed significant increase in alpha and low beta suppression in TD children compared

to ASD children.

Secondly, in terms of familiarity, it showed significant alpha and low beta suppression for a familiar person compared to an unfamiliar person. In addition, it showed significant low beta suppression for a familiar action; however, in the alpha range, familiar action was significantly higher in ASD; as for TD children, although the mean of familiar action was higher, it did not reach significance level.

In the exceptional case of increased neural activation for unfamiliar gestures in the TD children, there is a suggestion that TD children are likely to make an additional effort to reason out the action. This is in partial agreement with the findings of Vivanti et al. (2008) whose TD sample demonstrated increased attention to the face region, instead of the action region, when observing meaningless gestures; however, it did not lead to effective imitation performance, though their sample was older than ours. Vivanti and colleagues attribute this to the neutral facial expression of the actor, which seemed less likely to provide any cue to the meaning of the action. This argument fits well with the nature of our stimuli, as all the actors held a unifying neutral facial expression, across both categories of actions. Thus, facial expressions evoked no sufficient information to ease the imitation.

Thirdly, according to significant 'person*action*task' interaction in ASD, the low beta suppression of imitating a familiar person and familiar action were significantly higher compared to that during unfamiliar scenarios. As a most distinctive finding for ASD children, observing an unfamiliar person performing a familiar action did not evoke significant suppression compared to an unfamiliar action.

Absence of significant difference would suggest that ASD children failed to operate 'motor resonance' for meaningful communicative gestures as they were performed by an unfamiliar person; this seems not to be the case in TD children. Although TD children demonstrated greater suppression for familiar action when it was performed by a familiar person, they showed a significant capacity to operate 'motor resonance' for meaningful communicative gestures when it was performed by an unfamiliar person, an

effect that was clearly demonstrated by low beta suppression.

These findings critically indicated that the greater suppression that the ASD children demonstrated for operating 'motor resonance' in the case of familiar action did not operate in isolation from the effect of person familiarity. In another word, familiarity of the person seemed to facilitate action understanding. As for TD children, the result was not conclusive, for one reason: in the alpha range, comparing meaningful and meaningless actions when they were performed by an unfamiliar model did not elicit significance; however, in the low beta range, the result was clear in that the TD children were able to operate 'motor resonance' for meaningful action when it was performed by an unfamiliar model.

In referring to Oberman et al.'s argument (2007), understanding other's behaviour seems to rely on the capacity of the observer to perceive the observed individual as 'like me' (Meltzoff & Moore, 1995), and on the capacity to simulate the action 'operating motor resonance' within the observer's own motor representations. This view was supported by Oberman et al.'s finding (2008) in showing that the level of MNs suppression corresponded with the level of simulation and 'likeness' conceptualization in which the greatest suppression was evoked by observing one's own hand, and then the parent's hand; the least suppression was produced by observing a stranger's hand.

As for our predictions in relation to imitative performance, the effect of group did not reach significance level in either correct or incorrect imitation. However, the mean of correct imitation showed a trend towards an increased number of correct imitations in TD children compared to ASD children.

A question may arise as to why imitative performance would not statistically correspond with EEG neural suppression data. Besides the arguments that have been discussed in Chapter 1-section 1.13, it is worth incorporating the findings of present experiments with these arguments. Imitating an action implicates many cognitive mechanisms (Hamilton et al., 2007). Placing them under investigation, should lead to a possibility of addressing the accurate imitation profile in children with ASD. Many

researchers have discussed the possible impaired mechanisms (e.g. Rogers et al., 2003) arguing that memory is one of the implicated mechanisms that affects imitation; the argument which could be asserted relating to our work concerns the ability of current participants to ŕecall ʾthe action that they had observed; in the light of their current intellectual abilities score, the finding seems unlikely to stand for that claim, which seems in line with (Rogers et al., 1996), as they also found that no supportive evidence emerged.

Another piece of evidence, which relates to ASD imitation skills, argues that visual attention to the model ś face was positively related to the reproduction of the observed action (Carpenter et al., 1995) in which the reflected emotions, facial expressions and gaze ease understanding of the intention and the goal. Although there were some data that led to an agreement among scholars that ASD demonstrated a reduced tendency to look at the model ś face (Hobson & Hobson, 2007), there was, additionally, evidence to show that children with ASD illustrated equally atypical viewing patterns with social (Hobson & Hobson, 2007) and non-social scenes (Anderson et al., 2006).

In fact, attention to the actor ś face was only effective in the case where his facial expressions were reflecting relative emotions and facial expressions that could assist in understanding his mental state (Vivanti et al., 2008), which undoubtedly do not contribute in explaining our findings as all models in this experiment held unifying neutral facial expressions.

2.5 Conclusion

In this experiment, we have demonstrated four important findings. Firstly, consistent with the major findings of previous studies (Oberman et al., 2005), our data revealed significant increase in alpha and low beta suppression in TD children compared to ASD children. Secondly, in agreement with previous literature (e.g. Oberman et al., 2008), the effect of person familiarity was clearly revealed through significant alpha and low beta suppression for a familiar person compared to an unfamiliar person. Thirdly, in agreement with Oberman et al. (2008), ASD children demonstrated significant alpha and

low beta suppression for familiar action, as did TD children. Although the mean of familiar action was higher, it did not reach significance level. Fourthly, significant interactions in ASD children data revealed that ASD children could not operate 'motor resonance' for meaningful communicative gestures in isolation of the familiar person effect.

This, from the result, suggested that simulating familiar actions did not occur independently. Person familiarity eases that simulation. In contrast, TD children's data were not certain; these finding showed no significant difference between meaningful and meaningless gestures when they were performed by an unfamiliar model; nonetheless, in low beta range, the result was clear; the TD children were able to operate 'motor resonance' for meaningful action when it was performed by an unfamiliar model.

These findings, as intended, raised questions in two major areas: if 'motor resonance' of familiar action in ASD children seems to be facilitated by person familiarity, could person status (e.g. gender, age, and ethnicity) have a facilitative effect on 'motor resonance'? If yes, could this facilitative effect be used in a priming paradigm to facilitate the simulation? These two questions are addressed in the following three experiments.

Footnote

[1] Analysis of group differences in baseline condition demonstrated significant difference on the alpha frequency band for C3, $t(27) = 2.52$, $p = .018$, $d = 0.94$, Cz, $t(27) = 2.49$, $p = .019$, $d = 0.93$, and C4, $t(27) = 2.50$, $p = .019$, $d = 0.93$. However, after excluding the significant data in two cases, the baseline analyses showed insignificant difference ($t < 1.96$, $p > .61$). In addition, when conducting analyses on the alpha frequency band, after excluding the two significant cases, the main effect of group, $F = 83.78$, $p < .001$, $_p\eta^2 = .77$, and the significant interaction of person*action in the control group, $F = 49.50$, $p < .001$, $_p\eta^2 = .80$, and ASD group, $F = 4.27$, $p = .059$, $_p\eta^2 = .24$, remained consistent with the p values of full sample analyses. As for the low beta frequency band, analysis of group differences in the baseline condition did not reach significance ($t < .28$, $p > .77$).

[2] When we conducted analyses on the alpha frequency band in the sub-samples, the main effect of group (full sample: $F = 97.60$, $p < .001$, $_p\eta^2 = .56$/ sub-sample: $F = 213.33$, $p < .001$, $_p\eta^2 = .91$) remained consistently significant; however, the effect size associated with the main effect in the full-sample analysis was moderate, whereas the effect size associated with the main effect in the sub-sample analysis was large, which indicated a stronger effect. . The main effect of person (full sample: $F = 12.56$, $p = .001$, $_p\eta^2 = .32$/ sub-sample: $F = 7.97$, $p = .011$, $_p\eta^2 = .29$) remained consistently significant and yielded the same range of small effect size. As for the control group, the main effect of person (full sample: $F = 93.67$, $p < .001$, $_p\eta^2 = .87$/ sub-sample: $F = 64.67$, $p < .001$, $_p\eta^2 = .87$) and the interaction between person*action for the control group (full sample: $F = 56.70$, $p < .001$, $_p\eta^2 = .80$/ sub-sample: $F = 34.54$, $p < .001$, $_p\eta^2 = .79$) remained consistently significant and yielded the same range of large effect size. As for ASD, the main effect of person (full sample:

F= 3.84, p = .07, $_p\eta^2$= .22/ sub-sample: F= 3.19, p = .10, $_p\eta^2$= .24) and the interaction between person*action for the ASD group (full sample: F= 4.27, p = .059, $_p\eta^2$= .24/ sub-sample: F= 2.31, p = .15, $_p\eta^2$= .18) yielded the same range of small effect size.

[3] When we conducted analyses on the low beta frequency band in the sub-samples, the main effect of group (full sample: F= 230.73, p < .001, $_p\eta^2$= .97/ sub-sample: F= 1505.20, p < .001, $_p\eta^2$= .98), person (full sample: F= 58.03, p < .001, $_p\eta^2$= .68/ sub-sample: F= 45.99, p < .011, $_p\eta^2$= .70) and action (full sample: F= 39.25, p < .001, $_p\eta^2$= .59/ sub-sample: F= 27.10, p < .001, $_p\eta^2$= .58) remained consistently significant and yielded the same range of effect size. As for the control group, the main effect of person (full sample: F= 43.57, p < .001, $_p\eta^2$=. 78/ sub-sample: F= 24.22, p =.001, $_p\eta^2$= .72) and action (full sample: F= 43.43, p < .001, $_p\eta^2$=. 76/ sub-sample: F= 26.37, p =.001, $_p\eta^2$= .76) remained consistently significant and yielded the same range of large effect size. As for the ASD group, the main effect of person (full sample: F= 16.61, p = .001, $_p\eta^2$= .56/ sub-sample: F= 23.96, p = .001, $_p\eta^2$= .70) and action (full sample: F= 4.98, p = .044, $_p\eta^2$= .28/ sub-sample: F= 4.71, p = .055, $_p\eta^2$= .32) remained significant and within the same range of effect size. Although the effect size in the sub-sample analysis of person appeared to be larger in the value, both effect sizes are still within moderate range.
Although the interaction between person*action*task for the control group (full sample: F= 20.26, p < .001, $_p\eta^2$= .59/ sub-sample: F= 8.74, p = .016, $_p\eta^2$= .49) showed that the effect size associated with the interaction in the sub-sample analysis tended to be (theoretically) within small range compared to the effect size associated with the full-sample analysis, which tended to be within moderate range, the actual value of the sub-sample (statistically) was within the sealing level of the small range (toward moderate) and thus the actual difference is not large.
. As for the interaction between person*action*task for the ASD group (full sample: F= 10.17, p = .007, $_p\eta^2$= .43/ sub-sample: F= 6.31, p = .031, $_p\eta^2$= .38), although this showed that the effect size of sub-sample analysis tended to be smaller in the value than full-sample analysis, both effect sizes are still within small range, and therefore there is no significant difference between the value of full-sample and sub-sample analysis.
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3 The role of prime familiarity in action understanding and imitation

3.1 EXPERIMENT 2

In **Chapter 2-Experiment 1**, we have replicated the findings in showing the significant effect of person familiarity (Oberman et al., 2008) and action familiarity (Hwang and Hughes, 2000) in activating MNs. Additionally, we have added to the literature by demonstrating the great effect of person familiarity on easing the simulation of familiar action in ASD. Besides the remarkable influence of using video modelling with ASD individuals, that has been discussed earlier, we aim, in this chapter, to combine it with visual priming.

A much of the previous literature which entails observational learning has investigated the use of video and live priming, the form, purpose and context in which this priming is instructed, have varied. For instance, Schreibman et al. (2000) studied the efficacy of using video priming with ASD children. They hypothesized that, within transition situations (i.e. leaving the home, or going shopping), introducing children with prior priming to upcoming transitions, will aim to reduce or eliminate the disruptive behaviour of children with ASD.

In their study, three children with ASD who displayed severely disruptive behaviour during transitions, were instructed to view a short video explaining transition situations. Two major findings have been demonstrated: reduction or elimination of the disruptive behaviour, and the reduction of disruptive behaviour generalized to new transition situations.

McCann et al. (2005) have implemented live priming with a 4 year old participant with ASD within the school classroom targeting his impaired physical and verbal sharing. Priming was one component of their intervention beside prompting and praising during play time. The priming was specifically organised so that the instructor was sitting on a separate table with the participant and describing the importance of sharing, and explaining how to share. Their outcomes revealed that, relative to the initial baseline, there was an increase in verbal and physical sharing displayed by the participant,

compared to his peers, who were only prompted and praised, which apparently supports the critical effect of priming. In improving social reciprocity, Zanolli et al. (1996) conducted a priming study with two preschool children with ASD aiming to increase the spontaneous social initiations. Their priming methods included a high reinforcement session prior to the regular school activity. They demonstrated that levels of initiation increased compared with other peers, and they were able to respond effectively to most initiations with minimal teacher support.

In a recent study, David et al. (2011) investigated if defective multisensory processing in ASD persists while semantic non-social stimuli are presented. In their study, adult participants with Asperger syndrome (AS), with a matched control group, were instructed to perform a visual-audio priming task. Participants were presented with sounds which were primed by either semantically congruent or incongruent pictures of objects. The effect of priming was clearly revealed as participants with AS displayed accurate performance on congruent trials, compared to incongruent trials.

With a huge range of research studies emphasising the considerable effect of priming, inconsistent findings have also been reported. Pierno et al. (2006) investigated motor priming in children with ASD and TD children. They hypothesized that observing priming movements would facilitate the subsequent execution of an action.

Their participants were instructed to observe a human hand grasping action to an object (e.g. small sized object), which was followed by an execution task, in which the participants had to grasp the same sized object that was observed before, or different sized objects. The findings showed that reaching was found to be faster and more precise when the execution task consisted of the same sized object as that which had been observed. Researchers argued that prime observation facilitates the execution of matching actions. However, this priming effect was limited to control children. ASD participants failed to demonstrate a facilitation effect.

In the current experiment, a novel visual priming paradigm is used to investigate an

unanswered question:

Does priming by an unfamiliar model (adult male stranger) with a familiar model (parent) elicit qualitatively improved MN activation when observing communicative hand gestures in children with ASD?

Accordingly, two groups of children- with ASD and TD- watched 4 pairs of videos that depicted either a familiar model (one of the participant's parents), or an unfamiliar model (an unknown adult Saudi male) performing a familiar or unfamiliar hand action. The second video in each pair always depicted an unfamiliar person performing the same hand action. After each video pair, participants were asked to imitate the observed action. A video recording of the behavioural performance was also taken and used for further analysis. Based on the available literature, and the findings from Experiment 1, the following predictions were made:

1) Children with ASD will demonstrate decreased levels of MN activation relative to those in TD children, as reflected in mu suppression.
2) Children with ASD will demonstrate fewer correct imitations of the hand action relative to those in TD children.
3) Both children with ASD and TD children will demonstrate decreased levels of MN activation when observing an unfamiliar action relative to observing a familiar action.
4) Children with ASD and TD children will demonstrate increased levels of MN activation when observing a familiar model, relative to an unfamiliar model.
5) The level of MN activation when observing a familiar person performing a familiar action will be higher than when observing an unfamiliar action in TD and ASD children.
6) The neural activation to an unfamiliar model will be improved in conditions where that action was first primed by a familiar model.

3.2 METHODS

3.2.1 Participants

The same sample of participants who completed Experiment 1 participated in this experiment. Thus, the final sample comprised 14 children in the ASD group and 15 children in the control group, ranging in age from 3 years/5 months to 5 years/1 month (see sections 2.2.1 and 2.2.3.1 for full details). All participants' parents and legal guardians gave informed, signed consent. Permission was granted by the Research Ethics Committee of the School of Psychology at the University of Kent, in collaboration with the Autism Research and Treatment Center at King Khaled Hospital in Riyadh, Saudi Arabia. The research was conducted in accordance with the ethical standards of the British Psychological Society.

3.2.2 Visual stimuli

Four critical types of visual stimuli were created for the current experiment: (I) a familiar person performing a familiar action, (II) a familiar person performing an unfamiliar action, (III) an unfamiliar person performing a familiar action, and (IV) an unfamiliar person performing an unfamiliar action. As in Experiment 1, 'familiar person' stimuli referred to one of the participant's parents performing an action, while 'unfamiliar person' stimuli referred to different unknown middle-aged males performing an action.

Familiar hand actions involved the performance of either a 'come here' sign (by moving the index finger in a beckoning action), or a 'bye' sign (by waving an open hand). Unfamiliar (meaningless) hand actions involved the performance of either opening and closing the right hand vertically, or making a fist with the four fingers and moving the thumb in a rotating movement. Still images of these video stimuli are displayed in figure 3.1.

Each video clip lasted for 80 seconds and all were silent, coloured video clips depicting the actor against a plain white background. Thus, person familiarity and action familiarity were manipulated in a fully crossed design, such that each familiar actor,

each unfamiliar actor, and each action (familiar and unfamiliar) in combination was seen only once during the entire experimental session.

Observing 1	Observing 2	Imitation
 Familiar Person/Familiar Action	 Unfamiliar Person/Familiar Action	
 Familiar Person/Unfamiliar Action	 Unfamiliar Person/Unfamiliar Action	
 Unfamiliar Person/Familiar Action	 Unfamiliar Person/Familiar Action	
 Unfamiliar Person/Unfamiliar Action	 Unfamiliar Person/Unfamiliar Action	

Figure 10 Four example experimental trials showing the four video stimuli combinations. Each trial consists of two observing periods (observing 1 & 2) followed by one imitation period, each lasting 80 seconds. The first video clip of each trial depicted one of the four conditions described above, crossing both person familiarity and action familiarity. The second video clip in each pair depicted the same familiar/unfamiliar action, but this time was always performed by an unfamiliar person.

As in Experiment 1, each of these experimental stimuli was analysed in comparison to a baseline visual stimulus condition: white visual noise. This white visual noise depicted a unified, silent video clip that lasted for 30 seconds.

3.2.3 Procedures

Clinical assessment

Initially, participants in the control group were assessed with the Egyptian version of the Wechsler Intelligence Scale for Children (WISC) (Ismail & Malika, 1974). ASD participants' diagnoses were confirmed by clinical evaluations based on DSM-IV criteria as well as the Autism Diagnostic Interview –Revised (ADI-R; Lord et al. 1994).

EEG data acquisition

During the main experiment, the experimenter prepared and tested each participant individually. Preparation for the EEG recording was carried out as described in Experiment 1. Videos were presented on a 16-inch computer screen within comfortable viewing distance. Participants initially viewed the video of white visual noise (baseline condition), lasting for 30 seconds. This baseline period was followed by the four experimental trials, which presented participants with the visual stimuli described above, in a counterbalanced order.

On each trial, participants viewed two consecutive video clips, each lasting 80 seconds: the first video clip depicted one of the four conditions described above, crossing both person familiarity and action familiarity and the second depicted the same familiar/unfamiliar action, but this time, it was always performed by an unfamiliar person. This allowed us to examine whether the familiarity cues available in the first video influenced understanding of later repetitions of that action. Immediately after

each pair of videos, participants were instructed to imitate the observed hand action for 80 seconds.

With prior permission from the parents, participants' hand actions were recorded throughout the imitation period for later analysis of behavioural performance. Participants were invited to take short breaks between experimental videos to ensure they were alert and prepared for each recording phase. The entire EEG recording period for Experiment 2 lasted approximately 30 minutes.

EEG data were collected from three electrodes over the sensorimotor cortex (C3, Cz and C4) and from the left and right mastoids, positioned according to the international 10-20 system. Impedance levels were lowered to at least 10 kΩ in all electrodes. The EEG signal was acquired using BIOPAC system (MP150) and Acknowledge software, as in Experiment 1. EEG data were recorded against a linked mastoids reference at a sampling rate of 1000 Hz. EEG data were collected for all observation and imitation periods[1].

3.2.4 EEG data preparation and statistical analysis

EEG data were analysed using Vision Analyzer 2 (Brain Products). Firstly, the continuous EEG signal for each participant was filtered using a 40Hz low-pass cut-off and 0.5Hz high-pass cut-off. Each 80-second period of continuous EEG for each condition was then divided into epochs of 2 seconds, with 50% overlap. Using a semi-automatic artefact rejection method, segments containing artefacts, such as muscle movement or drift, were identified and removed. Fast Fourier transform was then performed on the data using a 10% Hanning window. Averaged power data of alpha (8 – 12 Hz) and low beta (12 –20 Hz) frequency bands were obtained and ERD was calculated as described in Experiment 1.

IBM SPSS version 20 software was used to perform ANOVAs on the EEG data, comparing the between-participants factor, group (control/ ASD) with the repeated-measures factors, ethnicity (similar/ dissimilar), action (familiar/ unfamiliar) and task

(observation 1/ observation 2/ imitation) averaged across electrodes (C3, Cz, C4). Given that corresponding behavioural data were not available for the full sample, EEG data were analysed primarily on a full sample and a sub-sample who had only completed the behavioural and EEG measures. Note that degrees of freedom were corrected using Greenhouse-Geisser when Mauchly's test of Sphericity indicated that sphericity had been violated.

3.3 RESULTS

3.3.1 Behavioural results

As in Experiment 1, behavioural analyses were conducted to examine children's explicit ability to imitate observed actions in each condition. These analyses were conducted by hand-coding the video recordings of each participant performing the imitation actions in each condition. The number of times a child performed the correct action during each 80 -second imitation period was counted, along with the number of incorrect actions performed in the same period.

Eight participants from the total experimental population did not consent to the use of video recording during the task. Therefore, the behavioural analyses were conducted on a smaller set of ten participants in the control group and eleven participants in the ASD group.

Correct imitations

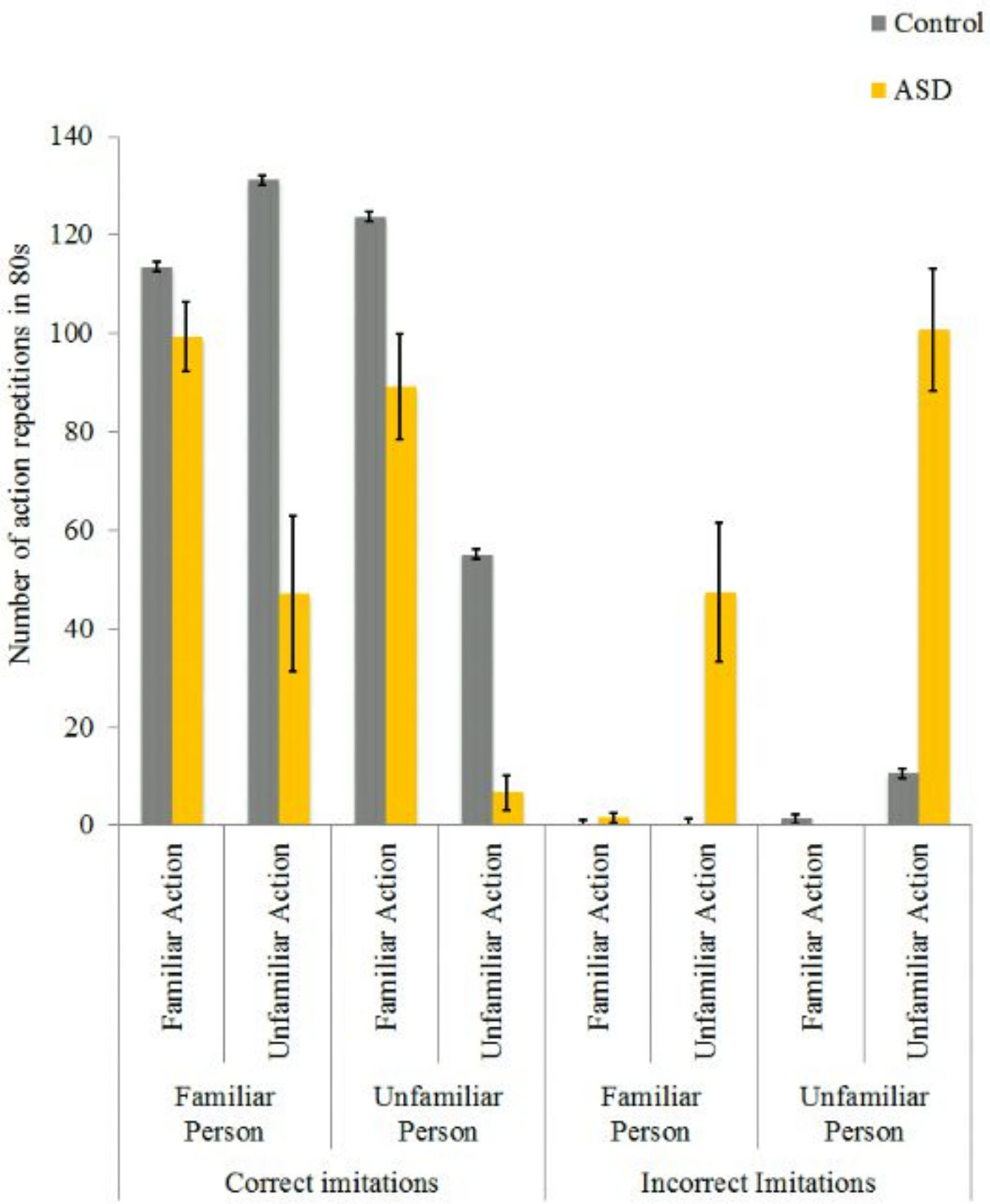

Figure 11 Mean number of correct and incorrect imitations for control and ASD groups for each of the four imitation conditions. Error bars represent the standard error of the mean.

Is imitating communicative gestures impaired in ASD?

A three-way mixed 2 x 2 x 2 ANOVA was conducted to examine the effects of person (familiar/ unfamiliar) and action (familiar/ unfamiliar) as within-subject factors and

group (control/ ASD) as a between-subject factor, on the number of correct action imitations. Results showed a number of significant effects and interactions; critically, the main effect of group was significant, $F(1, 19) = 21.55$, $p < .001$, $_p \eta^2 = .53$, demonstrating an increased number of correct imitations in the control group ($M = 105.90$), compared to the ASD group ($M = 60.54$).

Does person and action familiarity facilitate imitation performance?

A main effect of person, $F(1, 19) = 36.71$, $p < .001$, $_p \eta^2 = .65$, demonstrated an increased number of correct imitations for imitating an action performed first by a familiar person ($M = 97.81$), compared to imitating an action first performed by an unfamiliar person ($M = 68.63$). Further, a main effect of action, $F(1,19) = 186.21$, $p < .001$, $_p \eta^2 = .90$, demonstrated an increased number of correct imitations when participants were imitating a familiar action ($M = 106.43$), compared to imitating an unfamiliar action ($M = 60.00$).

In addition, a significant interaction was found between person*action, $F(1,19) = 16.14$, $p < .001$, $_p \eta^2 = .45$. Post hoc tests indicated significantly fewer correct imitations of an unfamiliar action when that action was performed by an unfamiliar person ($M = 29.71$), compared to a familiar person $M = 87.14$, $t(20) = 6.27$, $p < .001$, $d = 1.3$. In contrast, there was no significant difference in the number of correct imitations between familiar ($M = 106.14$) and unfamiliar persons ($M = 105.57$) when the action was familiar, $t(20) = .06$, $p = .95$, $d = .01$.

Finally, analyses revealed a significant interaction between group*action, $F(1, 19) = 37.84$, $p < .001$, $_p \eta^2 = .66$, and a marginal group*person*action interaction, $F(1, 19) = 3.74$, $p = .06$, $_p \eta^2 = .16$. The group*person interaction was not significant ($F < .63$, $p > .43$).

To examine these effects further, separate ANOVAs were conducted for control and ASD participants, comparing person (familiar/ unfamiliar) and action (familiar/ unfamiliar). Analysis of the control group showed a main effect of person, $F(1, 9) = 34.14$, $p < .001$, $_p \eta^2 = .79$, reflecting an increased number of correct imitations for imitating a familiar

person (M = 122.40) compared to an unfamiliar person (M = 89.40). A main effect of action, $F(1, 9) = 25.62$, $p < .001$, $_p\eta^2 = .74$, demonstrated an increased number of correct imitations for imitating a familiar action (M = 118.65) compared to imitating an unfamiliar action (M = 93.15). Furthermore, a significant interaction was found between person*action, $F(1, 9) = 29.52$, $p < .001$, $_p\eta^2 = .76$, showing no significant effect of performer familiarity when the action was familiar ($F < -.84$, $p = .41$), but the reverse pattern when the action was unfamiliar (familiar person: M = 131.20, unfamiliar person: M = 55.10), $t(9) = 11.03$, $p < .001$, $d = .3.5$.

Analysis of the ASD participants also showed a main effect of person, $F(1, 10) = 11.12$, $p = .008$, $_p\eta^2 = .52$, revealing an increased number of correct imitations of an action performed by a familiar person (M = 94.22) compared to an unfamiliar person (M = 26.86). The main effect of action, $F(1, 10) = 214.64$, $p < .001$, $_p\eta^2 = .95$, demonstrated an increased number of correct imitations for imitating a familiar action (M = 73.22) compared to imitating an unfamiliar action (M = 47.86). However, the interaction between person*action did not reach significance ($F < 1.65$, $p = .22$).

Incorrect imitations

Is imitating communicative gestures impaired in ASD?

A similar three-way mixed 2 x 2 x 2 ANOVA was conducted to examine the effects of person (familiar/ unfamiliar) and action (familiar/ unfamiliar) as within-subject factors, and group (control/ ASD) as a between-subject factor, on the number of incorrect action imitations. Results showed a number of significant effects. A main effect of group, $F(1, 19) = 65.10$, $p < .001$, $_p\eta^2 = .77$, demonstrated an increased number of incorrect imitations in the ASD group (M = 37.40), compared to the control group (M = 3.10).

Does person and action unfamiliarity have an effect on the number of incorrect imitations?

A main effect of person, $F(1, 19) = 7.12$, $p = .015$, $_p\eta^2 = .27$, demonstrated an increased number of incorrect imitations when individuals were imitating an unfamiliar person (M = 28.15), compared to imitating a familiar person (M = 12.35). A main effect of action, $F(1,19) = 80.61$, $p < .001$, $_p\eta^2 = .80$, demonstrated an increased number of incorrect

imitations when imitating an unfamiliar action (M = 39.79) compared to when imitating a familiar action (M = .71). In addition, a significant interaction between person*action, $F(1,19) = 7.11$, $p = .015$, $_p\eta^2 = .27$, showed significantly higher incorrect imitations for an unfamiliar action, compared to familiar actions, despite the familiarity of the performer (familiar person: $M = 25.04$ *vs* $M = .80$, $t(20) = -2.73$, $p = .013$, $d = -.06$, unfamiliar person: $M = 57.80$ *vs* $M = .61$, $t(20) = -4.60$, $p < .001$, $d = -1$). In addition, when looking at performer familiarity, unfamiliar person trials were relatively higher (M = 57.80) in incorrect imitations, compared to familiar person trials, M = 25.04, $t(20) = -2.62$, p = .017, $d = -.6$.

To follow up on this significant interaction, separate ANOVAs were conducted for control and ASD groups, comparing person (familiar/ unfamiliar) and action (familiar/ unfamiliar). Analysis of the control group showed no significant effects (Fs < 3.02, $ps > .11$). In contrast, analysis of the ASD group showed a significant main effect of person, $F(1, 10) = 5.68$, $p = .038$, $_p\eta^2 = .36$, reflecting an increased number of incorrect imitations when ASD participants were imitating an unfamiliar person (M = 50.36), compared to a familiar person (M = 24.45).

A main effect of action, $F(1, 10) = 90.96$, $p < .001$, $_p\eta^2 = .90$, was also significant in showing an increased number of incorrect imitations for imitating an unfamiliar action (M = 74.04), compared to a familiar action (M = .77). In addition, a significant interaction was found between person*action, $F(1, 10) = 6.33$, $p = .031$, $_p\eta^2 = .38$. This shows significant higher error rates for imitating unfamiliar actions, compared to familiar actions, despite the familiarity of the performer (familiar person :M = 47.36 *vs* M = 1.54, $t(10) = -3.21$, $p = .009$, $d = -.9$, unfamiliar person: M = 100.72 *vs* M = .000, $t(10) = -8.16$, $p < .001$, $d = -2.5$). In addition, when looking at performer familiarity, unfamiliar person trials were relatively higher in error rate, M = 100.72 vs M = .000, $t(10) = -2.45$, p = .034, $d = -.7$.

3.3.2 Electroencephalographic results

As in experiment 1, ERD was calculated using two frequency bands: alpha (8-12Hz) and

low beta (12-20Hz).

Alpha frequency band (8-12Hz)

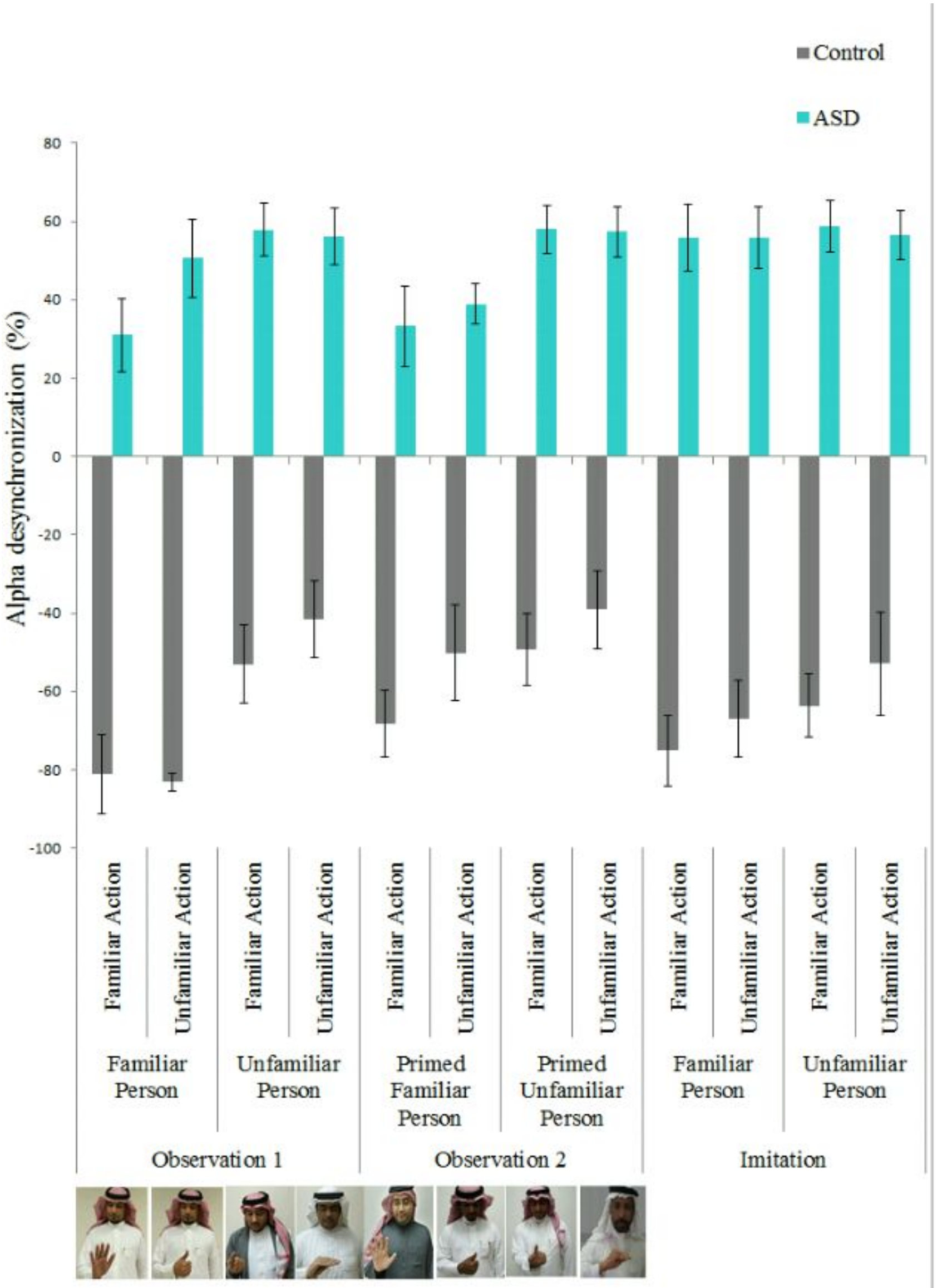
Control
ASD
Alpha desynchronization (%)
80
60
40
20
0
-20
-40
-60
-80
-100
Familiar Action
Unfamiliar Action
Familiar Person
Unfamiliar Person
Primed Familiar Person
Primed Unfamiliar Person
Observation 1
Observation 2
Imitation

Figure 12 The percentage of event-related changes in alpha power for control and ASD group across 12 conditions. Error bars represent the standard error of the mean.

A four-way mixed 2 x 2 x 2 x 3 ANOVA was conducted to examine the effects of person (familiar/ unfamiliar), action (familiar/ unfamiliar) and task (*observation 1*/ *observation 2*/ imitation) as within-subject factors, and group (control/ ASD) as a between-subject factor on alpha suppression[2]. Analysis revealed a number of significant effects and interactions which are represented in table 3.1.

	All data			Control group			ASD group		
	df	F-value	p-value	df	F-value	p-value	df	F-value	p-value
Group	1,27	90.95	0.001***	-	-	-	-	-	-
Person	1,27	164.57	0.001***	1,14	172.71	0.001***	1,13	37.37	0.001***
Action	1,27	30.60	0.001***	1,14	445.7	0.001***	1,13	2.15	0.16
Task	2,54	6.68	0.003**	1,14,9	32.18	0.001***	2,26	8.64	0.001***
Group*person	1,27	8.28	0.008**	-	-	-	-	-	-
Group*Action	1,27	6.51	0.017**	-	-	-	-	-	-
Group*Task	2,54	28.12	0.001***	-	-	-	-	-	-
person*Action	1,27	1.39	0.24	1,14	0.63	0.43	1,13	3.50	0.08
person*Task	1.2,33.9	20.51	0.001***	1,14.3	24.37	0.001***	1.2,15.6	9.70	.005**
Action*Task	1.4,38	0.60	0.49	1,14.1	1.36	0.26	2,26	2.26	0.12
Group*person*Action	1,27	4.16	0.051	-	-	-	-	-	-
Group*person*Task	2,54	10.03	0.001***	-	-	-	-	-	-
Group*Action*Task	2,54	2.82	0.06	-	-	-	-	-	-
person*Action*Task	1.2,33.4	0.59	0.48	1,14.2	3.48	0.08	1.3,18	1.73	0.20
Group*person*Action*Task	2,54	4.23	.019**	-	-	-	-	-	-

* for p<.05, ** for p<.01 and *** for p<.001.

Table 6 Mixed & separate ANOVAs for alpha suppression across person and task

Is MNs activity impaired in ASD?

A main effect of group, $F(1, 27) = 90.95$, $p < .001$, $_p\eta^2 = .77$, demonstrated an increased level of alpha suppression in the control group (M = -60.36%), compared to the ASD group (M = 50.86%).

Is MNs activity mediated by person and action familiarity?

A main effect of person, $F(1, 27) = 164.57$, $p < .001$, $_p\eta^2 = .85$, demonstrated an increased level of alpha suppression for a familiar person (M = -13.25%), compared to an unfamiliar person (M = 3.75%). A main effect of action, $F(1, 27) = 30.60$, $p < .001$, $_p\eta^2 = .53$, demonstrated an increased level of alpha suppression for a familiar action (M = -7.98%), compared to an unfamiliar action (M = -1.51%).

Separate repeated-measure ANOVAs were conducted for control and ASD participants, comparing person (familiar/ unfamiliar), action (familiar/ unfamiliar), and task (*observation 1*/ *observation 2*/ imitation). Analysis of the control group showed a main effect of person, $F(1, 14) = 172.71$, $p < .001$, $_p\eta^2 = .92$, which demonstrated an increased level of alpha suppression for a familiar person (M = -70.77%) compared to an unfamiliar person (M = -49.94%). A main effect of action, $F(1, 14) = 445.70$, $p < .001$, $_p\eta^2 = .97$, demonstrated an increased level of alpha suppression for a familiar action (M = -65.09%) compared to an unfamiliar action (M = -55.63%).

In comparison, analysis of the ASD group showed a main effect of person, $F(1, 13) = 32.37$, $p < .001$, $_p\eta^2 = .74$, which demonstrated an increased level of alpha suppression for a familiar person (M = 44.26%) compared to an unfamiliar person (M = 57.46%). Although the suppression of a familiar action was higher (M = 49.12) relative to an unfamiliar action (M = 52.61), it did not reach significant level ($F < 2.15$, $p > .16$).

Does priming by familiar person facilitate action understanding?

Planned comparisons (paired-samples t-tests), collapsed across action, were conducted to explore the two-way interaction between person and task in the control group. Comparisons revealed that the suppression for a familiar model was significantly higher

relative to an unfamiliar model at all three task levels: *observation 1* (familiar: M = -82.13%, unfamiliar: M = -47.32%), $t(14) = -8.44$, $p < .001$, $d = -2.18$, *observation 2* (familiar: M = -59.19%, unfamiliar: M = -44.20%), $t(14) = -16.48$, $p < .001$, $d = -4.25$, and imitation (familiar: M = -71.01%, unfamiliar: M = -58.31%), $t(14) = -9.30$, $p < .001$, $d = -2.40$.

Moreover, for a familiar person, comparisons across the three task levels revealed that the suppression during *observation 2* was significantly lower (M = -59.19%) relative to *observation 1* (M = -82.13%), $t(14) = -5.01$, $p < .001$, $d = -1.29$, and imitation (M = -71.01%), $t(14) = 10.50$, $p < .001$, $d = 2.71$. Similarly, for an unfamiliar model, comparisons across the three task levels revealed the same trend, in which the suppression of *observation 2* was significantly lower (M = -44.20%) relative to *observation 1* (M = -47.32%), $t(14) = -3.97$, $p = .001$, $d = -1.02$, and imitation (M = -58.31%), $t(14) = 11.90$, $p < .001$, $d = 3.08$.

AS for ASD, planned comparisons (paired-samples t-tests), collapsed across action, were conducted to explore the two-way interaction between person and task in the ASD group. Comparisons revealed that the alpha suppression of observing a familiar model was significantly higher relative to observing an unfamiliar model during two observation task levels: *observation 1* (familiar: M = 38.69%, unfamiliar: M = 52.91%), $t(13) = -5.96$, $p < .001$, $d = -1.59$, and *observation 2* (familiar: M = 32.04%, unfamiliar: M = 53.67%), $t(13) = -5.77$, $p < .001$, $d = -1.54$; however, comparison during imitation level did not elicit significance ($t < .86$, $p < .40$).

For a familiar person, comparisons across the three task levels revealed that the alpha suppression during imitation was significantly lower (M = 56.97%) relative to *observation 1* (M = 38.69%), $t(13) = -4.47$, $p = .001$, $d = -1.19$, and *observation 2* (M = 32.04%), $t(13) = -9.42$, $p < .001$, $d = -2.51$; however, alpha suppression of *observation 1* relative to *observation 2* did not elicit a difference ($t < 1.56$, $p < .14$). None of the comparisons for an unfamiliar person elicited significance ($t < -1.37$, $p > .19$).

Independent sample t-tests were run to explore the differences between the group effect of group*person*task interaction. As for familiar person, comparisons of *observation 1* demonstrated that alpha suppression of the control group (M = -82.13%) was significantly higher compared to the ASD group (M = 38.69%), $t(27) = -10.08$, $p < .001$, $d = 3.71$. Similarly, comparisons of *observation 2* demonstrated that alpha suppression of the control group (M = -59.19%) was significantly higher compared to the ASD group (M = 32.04%), $t(27) = -6.99$, $p < .001$, $d = 2.61$. In addition, comparisons of imitation demonstrated that alpha suppression of the control group (M = -71.00%) was significantly higher compared to the ASD group (M = 56.97%), $t(27) = -10.66$, $p < .001$, $d = 3.98$. As for unfamiliar person, comparisons of *observation 1* demonstrated that alpha suppression of the control group (M = -47.32%) was significantly higher compared to the ASD group (M = 52.91%), $t(27) = -7.49$, $p < .001$, $d = 2.79$. Similarly, comparisons of *observation 2* demonstrated that alpha suppression of the control group (M = -44.20%) was significantly higher compared to the ASD group (M = 53.67%), $t(27) = -7.53$, $p < .001$, $d = 2.80$. In addition, comparisons of imitation demonstrated that alpha suppression of the control group (M = -58.31%) was significantly higher compared to the ASD group (M = 55.15%), $t(27) = -8.55$, $p < .001$, $d = 3.20$.

Was MNs functioning influenced by IQ and hand imitation skills?

Correlational analyses revealed insignificant association between alpha suppression and intelligence quotient, $rs < -.49$, $ps > .12$. However, the data revealed significant association between behavioural imitation and alpha suppression for observation conditions, $r = .997$, $p < .001$, and imitation conditions, $r = -.666$, $p = .001$.

3.4 Low beta band (12-20Hz)

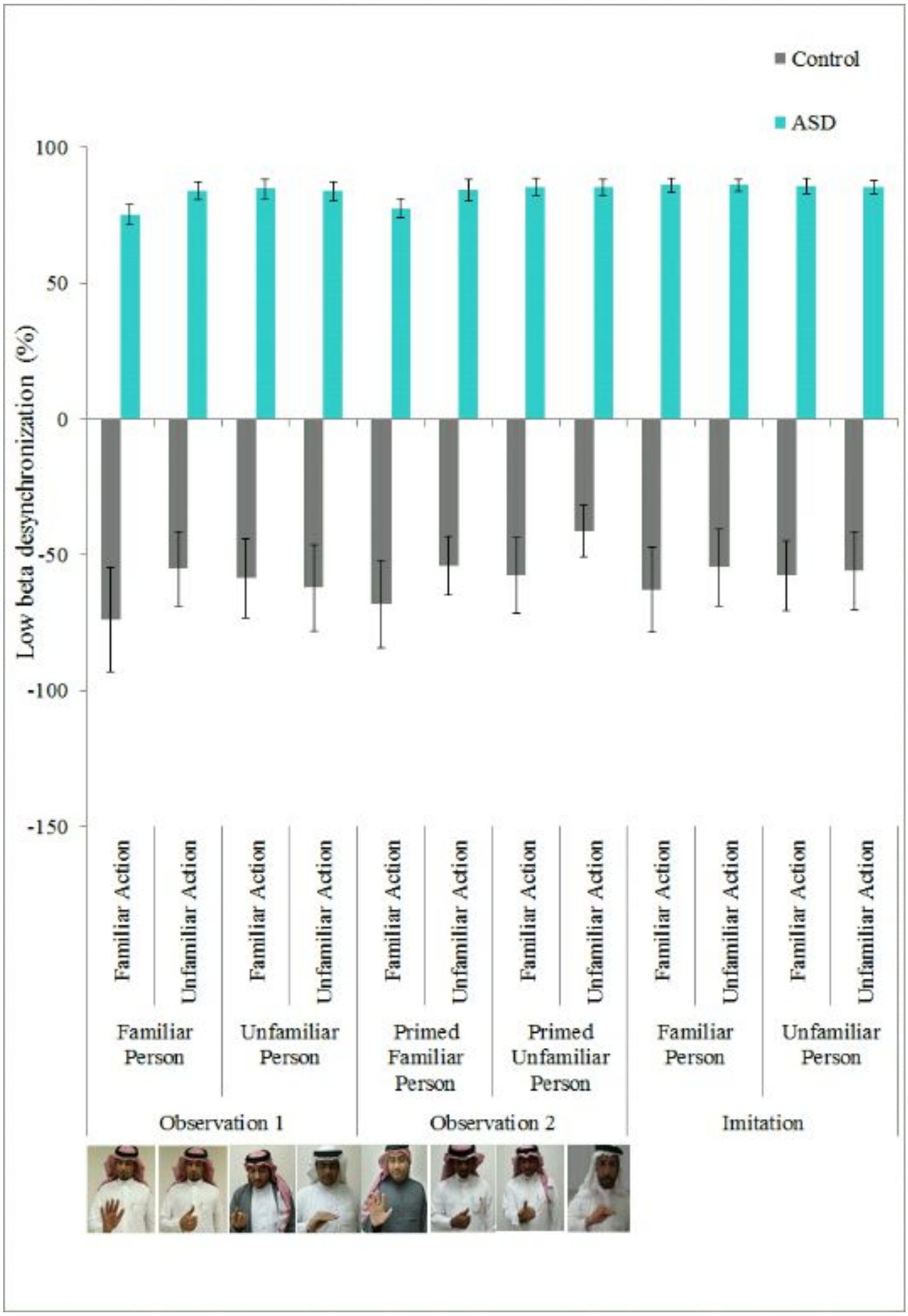

Figure 13 The percentage of event-related changes in low beta power for control and ASD group across 12 conditions. Error bars represent the standard error of the mean.

As in the alpha frequency band, a four-way mixed 2 x 2 x 2 x 3 ANOVA was conducted to

examine the effects of person (familiar/ unfamiliar), action (familiar/ unfamiliar), and task (*observation 1*, primed/ *observation 2*/ imitation) as within-subject factors, and group (control/ ASD) as a between-subject factor on low beta suppression[3]. Analysis revealed a number of significant effects and interactions which are represented in table 3.2.

	All data			Control group			ASD group		
	df	F-value	p-value	df	F-value	p-value	df	F-value	p-value
Group	1,27	89.93	0.001***	-	-	-	-	-	-
Person	1,27	28.26	0.001***	1,14	15.89	0.001***	1,13	22.84	0.001***
Action	1,27	21.70	0.001***	1,14	15.41	0.002**	1,13	18.95	0.001***
Task	1.2,31.2	7.51	.008**	1,14,1	5.05	.04*	1.3,17.2	13.93	0.001***
Group*person	1,27	3.64	0.06	-	-	-	-	-	-
Group*Action	1,27	7.53	.01**	-	-	-	-	-	-
Group*Task	2,54	3.62	0.03*	-	-	-	-	-	-
person*Action	1,27	12.84	0.001***	1,14	5.63	.03*	1,13	18.52	0.001***
person*Task	2,54	90.06	0.001***	1.4,20.1	168.86	0.001***	2,26	18.16	0.001***
Action*Task	1,28.1	6.34	.01*	1,14.1	4.36	0.055	2,26	8.11	.005**
Group*person*Action	1,27	0.58	0.45	-	-	-	-	-	-
Group*person*Task	2,54	25.99	0.001***	-	-	-	-	-	-
Group*Action*Task	2,54	2.52	0.12	-	-	-	-	-	-
person*Action*Task	1.2,32.9	18.83	0.001***	1,14.2	18.52	0.001***	2,26	6.32	.006**
Group*person*Action*Task	2,54	10.18	0.001***	-	-	-	-	-	-

* for p<.05, ** for p<.01 and *** for p<.001.

Table 7 Mixed & separate ANOVAs for low beta suppression across person, action and task.

Is MNs activity impaired in ASD?

A main effect of group, $F(1, 27) = 89.93$, $p < .001$, $_p\eta^2 = .76$, demonstrated an increased level of low beta suppression in the control group ($M = -58.48\%$), compared to the ASD group ($M = 83.67\%$).

Does person and action familiarity mediate MNs Activity?

Is MNs mediated by person and action familiarity?

A main effect of person, $F(1, 27) = 28.26$, $p < .001$, $_p\eta^2 = .51$, demonstrated an increased level of low beta suppression for a familiar person ($M = 10.42\%$) compared to an unfamiliar person ($M = 14.76\%$). A main effect of action, $F(1, 27) = 21.70$, $p < .001$, $_p\eta^2 = .44$, demonstrated an increased level of low beta suppression for a familiar action ($M = 9.69\%$) compared to an unfamiliar action ($M = 15.49\%$).

Separate repeated-measure ANOVAs were conducted for control and ASD participants, comparing person (familiar/ unfamiliar), action (familiar/ unfamiliar), and task (*observation 1*, primed/ *observation 2*/ imitation). Analysis of the control group showed a main effect of person, $F(1, 14) = 15.89$, $p = .001$, $_p\eta^2 = .53$, which demonstrated an increased level of low beta suppression for a familiar person ($M = -61.43\%$) compared to an unfamiliar person ($M = -55.53\%$).

A main effect of action, $F(1, 14) = 15.41$, $p = .002$, $_p\eta^2 = .52$, demonstrated an increased level of low beta suppression for a familiar action ($M = -63.09\%$) compared to an unfamiliar action ($M = -53.86\%$). In comparison to the control, analysis of the ASD group showed a main effect of person, $F(1, 13) = 22.84$, $p < .001$, $_p\eta^2 = .63$, which demonstrated an increased level of low beta suppression for a familiar person ($M = 82.28\%$) compared to an unfamiliar person ($M = 85.06\%$). A main effect of action, $F(1, 13) = 18.95$, $p = .001$, $_p\eta^2 = .59$, demonstrated an increased level of low beta suppression for a familiar action ($M = 82.47\%$) compared to an unfamiliar action ($M = 84.86\%$).

Is person familiarity is necessary to operate motor resonance in control? And does priming by familiar person facilitate action understanding?

Planned comparisons (paired-samples t-tests) were conducted to explore the three-way interaction between person, action, and task for the control group.

During *observation 1*, for a familiar person, comparisons revealed that low beta suppression was significantly higher for a familiar action (M = -73.95%), relative to an unfamiliar action (M = -55.09%), $t(14) = -3.38$, $p = .004$, $d = -0.87$. For an unfamiliar person, comparisons revealed that low beta suppression of a familiar action was significantly lower (M = -58.52%), relative to an unfamiliar action (M = -62.17%), $t(14) = 2.91$, $p = .011$, $d = 0.75$. Furthermore, when the action was familiar, the low beta suppression with a familiar person was significantly higher (M = -73.95%) relative to an unfamiliar person (M = -58.52%), $t(14) = -3.40$, $p = .004$, $d = -0.87$; however, for an unfamiliar action, the low beta suppression with a familiar person was significantly lower (M = -55.09%) relative to an unfamiliar person (M = -62.17%), $t(14) = 3.01$, $p = .009$, $d = 0.77$.

During observation 2, for priming by a familiar person, comparison revealed that the low beta suppression of a familiar action was significantly higher (M = -67.98%) relative to an unfamiliar action (M = -54.11%), t(14) = -2.52, p = .024, d = -0.65. For priming by an unfamiliar person, comparison revealed that the low beta suppression of a familiar action was significantly higher (M = -57.55%) relative to an unfamiliar action (M = -41.29%), $t(14) = -3.70$, $p = .002$, $d = -0.95$.

Furthermore, for a familiar action, the low beta suppression with an unfamiliar person that was primed by a familiar person was significantly higher (M = -67.98%) relative to an unfamiliar person that was primed by an unfamiliar person (M = -57.55%), $t(14) = -4.54$, $p < .001$, $d = -1.17$. Similarly, for an unfamiliar action, the low beta suppression with an unfamiliar person that was primed by a familiar person was significantly higher (M = -54.11%) relative to an unfamiliar person that was primed by an unfamiliar person (M = -41.29%), $t(14) = -9.84$, $p < .001$, $d = -2.54$.

During imitation, with a familiar person, comparison revealed significant low beta

suppression with a familiar action (*M* = -62.76%) relative to an unfamiliar action (*M* = -54.66%), *t*(14) = -5.18, *p* < .001, *d* = -1.33. Furthermore, with an unfamiliar person, comparison revealed that the low beta suppression with a familiar action was higher (*M* = -57.78%) relative to an unfamiliar action, but did not reach significance (*M* = -55.85%), (*t* < -1.18, *p* = .25). With a familiar action, comparison revealed that the low beta suppression with a familiar person was higher (*M* = -62.76%) relative to an unfamiliar person, but did not reach significance (*M* = -57.78%), (*t* < -1.60, *p* = .13). However, with an unfamiliar action, comparison revealed that the low beta suppression with a familiar person was significantly lower (*M* = -54.66%) relative to an unfamiliar person (*M* = -55.85%), *t*(14) = 3.28, *p* = .005, *d* = .85.

To further investigate the effect of priming during observation, we compared *observation 1* and *observation 2* in each trial, to see if there was any significant difference in the suppression. Results revealed one significant difference between the conditions that depicted an unfamiliar person performing an unfamiliar action (*observation 1*: M = -62.17%, *observation 2*: M = -41.29%), *t*(14) = -3.27, *p* = .006, *d* = -0.84. None of the remaining comparisons reached significant level (*ts* < -1.97, *ps* < .068).

Planned comparisons (paired-samples t-tests) were conducted to explore the three-way interaction between person, action and task for the ASD group.

During *observation 1*, with a familiar person, comparisons revealed that the low beta suppression with a familiar action was significantly higher (*M* = 75.35%) relative to an unfamiliar action (*M* = 84.18%), *t*(13) = -6.39, *p* < .001, *d* = -1.70. Furthermore, with a familiar action, comparison revealed that the low beta suppression with a familiar person was significantly higher (*M* = 75.35%) relative to an unfamiliar person (*M* = 84.77%), *t*(13) = -6.50, *p* < .001, *d* = -1.73. Interaction of person and action unfamiliarity revealed that none of the comparisons reached significance (*ts* < 1.24, *ps* < .23).

During observation 2, for priming by a familiar person, comparison revealed a similar trend of suppression to the control and the low beta suppression with a familiar action was significantly higher (M = 77.51%) relative to an unfamiliar action (M = 84.33%), $t(13) = -2.72$, $p = .017$, $d = -0.72$. Furthermore, when the action was familiar, the low beta suppression with an unfamiliar person that was primed by a familiar person was significantly higher (M = 77.51%) relative to an unfamiliar person that was primed by an unfamiliar person (M = 85.28%), $t(13) = -3.72$, $p = .003$, $d = -0.99$. Interaction of person and action unfamiliarity revealed that none of the comparisons reached significance ($ts < -.57$, $ps < .57$).

During imitation, interaction of person and action revealed that none of the comparisons reached significance ($ts < 1.28$, $ps < .22$).

To investigate the effect of priming, we compared *observation 1* with *observation 2* of each trial to see if there was any significant difference. Results revealed that none of the comparisons reached significant level ($ts < -1.89$, $ps < .081$).

Independent sample t-tests were run to explore the differences between the group effect of group*person*action*task interaction.

Comparisons of *observation 1* of familiar person performing familiar action demonstrated that low beta suppression of the control group (M = -73.95%) was significantly higher compared to the ASD group (M = 75.35%), $t(27) = -7.42$, $p < .001$, d = 2.80. Similarly, comparisons of *observation 2* of familiar person performing familiar action demonstrated that low beta suppression of the control group (M = -67.98%) was significantly higher compared to the ASD group (M = 77.51%), $t(27) = -8.53$, $p < .001$, d = 3.22. In addition, comparisons of an imitation of familiar person performing familiar action demonstrated that low beta suppression of the control group (M = -62.76%) was significantly higher compared to the ASD group (M = 86.12%), $t(27) = -9.02$, $p < .001$, d = 3.41.

Comparisons of *observation 1* of familiar person performing unfamiliar action demonstrated that low beta suppression of the control group (M = -55.09%) was significantly higher compared to the ASD group (M = 84.18%), $t(27) = -9.66$, $p < .001$, d = 3.64. Similarly, comparisons of *observation 2* of familiar person performing unfamiliar

action demonstrated that low beta suppression of the control group (M = -54.11%) was significantly higher compared to the ASD group (M = 84.33%), $t(27) = -11.86$, $p < .001$, d = 4.47. In addition, comparisons of an imitation of familiar person performing unfamiliar action demonstrated that low beta suppression of the control group (M = -54.66%) was significantly higher compared to the ASD group (M = 86.16%), $t(27) = -9.47$, $p < .001$, $d = 3.58$.

Comparisons of *observation 1* of unfamiliar person performing familiar action demonstrated that low beta suppression of the control group (M = -58.52%) was significantly higher compared to the ASD group (M = 84.77%), $t(27) = -9.23$, $p < .001$, d = 3.48. Similarly, comparisons of *observation 2* of unfamiliar person performing familiar action demonstrated that low beta suppression of the control group (M = -57.55%) was significantly higher compared to the ASD group (M = 85.28%), $t(27) = -9.69$, $p < .001$, d = 3.66. In addition, comparisons of an imitation of unfamiliar person performing familiar action demonstrated that low beta suppression of the control group (M = -57.78%) was significantly higher compared to the ASD group (M = 85.80%), $t(27) = -10.70$, $p < .001$, d = 4.04.

Comparisons of *observation 1* of unfamiliar person performing unfamiliar action demonstrated that low beta suppression of the control group (M = -62.17%) was significantly higher compared to the ASD group (M = 83.92%), $t(27) = -8.70$, $p < .001$, d = 3.28. Similarly, comparisons of *observation 2* of unfamiliar person performing unfamiliar action demonstrated that low beta suppression of the control group (M = -41.29%) was significantly higher compared to the ASD group (M = 85.17%), $t(27) = -12.27$, $p < .001$, $d = 4.63$. In addition, comparisons of an imitation of unfamiliar person performing unfamiliar action demonstrated that low beta suppression of the control group (M = -55.85%) was significantly higher compared to the ASD group (M = 85.40%), $t(27) = -9.44$, $p < .001$, $d = 3.56$.

Was MNs functioning influenced by IQ and imitation skills?

Correlational analyses revealed insignificant association between low beta suppression and intelligence quotient, $rs < .07$, $ps > .75$. However, there was significant association between behavioural imitation and low beta suppression for imitating conditions, r = -.664, p = .001, and observation conditions, r= .999, p = .001. Both revealed a large correlation.

3.5 DISCUSSION

The aim of this experiment was to investigate whether priming an unfamiliar model (adult male stranger) with a familiar model (parent) elicits qualitatively improved MN activation when observing communicative hand gestures in children with ASD?

In accordance with our predictions, current EEG data revealed the main effect of group was significant, and thus, children with ASD demonstrated a decreased level of MNs activation relative to TD children as reflected in their alpha and low beta suppression. Similarly, current behavioural data revealed the main effect of group, demonstrating less correct hand actions in ASD relative to TD children. As predicted, and as previous literature established (Buccino et al., 2001), both groups demonstrated decreased level of MNs activation in alpha and low beta bands, while observing an unfamiliar – meaningless - action. In addition, as predicted and as previous work showed (Oberman et al., 2008), both groups demonstrated increased level of MNs activation while observing a familiar model compared to a stranger model. The level of MNs activation when observing a familiar model performing a familiar action was, consistent with our prediction, higher than if the familiar model was performing an unfamiliar action.

The novel result that we aimed for, interestingly, led to our touching upon new findings in relation to person and action familiarity interactions; in particular, control children demonstrated the highest level of suppression for simulating their parent while performing a familiar action and a reasonable capacity to simulate and imitate an unknown model when he was performing a familiar action.

They were further able to simulate and imitate a familiar model while performing a ‘meaningless’ action. Unlike the control children, the ASD children were impaired at simulating an unknown person, even when he was performing a familiar action. However, children with ASD showed a reasonable capability to simulate their parents, eliciting the greatest suppression, the highest number of correct imitations and the lowest number of imitative errors for observing two familiar properties (person, action). Both EEG and behavioural profiles suggested a reduced capacity to simulate a ‘

meaningless ʾaction; observing a parent performing a ʿmeaningless ʾaction was less likely to trigger any simulation of that action.

In accordance with our hypothesis, we approached the main question by comparing the level of suppression during the first observation period (*observation 1*), which either depicted familiar or unfamiliar models, with the level of suppression during the second observation period (*observation 2*), which, across all conditions, made use of unfamiliar models. If the results revealed significantly lower suppression for *observation 2*, relative to *observation 1*, this would potentially suggest that observing a prime familiar model did not facilitate simulating unfamiliar models. However, non-significant comparisons would suggest that the observing of a prime familiar model would facilitate the simulation of unfamiliar models by eliciting similar levels of suppression. The results reported here revealed a significant difference in both alpha and low beta suppression during *observation* 2 for familiar versus unfamiliar person conditions. Moreover, suppression was not significantly different when observing a (primed) unfamiliar model (*observation 2*) compared to when observing the familiar model (*observation 1*).

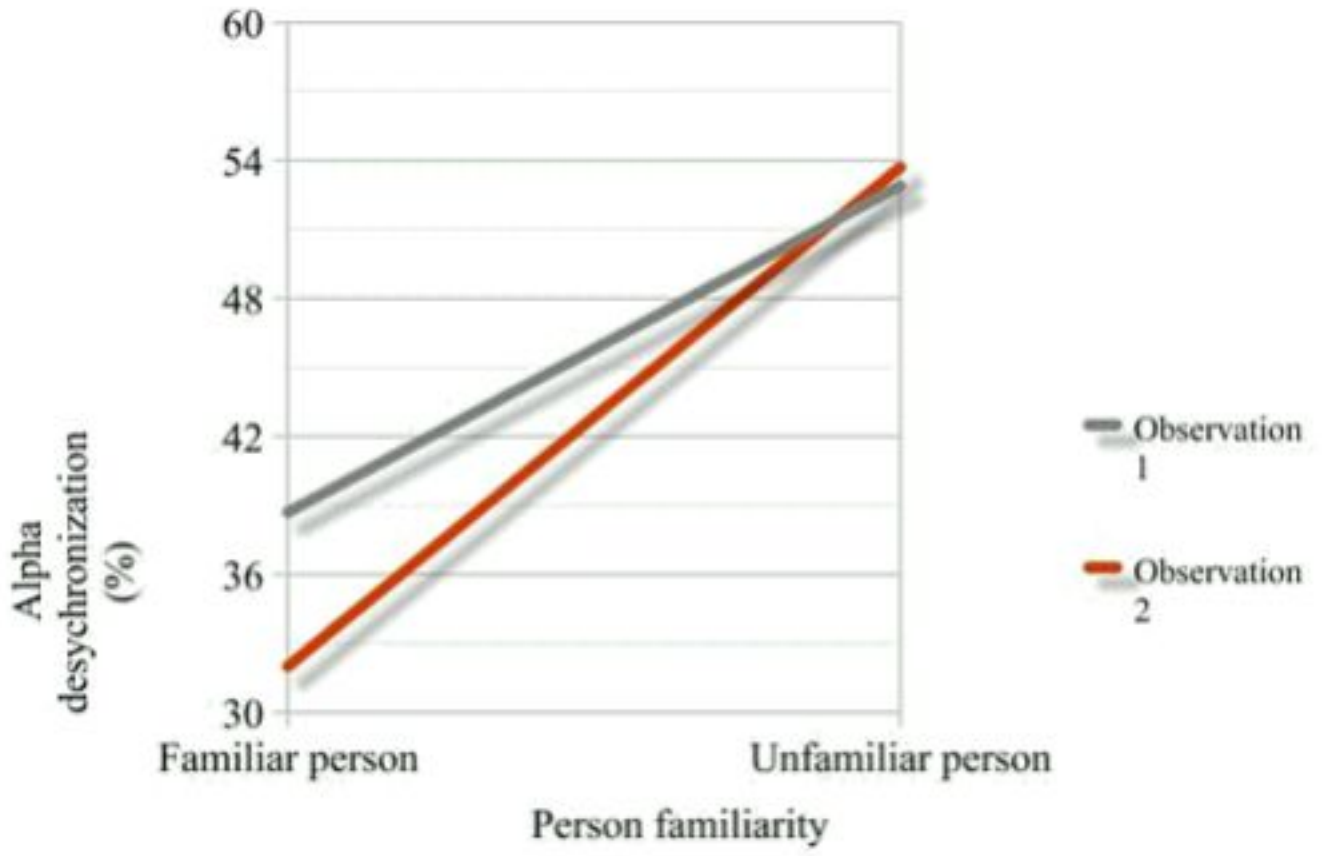

Figure 14 Line graph represents the comparison of alpha suppression between observation 1 & observation 2 for a familiar and unfamiliar person for the ASD group.

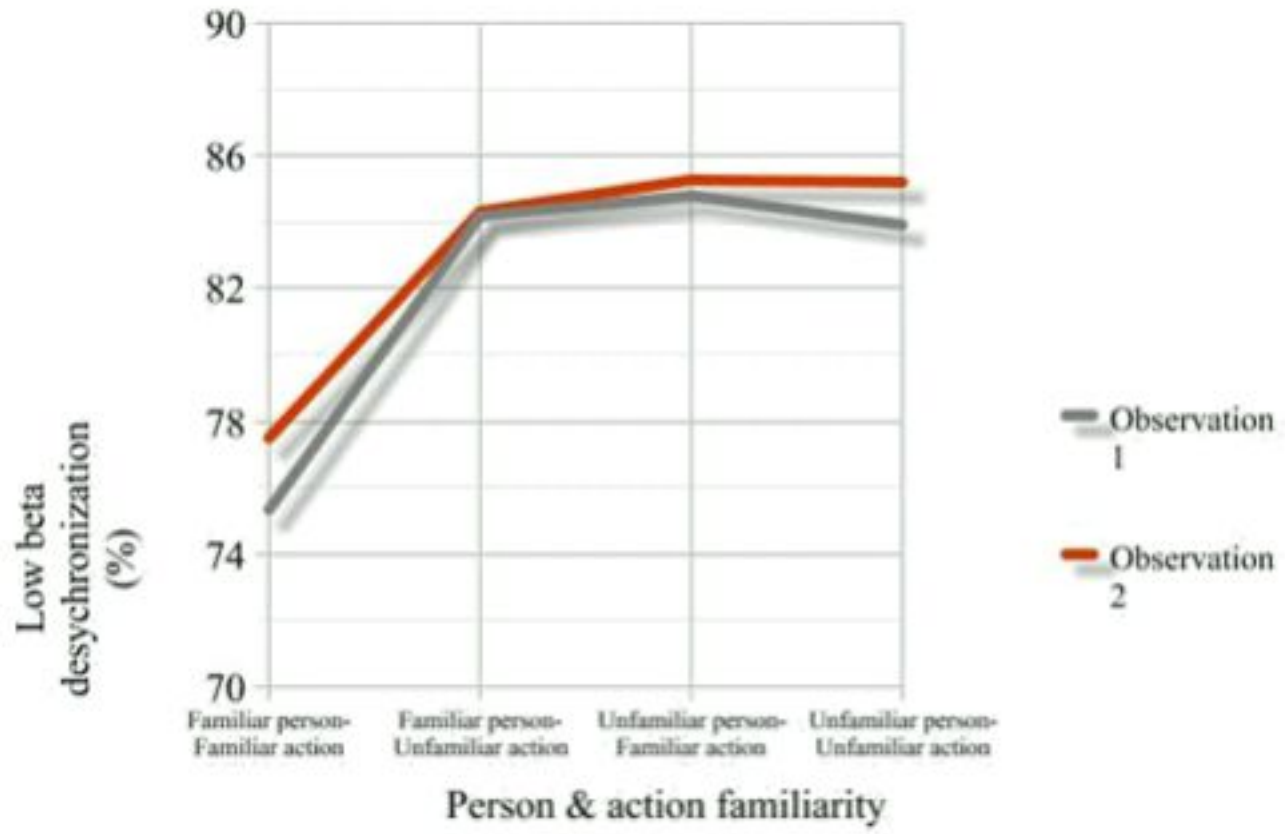

Figure 15 Line graph represents the comparison of low beta suppression between 4 observing periods of observation 1 & observation 2 for the ASD group.

It is important to note that presenting stimuli twice, despite manipulating the model familiarity, could arguably facilitate the suppression in the second observation period (*observation 2*), thus leading to improved imitation. However, the review of the neurophysiological and behavioural profiles of ASD participants suggests the inaccuracy of this argument. Comparing the level of suppression related to a familiar person with an unfamiliar person across the three task levels (*observation 1*, *observation 2*, and Imitation) implies that all task levels related to a familiar person led to significantly higher suppression than those with an unfamiliar person.

Furthermore, evaluating the data of low beta suppression (where task, person and action interacted in both groups) showed a similar effect, whereby both *observation 1* and *observation 2* elicited significantly higher suppression for ‘familiar person - familiar action’ scenarios compared to ‘unfamiliar person - unfamiliar action’ scenarios as displayed by Figure 3.14. Nonetheless, the imitation period did not show a significant difference in low beta, which initially appeared to suggest that the effect of familiarity was limited to action observation, but not imitation.

~~In fact, the behavioural imitation findings were consistent with low beta suppression in~~

eliciting a significant increase of the number of correct imitations for observing a ‘familiar person - familiar action’ and a significant increase in the number of incorrect imitations for observing an ‘unfamiliar action - unfamiliar person’, as displayed by Figure 3.15. Thus, both illustrated an increased level of imitation; however, each increase was qualified in a reverse way.

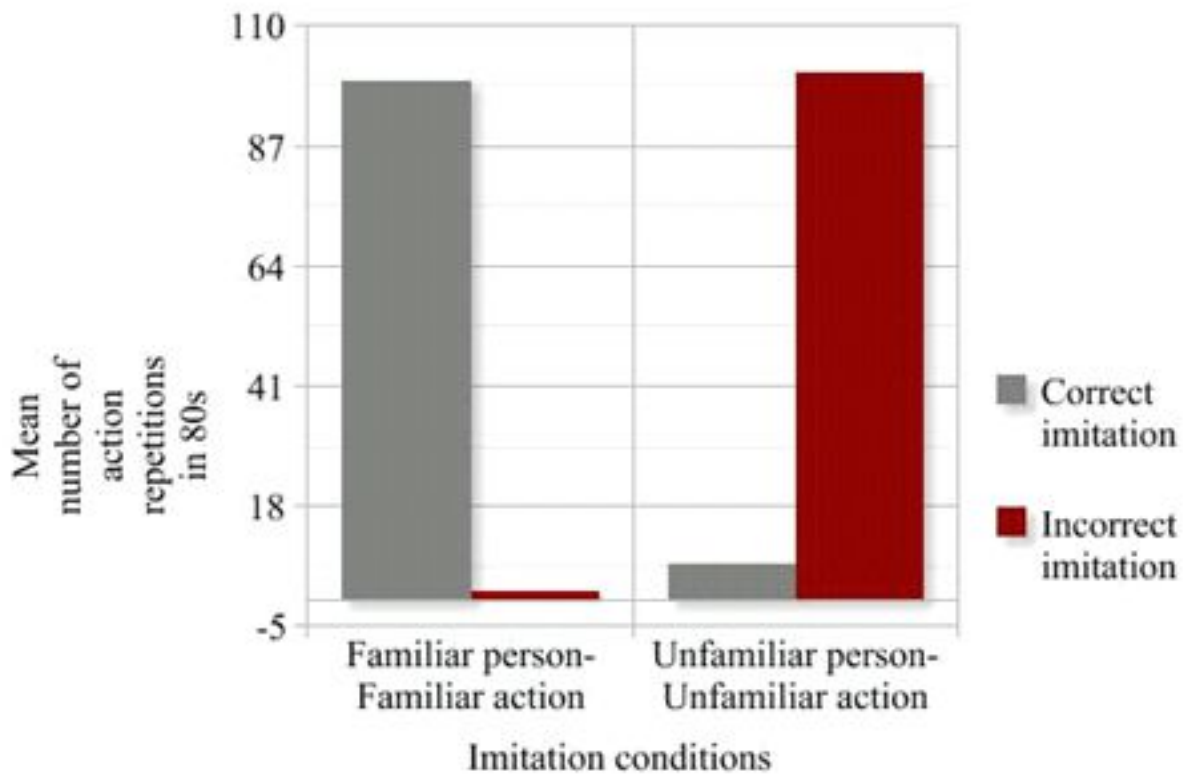

Figure 16 Mean number of correct and incorrect imitations for the ASD group for ‘familiar person - familiar action’ and ‘unfamiliar person - unfamiliar action’

One possible concern may arise regarding the independence of the effect of action familiarity, in isolation from the effect of person familiarity, as this directly concerns the usefulness of person familiarity involvement. In other words, was it possible for the Saudi children, from both populations, to operate motor resonance effectively, in spite of the actor’s familiarity? If the effects were being driven solely by the action familiarity, this statistically should be revealed as a significant difference in familiarity of the action, when controlling the person familiarity. In particular, it should reveal significant difference when putting the ‘unfamiliarity of the person’ under control, and manipulating the familiarity of the action.

The ASD group revealed that this is not the case, however; when putting the ‘person familiarity’ under control, and manipulating the familiarity of action, the difference was ~~significant. In addition, analysis of the ASD group only showed a main effect of~~ action

familiarity in the low beta band, and not in the alpha band. In contrast, the main effect of person familiarity was significant in both bands. This suggests that when children with ASD are undergoing a cognitive task that requires them to infer a meaning, intention or goal for a communicative gesture is strongly influenced by the person familiarity and motor resonance that seem to have a reciprocal effect. Hence, if the demonstrator is a familiar person, the ASD child will find it easier to simulate the actor; conversely, when the demonstrator is not familiar to the child, the child finds it harder to simulate the actor.

In contrast to the ASD participants, the control group revealed an overall main effect of action familiarity and person familiarity in both alpha and low beta bands. Here, undergoing a cognitive task that required the TD child to infer a meaning, intention or a goal for a communicative gesture is more likely to be modulated if it was performed by a well-known person; however, the child is still capable of simulating an unknown person. This finding is in agreement with previous research (e.g. Oberman et al., 2008; Wolff & Barlow, 1979) in which the familiarity of the actor was found to modulate the neural suppression of both TD and ASD children.

Taken together, these findings provide a clear distinction of how familiar actions' resonance operates in relation to the actor familiarity between ASD and TD groups. This seems in parallel with the proposal of Sai (2005) in showing that since birth, children show a preference for looking at their mother's face compared to a stranger, since the majority of their time is spent in their parent's presence. The effect of early exposure and experience is likely to drive this effect in children, showing better simulation of their parent compared to a stranger, and this also could be explained by the argument that people tend to sympathise more with known, and in-group people, compared to unknown and out-group members (Hornstein, 1976).

These effects in EEG and behavioural performance synchronise with the clinical assessment, in which TD children, whose ADI-R manifests in the normal social, communicative and behavioural domain, did not appear to experience difficulty simulating an unknown adult, but they engaged more with observing one of their parents performing a communicative gesture, which subsequently reflected on their

high rate of correct imitation. However, ASD children, showed the reverse effect in which all three domains were evidently impaired.

3.6 CONCLUSION

The current results are consistent with previous literature and the results from Experiment 1 in validating the critical effect of person familiarity in children with ASD (Oberman et al., 2008). Crucially, the results extend this previous research by showing a facilitated capacity to 'simulate' a stranger, in *observation 2* conditions, while performing a familiar hand action, when these conditions were first performed by the child's parent. This suggests that the difficulty associated with simulation can be reduced by first priming that action with a familiar person.

The other major finding was seen in the reduced capacity to simulate a familiar action in children with ASD when it was not supported by person familiarity cues. This effect in the ASD group distinguished them from the TD children, whose ability to imitate an action was also influenced by person familiarity, but for whom the effects of action and person familiarity occurred independently from each other. Finally, the EEG and behavioural profiles of TD and ASD children suggested a low capacity to simulate 'meaningless' (unfamiliar) hand gestures; even observing a parent performing 'meaningless' gestures was less likely to trigger action reasoning to that action.

Footnote

[1] Analysis of group differences in the baseline condition demonstrated significant difference on the alpha frequency band for C3, $t(27) = 2.52$, $p = .018$, $d = 0.35$, Cz, $t(27) = 2.49$, $p = .019$, $d = 0.35$, and C4, $t(27) = 2.50$, $p = .019$, $d = 0.35$. However, after excluding the significant data of two cases, the baseline analyses showed insignificant difference ($t < 1.96$, $p > .61$). In addition, when conducting analyses on the alpha frequency band, after excluding the two significant cases, the main effect of group, $F = 90.59$, $p < .001$, $_p\eta^2 = .78$, and the significant interaction of person*task in the control group, $F = 20.69$, $p < .001$, $_p\eta^2 = .63$, and ASD group, $F = 9.70$, $p = .001$, $_p\eta^2 = .42$, remained consistent with the p values of full sample analyses. As for the low beta frequency band, analysis of group differences in the baseline condition did not reach significance ($t < .28$, $p > .77$).

[2] When we conducted analyses on the alpha frequency band in the sub-samples, the main effect of group (full sample: $F = 21.55$, $p < .001$, $_p\eta^2 = .53$/ sub-sample: $F = 52.81$, $p < .001$, $_p\eta^2 = .73$), remained consistently significant; however, the effect size associated with the main effect in the full-sample analysis was moderate, whereas the effect size associated with the main effect in the sub-sample analysis was large, which indicated a stronger effect. . The main effect of person (full sample: $F = 164.57$, $p < .001$, $_p\eta^2 = .85$/ sub-sample: $F = 116.90$, $p < .001$, $_p\eta^2 = .86$) remained consistently significant and yielded the same range of large effect size. The main effect of action (full sample: $F = 30.60$, $p < .001$, $_p\eta^2 = .53$/ sub-sample: $F = 18.41$, $p < .001$, $_p\eta^2 = .49$) remained consistently significant and yielded the same range of moderate effect size. As for the control group, the main effect of person (full sample: $F = 172.71$, $p <$

.001, $_p\eta^2$ = .92/ sub-sample: *F*=126.19, *p* < .001, $_p\eta^2$ = .93) and action (full sample: *F*= 445.70, *p* < .001, $_p\eta^2$ = .97/ sub-sample: *F*= 256.14, *p* < .001, $_p\eta^2$ = .96) remained consistently significant and yielded the same range of large effect size. As for ASD, the main effect of person (full sample: *F*= 32.37, *p* < .001, $_p\eta^2$ = .74/ sub-sample: *F*= 28.93, *p* < .001, $_p\eta^2$ = .74) remained consistently significant and yielded the same range of large effect size.

In addition, interaction between person*task for the control group (full sample: *F*= 24.37, *p* < .001, $_p\eta^2$ = .63/ sub-sample: *F*= 19.36, *p* = .002, $_p\eta^2$ = .63) remained consistently significant and yielded the same range of moderate effect size. Although the ASD group (full sample: *F*= 9.70, *p* = .005, $_p\eta^2$ = .42/ sub-sample: *F*= 5.47, *p* = .037, $_p\eta^2$ = .35) showed that the effect size of sub-sample analysis tended to be smaller than full sample analysis, both effect size are within small range. Therefore, there seems to be no significant difference between the value of full-sample and sub-sample analysis in the alpha frequency band.

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[3] When we conducted analyses on the low beta frequency band in the sub-samples, the main effect of group (full sample: *F*= 89.93, *p* < .001, $_p\eta^2$ = .76/ sub-sample: *F*= 59.70, *p* < .001, $_p\eta^2$ = .75) remained consistently significant and yielded the same range of large effect size. The main effect of person (full sample: *F*= 28.26, *p* < .001, $_p\eta^2$ = .51/ sub-sample: *F*= 19.27, *p* < .001, $_p\eta^2$ = .50) remained consistently significant and yielded the same range of moderate effect size. The main effect of action (full sample: *F* = 21.70, *p* < .001, $_p\eta^2$ = .44/ sub-sample: *F*=14.19, *p* = .001, $_p\eta^2$ = .42) remained consistently significant and yielded the same range of small effect size. As for the control group, the main effect of person (full sample: *F*= 15.89, *p* = .001, $_p\eta^2$ = .53/ sub-sample: *F*= 8.21, *p* = .019, $_p\eta^2$ = .47) and action (full sample: *F*= 15.41, *p* = .002, $_p\eta^2$ = .52/ sub-sample: *F*= 7.98, *p* = .020, $_p\eta^2$ = .47) remained consistently significant and yielded the same range of moderate effect size. As for ASD, the main effect of person (full sample: *F*= 22.84, *p* < .001, $_p\eta^2$ = .63/ sub-sample: *F*= 17.31, *p* = .002, $_p\eta^2$ = .63) and action (full sample: *F*= 18.95, *p* = .001, $_p\eta^2$ = .59/ sub-sample: *F*= 13.43, *p* =.004, $_p\eta^2$ = .57) remained consistently significant and yielded the same range of moderate effect size.

In addition, interaction between person*action*task for the control group (full sample: *F*= 18.52, *p* = .001, $_p\eta^2$ = .57/ sub-sample: *F*= 9.73, *p* = .012, $_p\eta^2$ = .52) remained consistently significant and yielded the same range of moderate effect size as well. The main effect of the ASD group (full sample: *F*= 6.32, *p* = .006, $_p\eta^2$ = .32/ sub-sample: *F*= 3.87 *p* = .038, $_p\eta^2$ = .27) remained consistently significant and yielded the same range of small effect size. Therefore, there seems to be no significant difference between the value of full-sample and sub-sample analysis in the low beta frequency band.

4 The effect of social characteristics on action understanding and imitation: age similarity

4.1 EXPERIMENT 3

Peer modelling incorporates a number of instructional techniques in which physical adjustment of an environment is arranged which include a child, live or televised, demonstrating target behaviour for other children who are less skilled, to encourage imitation of that behaviour (Strain, 1981). Recent research has demonstrated great support for the facilitatory effect of 'peer-modelling' in supporting academic achievement (Utley et al., 1997), behavioural change and social skills (McConnell, 2002). As one example of conducted 'peer-modelling' intervention, young mentally retarded children were found to increase their social reciprocity after observing peer modelling play behaviour in which they were prompted and reinforced for imitating that behaviour (Apolloni et al., 1977).

Introducing peers - 'child-sibling'- in therapeutic intervention has been shown to lead to critical improvement in the functional skills of siblings with disabilities (Cash & Evans, 1975) and siblings with ASD (Colletti & Harris, 1977). Schreibman et al. (1983) investigated the efficiency of teaching some behaviour modification techniques to normal siblings of ASD children. Normal siblings were then required to teach their ASD siblings some learning tasks. Their data revealed improvements in ASD behaviour, and a decrease in negative statements made by normal children about their ASD siblings after the training.

These findings were not solely based on 'live' peer-modelling; televised peer-modelling was found to be similarly effective. O'Conner (1972) found that socially withdrawn children demonstrated increased social interactions after presenting them with televised peers demonstrating appropriate social behaviour. It is further seen that improvement of play and level of activity is correlated with the level of peers' involvement (e.g., Kern & Aldridge, 2006). Studies such as this notably endorse the influence of peer models who can offer the most potential as part of an intervention

strategy to prompt the use of motor and social skills in children with ASD (Chan et al., 2009). The primary interest here lies in reflecting on the mechanisms underpinning these peer mediation effects on action understanding and imitation.
Bandura (1977) suggested that neural activation is selective and that the child will be more influenced by those who symbolise engaging qualities.

This is explained by the notion that the characteristics of an observed model (e.g., age, ethnic status) influence the degree to which social attitudes and behaviour will be produced by others (Epstein, 1966). Children appear to consider their peers as potential social partners, thus, increasingly, they start to integrate their activities with unfamiliar peers and imitate their actions (Maudry & Nekula, 1939; Harlow, 1969), which allows them to elaborate on a social engagement with an unfamiliar 'peer,' compared to a familiar 'mother' (Eckerman et al., 1975).

Meltzoff (1990) proposed that this tendency synchronises the 'like-me' view, in which peer preference stems from childrens' ability to recognize others as being similar to the self, which in turn requires the linking of an observed action to the self to understand others' actions and mental states.

Nonetheless, to date, no attempt has been made to investigate how age similarity might influence the neural activations of MNs in TD children and children with ASD.
Therefore, the current experiment utilises the same experimental paradigm as described in Experiment 2, and aims to investigate two key questions:

1) *Do children with or without ASD show evidence of enhanced action understanding when observing a similar-aged person (child) performing a hand gesture, compared to observing a dissimilar-aged person (adult) performing the same gesture?*
2) *If the effect of age similarity is present in children with ASD, can this similar-age facilitation effect be used to prime qualitative changes in behaviour when observing a dissimilar-aged person performing a hand gesture?*

As discussed in Chapter 1, it has been established that neural oscillations at different EEG frequencies change with age from infancy to at least 16 years (Somsen et al., 1997; see Chapter 1, section 1.6). These findings seem qualitatively consistent with results from a recent study of the developmental course of mu suppression across the age span, in which mu rhythm suppression appeared to increase with age (Oberman et al., 2012). Such an effect suggests that mu frequency could be more suited for investigations of action observation in older childhood and adults, compared to younger children (for a review, see Oberman et al., 2012).

Lepage and Théoret (2006) examined the mu rhythm amplitude over central sites in a group of children aged between 4 and 11 years old, during a task that required execution and observation of grasping hand actions. The pattern of mu rhythm suppression during action execution was similar to that found during observation of the same action. Interestingly, Lepage and Théoret conducted additional analyses of the same data, focusing on two theta frequency ranges to investigate the neural activity during execution and observation conditions in young children: 3.5 –5.5Hz and 5.5 – 7.5Hz. Results showed that theta modulation, in contrast to the mu rhythm, did not qualitatively show neural activity during action execution, only during action observation.

Experimental conditions seem to require continuous attention in order to perform a cognitive task. This cognitively demanding skill has been reported to entail specific rhythm at midline Cz and Fz leads (the frontal-midline theta rhythm). This rhythmic wave is observed in the frequency band of 5.5-7.5 Hz. The generator source of theta was demonstrated through electrophysiological studies in the lateral frontal cortex and the medial prefrontal cortex including the anterior cingulate cortex (Miruki et al., 1980).

Kawamata et al (2007), in their EEG study with normal adults, investigated the ERS/ ERD of the frontal-midline theta rhythm (5.5-7.5 Hz) during joint tasks of videogames and task-irrelevant auditory stimuli. They found that theta was desynchronized by the auditory stimuli. They hypothesized that the ERD of theta was

either reflecting the mental inhibition to the attentional resources from being unnecessarily allocated to those stimuli, or it was reflecting the information processing mediated by multi-item working memory requirements for playing the videogame and the simultaneous auditory processing.

Studies of theta rhythm have also appeared in 'perceptual narrowing' in infants and preschool children. Perceptual narrowing is a developmental process in which the perceptual abilities are shaped and mediated by environmental experience; therefore, perception of properties that we are usually exposed to are better in comparison to those that we have rarely experienced (Scott & Monesson, 2010).

For instance, a 12 month old infant's ability to discriminate native sound increases, while performance on non-native sound discrimination declines (Kuhl et al., 2006). The most commonly proposed mechanism underlying perceptual narrowing is executive function (see Diamond, 1994). Executive functions appear to control attention and inhibition. Researchers have found that the decline in the perception of non-native phonemes in infancy is significantly correlated with the growth of attentional control skills; upon this finding, it has been proposed that this correlation mirrors the individual ability to ignore less relevant information and attend to relevant information. EEG and MEG literature indicates that brain oscillations in the theta band index the control of attention and cognitive effort (Klimesch, 1999).

However, there has been a limited amount of research that employs theta frequency band as an index to detect the MNs activation in children participants. Therefore, *the work in this chapter aims to incorporate analyses of activity in the theta frequency band (5.5 –7.5Hz) as an extra index of action understanding and its underlying neural mechanisms*.

The current experiment adopts a similar design to that used in Experiment 2, where two groups of children (ASD and control) watched 4 pairs of videos that depict actors performing a simple hand action, then imitated the action after each video pair. Crucially, in this experiment, we cross age similarity (similar aged actor (child) *vs*. dissimilar aged

actor (adult), with action familiarity (familiar *vs*. unfamiliar). As in the previous experiments, video recordings of the children ś imitation performance were collected for later coding and analysis. Given the existing behavioural literature described above, which has suggested a great potential for facilitating of performance via a similar-age effect, we hypothesized that:

1) ASD children will demonstrate a decreased level of MNs activation relative to that in TD children
2) ASD children will demonstrate fewer correct imitative performances relative to TD children.
3) ASD and TD children will demonstrate a decreased level of MNs activation for observing an unfamiliar action relative to observing a familiar action.
4) ASD and TD children will demonstrate an increased level of MNs activation for observing a similar-aged model, relative to a dissimilar-aged model.
5) Level of MNs activation for observing a similar-aged person performing a familiar action will be higher relative to an unfamiliar action in TD and ASD children
6) The neural activation with a dissimilar-aged model will be improved, if that action was primed by a similar-aged model.

In addition, in the light of the developmental course of EEG frequencies, we will investigate whether activity in the theta band range will yield novel insights into the underlying neural activity as well as those in the alpha and low beta bands. This simulation will be indexed by the ERD of both the alpha and low beta frequency bands, as evidence of MNs involvement.

4.2 METHODS

4.2.1 Participants

The participants originally comprised 17 control children and 16 children diagnosed with ASD; they ranged in age from 4 years to 5 years/4 months. The ASD participants were recruited from the Autism Research and Treatment Center at King Khaled Hospital

in Riyadh, Saudi Arabia. The control participants were recruited from selected nurseries in Riyadh, Saudi Arabia. All participants had normal or corrected-to-normal vision prior to testing. Autistic participants who had comorbid neurological conditions or full scale IQ<80 were excluded.

	Control group	ASD group	
Full sample	(*n*= 16)	(*n*= 14)	
Age (*M*/*SD*)	4.5 ± 0.3	4.5 ± 0.3	$t(28) = .06, p = .95$
Gender	9M/7F	6M/8F	
Handedness	14RH/2LH	13RH/1LH	
Ethnicity (% Saudi)	100%	100%	
Sub-sample	(*n*= 8)	(*n*= 9)	
Age (*M*/*SD*)	4.5 ± 0.3	4.6 ± 0.3	$t(15) = -.15, p = .88$
Gender	3M/5F	2M/6F	
Handedness	8RH	9RH	
Ethnicity (% Saudi)	100%	100%	

ASD, autism spectrum disorder; F, female; M, male; RH, right-handed; LH, left-handed

Table 8 Descriptive characteristics of the full and sub-sample

One participant from the control was excluded due to an excessively noisy EEG recording. Two participants from the ASD group were excluded (one with an unconfirmed ADHD diagnosis, and one due to an excessively noisy EEG recording). Thus, the final sample comprised 14 participants with ASD and 16 control participants of comparable age and handedness. Descriptive characteristics of the current sample are summarised in table 5.1.

Participants were either reimbursed for their participation at a rate of £20 per 30 minutes or rewarded with toys of a similar value. All participants ' parents or legal guardians gave informed, signed consent. Permission to conduct the current study was granted by the Research Ethics Committee of the School of Psychology at the University of Kent, in collaboration with the Autism Research and Treatment Center at King Khaled Hospital. The research was conducted in accordance with the ethical standards of the British Psychological Society.

4.2.2 Visual stimuli

Four types of visual stimuli were created for the current experiment: (I) *a similar aged person performing a familiar action*, (II) *a similar aged person performing an unfamiliar action*, (III) *a dissimilar aged person performing a familiar action*, and (IV) *a dissimilar aged person performing an unfamiliar action*. 'Similar aged person' stimuli depicted an unknown child from either gender performing an action, while 'dissimilar aged person' stimuli depicted an unknown middle-aged male performing an action. Familiar hand actions involved the repetitive performance of either a *'no'* sign (by moving the index finger from side to side), or a *'come here'* sign (by moving four fingers together in a beckoning action). Unfamiliar (meaningless) hand actions involved the repetitive performance of either the hand moving in a rotating movement, or making a fist with the four fingers and moving the hand horizontally. See Figure 4.1 for still images of these video stimuli in each of the four conditions.

Each 80-second silent video contained colour clips depicting the actor against a plain white background. Thus, age similarity and action familiarity were manipulated in a fully crossed design, such that each similar aged actor, dissimilar aged actor, and action (familiar and unfamiliar) was seen only once during the entire experimental session.

Observing 1	Observing 2	Imitation
 Similar Age / Familiar Action 	 Dissimilar Age / Familiar Action 	
 Similar Age / Unfamiliar Action 	 Dissimilar Age / Unfamiliar Action 	
 Dissimilar Age / Familiar Action 	 Dissimilar Age / Familiar Action 	
 Dissimilar Age / Unfamiliar Action 	 Dissimilar Age / Unfamiliar Action 	

Figure 17 Four example experimental trials showing the four video stimuli combinations. Each trial consists of two observation periods (observation 1 & 2) followed by one imitation period, each lasting 80 seconds. The first video clip of each trial depicted one of the four conditions described above, crossing both age similarity and action familiarity. The second video clip in each pair depicted the same familiar/unfamiliar action, but this time it was always performed by a dissimilar aged person.

Each of these experimental stimuli were analysed in comparison to a baseline visual stimulus condition: white visual noise. This white visual noise depicted a unified, silent clip that lasted for 30 seconds.

Figure 18 Still image of white visual noise used in the 'baseline condition'.

4.2.3 Procedures

Clinical assessment

Initially, participants in the control group were assessed by the Egyptian version of the Wechsler Intelligence Scale for Children (WISC) (Ismail & Malika, 1974). ASD participants' diagnoses were confirmed by clinical evaluations based on DSM-IV criteria as well as the Autism Diagnostic Interview –Revised (ADI-R; Lord et al., 1994).

	Control group	ASD group	t p Cohen's d
Full sample			
VIQ	58.81 ± 4.18	56.07 ± 3.68	$t(28) = 1.89, p = .06, d = .71$
PIQ	56.26 ± 3.90	53.92 ± 6.73	$t(28) = 1.13, p = .27, d = .42$
FSIQ	115 ± 7.58	110 ± 8.72	$t(28) = 1.67, p = .10, d = .63$
ADI-R:			
Reciprocal Social Interaction	6 ± 2.06	17.8 ± 1.91	$t(28) = -16.22, p < .001, d = -6.13$
Communication	4.1 ± 1.78	13 ± 1.17	$t(28) = -16.26, p < .001, d = -6.14$
Restricted, Repetitive, and Stereotyped Patterns of Behavior	0.3 ± .13	3.9 ± 1.14	$t(28) = -11.90, p < .001, d = -4.49$
Sub-sample			
VIQ	61 ± 2.87	58.22 ± 3.70	$t(15) = 1.71, p = .10, d = .88$
PIQ	57.75 ± 1.38	52.88 ± 6.79	$t(15) = 1.98, p = .06, d = 1.02$
FSIQ	113.37 ± 9.66	111.11 ± 9.61	$t(15) = .48, p = .63, d = .24$
ADI-R:			
Reciprocal Social Interaction	5.62 ± 1.76	17.55 ± 2.12	$t(15) = -12.47, p < .001, d = -6.43$
Communication	3.37 ± 1.68	12.88 ± 1.36	$t(15) = -12.86, p < .001, d = -6.64$
Restricted, Repetitive, and Stereotyped Patterns of Behavior	0.27 ± .13	3.88 ± 1.36	$t(15) = -7.43, p < .001, d = -3.83$

ADI-R, Autism Diagnostic Interview–Revised (ADI-R; Lord, Rutter, & Le Couteur, 1994)
FSIQ, full-scale intelligence quotient; average subtests: information,comprehension,arithmetic, similaritic, ,vocabulary,digit span, picture completion, picture arrangement, sblock design, object assembly, and coding (WISC; Ismail & Malika, 1974)

Table 9 Descriptive characteristics of the clinical assessments

EEG data acquisition

During the main experiment, the experimenter prepared and tested each participant individually. Preparation for the EEG recording was carried out as described in Experiment 2. Videos were presented on a 16-inch computer screen within comfortable viewing distance. Participants initially viewed the 30-second video of white visual noise (baseline condition). This baseline period was followed by the four experimental trials, which presented participants with the visual stimuli described above, in a counterbalanced order.

On each trial, participants viewed two consecutive video clips, each lasting 80 seconds. The first video clip depicted one of the four conditions described above, crossing both age similarity and action familiarity. The second video clip in each pair depicted the same familiar/ unfamiliar action, but this time was always performed by a dissimilar aged person. This allowed us to examine whether the similarity cues available in the first video influenced understanding of the later repetitions of that action. Immediately after each pair of videos, participants were instructed to imitate the observed hand action for 80 seconds. With prior permission from the child ś parent, hand actions were

recorded throughout the imitation period for later analysis of behavioural performance. Participants were invited to take short breaks between experimental videos to ensure that they were alert and prepared for each recording phase. In total, the entire EEG recording period for Experiment 3 lasted approximately 30 minutes.

EEG data were collected from three electrodes over the sensorimotor cortex (C3, Cz and C4) and from the left and right mastoids, positioned according to the international 10-20 system. Impedance levels were lowered to at least 10 kΩ in all electrodes. The EEG signal was acquired using BIOPAC system (MP150) and Acknowledge software, as in Experiment 1. EEG data were recorded against a linked mastoids reference at a sampling rate of 1000 Hz. EEG data were collected for all observation and imitation periods.

4.2.4 EEG data preparation and statistical analysis

EEG data were analysed using Vision Analyzer 2 (Brain Products). Firstly, the continuous EEG signal for each participant was filtered using a 40Hz low-pass cut-off and 0.5Hz high-pass cut-off. The first and last 10 seconds of each 80 second period of continuous EEG were removed. Each 60 seconds for each condition was then divided into epochs of 2 seconds, with 50% overlap. Using a semi-automatic artefact rejection method any segments containing artefacts, such as muscle movement or drift, were identified and removed.

Fast Fourier transform was then performed on the data using a 10% Hanning window. Averaged power data of theta, alpha/ Mu and low beta frequency bands was obtained and ERD was calculated, as described in Experiment 1. IBM SPSS version 20 software was used to perform ANOVAs on the EEG data crossing the between-participants factor, group (control/ ASD) with the repeated-measures factors, age (similar/ dissimilar), action (familiar/ unfamiliar), task (*observation 1*/ *observation 2*/ imitation), averaged across electrodes (C3, Cz, C4).

Given that corresponding behavioural data were not available for the full sample, EEG

data were analysed primarily on a full sample and a sub-sample who had only completed the behavioural and EEG measures. Note that degrees of freedom were corrected using Greenhouse-Geisser when Mauchly's test of Sphericity indicated that sphericity had been violated.

4.3 RESULTS

4.3.1 Behavioural results

As in Experiment 1, behavioural analyses were conducted to examine children's explicit ability to imitate the observed actions. These analyses were conducted by hand-coding the video recordings of each participant performing the imitation actions in each condition. The number of correct and incorrect actions performed during each 80-second imitation period was counted.

Fifteen participants from the total experimental population did not consent to the use of video recording during the task. Therefore, these behavioural analyses were conducted on a smaller set of 8 participants in the control group and 9 participants in the ASD group.

Correct imitations

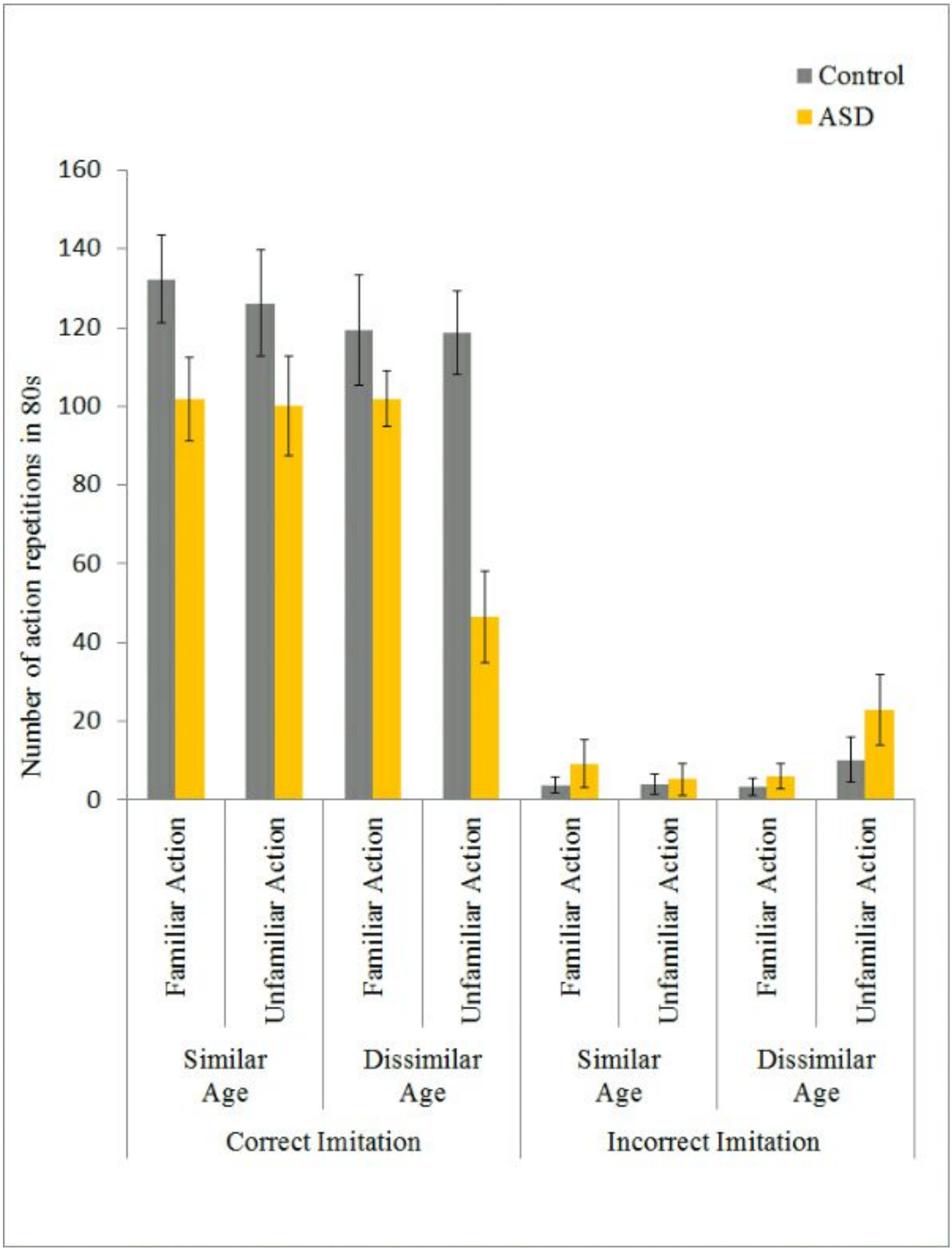


Figure 19 Bars represent the mean number of correct and incorrect imitations for control and ASD groups for each of the four imitation conditions, which lasted for 80 seconds. Error bars represent the standard error of the mean.

Is imitating communicative gestures impaired in ASD?

A three-way mixed 2 x 2 x 2 ANOVA was conducted to examine the effects of age (similar/ dissimilar) and action (familiar/ unfamiliar) as within-subject factors, and group (control/ ASD) as a between-subject factor on the number of correct action imitations. The main effect of group was significant, $F(1, 15) = 10.08, p = .006, \eta_p^2 = .40$, reflecting an increased number of correct action repetitions in the control group (M =124.12) compared to the ASD group (M =87.55).

Does imitating a familiar communicative gesture performed by a similar age actor facilitate the number of correct actions?

A main effect of age, $F(1, 15) = 5.81, p = .029, \eta_p^2 = .27$, demonstrated that participants performed more correct imitations when imitating an action performed first by a similar aged actor (M = 115.12) compared to an action first performed by a dissimilar aged actor (M = 96.55).

Furthermore, a main effect of action, $F(1, 15) = 8.02, p = .013, \eta_p^2 = .34$, showed an increased number of correct imitations when participants were imitating a familiar action (M =113.82) compared to an unfamiliar action (M =97.85). This effect was qualified by an interaction between group*action, $F(1, 15) = 4.99, p = .041, \eta_p^2 = .25$, and a significant 3-way group*action*age interaction, $F(1, 15) = 5.25, p = .037, \eta_p^2 = .25$. Interactions between group*age and age*action did not reach significance, (Fs < 3.55, ps >.08).

Separate repeated-measure ANOVAs were conducted for control and ASD participants, comparing age (similar/ dissimilar) and action (familiar/ unfamiliar). Analysis of the control group showed that none of the main effects or interactions reached significance (Fs < .07). In contrast, analysis of the ASD group showed that both main effects were significant. The main effect of age, $F(1, 8) = 6.37, p = .036, \eta_p^2 = .44$, demonstrated an increased number of correct imitations for imitating an action performed first by a similar aged actor (M = 101.00), compared to an action performed first by a dissimilar

aged actor (M = 74.11).

The main effect of action, $F(1, 8) = 12.87$, $p = .007$, $_p\eta^2 = .61$, revealed an increased number of correct imitations when imitating a familiar action (M = 101.83) compared to an unfamiliar action (M = 73.27). Furthermore, the interaction between age*action was significant, $F(1,8) = 9.75$, $p = .014$, $_p\eta^2 = .54$. Simple main effects analyses for the ASD group revealed that when the actor's age was similar to the child's, action familiarity did not influence imitation performance ($t < .12$, $p > .90$). However, when the actor's age was dissimilar to the child's, participants performed significantly fewer correct action imitations for an unfamiliar action (M = 46.33) compared to a familiar action (M = 101.88), $t(8) = 5.46$, $p < .001$, $d = 1.81$.

Similarly, when the action was familiar, similarity of the actor's age did not influence imitation performance ($t < -.01$, $p > .98$); however, when the action was unfamiliar, participants performed more correct imitations for a similar aged actor (M = 100.22) compared to a dissimilar aged actor (M = 46.33), $t(8) = 3.03$, $p = .016$, $d = 1.01$.

A similar three-way Mixed 2 x 2 x 2 ANOVA was conducted to examine the effects of age (similar/ dissimilar) and action (familiar/ unfamiliar) as within-subject factors, and group (control/ ASD) as a between-subject factor on the number of incorrect action imitations. A main effect of age, $F(1,15) = 6.08$, $p = .026$, $_p\eta^2 = .28$, demonstrated that participants made more incorrect imitations when imitating an action performed first by a dissimilar aged actor (M = 10.55) compared to an action performed first by a similar aged actor (M = 5.55). None of the remaining effects and interactions reached significance (Fs < 3.17, ps > .09).

4.3.2 Electroencephalographic results

ERD was calculated in three frequency bands: alpha (8-12Hz), low beta (12-20Hz) and theta (5.5-7.5Hz).

Alpha frequency band (8-12Hz)

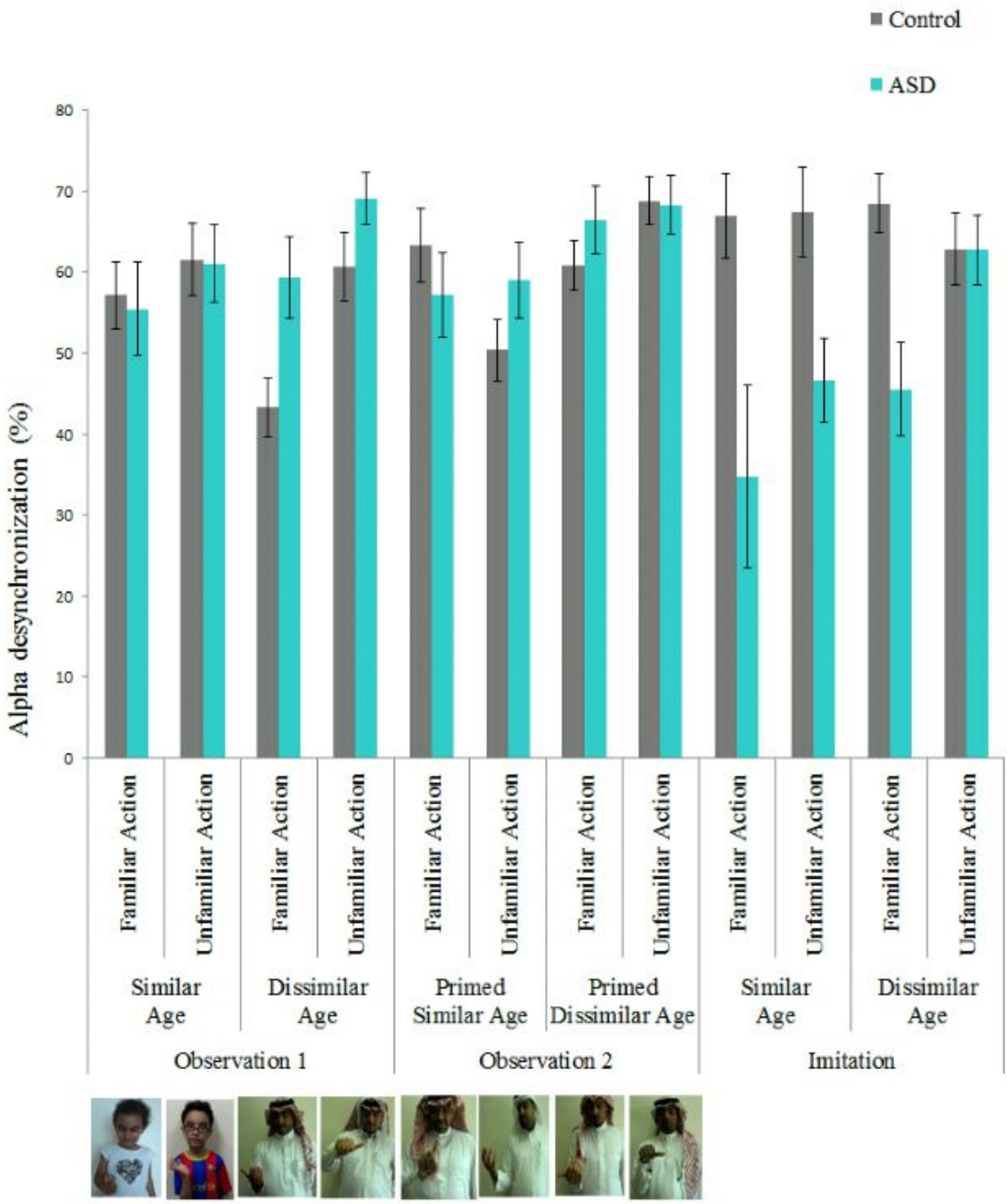


Figure 20 The bar chart represents the percentage of event-related changes in alpha (8-12Hz) power for control and ASD groups across the 12 conditions. Error bars represent the standard error of the mean.

A four-way mixed 2 x 2 x 2 x 3 ANOVA was conducted to examine the effects of age (similar/ dissimilar), action (familiar/ unfamiliar) and task (*observation 1*/ *observation*

2/ imitation) as within-subject factors, and group (control/ ASD) as a between-subject factor on alpha suppression[2]. Results revealed a number of significant effects and interactions which are summarised in Table 4.3.

	All data		
	df	F-value	p-value
Group	1,28	0.52	0.47
Age	1,28	70.09	0.001***
Action	1,28	16.53	0.001***
Task	1.5,40.6	29.61	0.001***
Group*Age	1,28	0.01	0.91
Group*Action	1,28	4.69	0.039*
Group*Task	2,56	2.98	0.059*
Age*Action	1,28	0.01	0.96
Age*Task	1.6,44.3	7.75	.003**
Action*Task	1.2,33.8	1.99	0.16
Group*Age*Action	1,28	2.30	0.14
Group*Age*Task	1,28	1.06	0.35
Group*Action*Task	2,56	1.69	0.19
Age*Action*Task	1.5,41.8	0.45	0.58
Group*Age*Action*Task	2,56	0.72	0.49

* for p<.05, ** for p<.01 and *** for p<.001.

Table 10 Mixed ANOVA for alpha suppression across age, action and task.

Is MNs activity modulated by action familiarity?

A main effect of action, $F(1, 28) = 16.53$, $p < .001$, $_p\eta^2 = .37$, demonstrated an increased level of alpha suppression for a familiar action ($M = 56.39\%$) relative to an unfamiliar action ($M = 61.62\%$). In addition, to follow up on the significant group*action interaction in the alpha frequency band, independent sample t-tests and paired sample t-tests were run to explore the effect of action familiarity in each group, and the difference between groups with familiar and unfamiliar actions.

Comparisons between familiar and unfamiliar actions in the control group revealed that alpha suppression with a familiar action was significantly higher (M = 53.62%) compared to an unfamiliar action (M = 62.84%), $t(15) = -5.51$, $p < .001$, $d = -1.37$. Similarly, analysis of the ASD group revealed that alpha suppression with a familiar action was significantly higher (M = 55.03%) compared to an unfamiliar action (M = 65.30%), $t(13) = -6.56$, $p < .001$, $d = -1.75$. However, alpha suppression did not differ between the control and ASD groups on familiar or unfamiliar action trials (ts < -.48, ps > .63).

Is MNs activity modulated by age similarity? And does priming by age similarity facilitate action understanding?

A main effect of age, $F(1, 28) = 70.09$, $p < .001$, $_p\eta^2 = .71$, demonstrated an increased level of alpha suppression with a familiar aged actor (M = 54.16%) relative to an unfamiliar aged actor (M = 63.86%).

In addition, paired sample t-tests were conducted to explore the two-way interaction between age similarity and task in control and ASD. Exploring the two different age levels at each task level demonstrated that alpha suppression was significantly higher when participants were observing a similar aged actor (M = 58.20%) compared to when they were observing a dissimilar aged model in *observation1* (M = 64.50%), $t(29) = -4.28$, $p < .001$, $d = -0.78$. Similarly, alpha suppression was significantly higher during the imitation period, when participants were imitating a similar aged actor (M = 48.07%) compared to when they were imitating a dissimilar aged actor (M = 55.68%), $t(29) = -1.01$, $p = .058$, $d = -0.18$. However, alpha suppression did not differ between similar (M = 63.57%) and dissimilar aged actors (M = 64.79%) during *observation 2* ($t < -1.01$, $p > .32$).

Furthermore, exploring the three task levels at each age level revealed that when the initial actor was a similar age to the participant, alpha suppression during *observation 2* was significantly lower (M = 63.57%) than during *observation 1* (M = 58.20%), $t(29) = -$

3.70, p = .001, d = -0.67, and imitation (M = 48.07%), t(29) = 4.03, p < .001, d = .73. Alpha suppression was also higher during the imitation period (M = 48.07%) compared to *observation 1* (M = 58.20%), t(29) = 2.94, p = .006, d = .53.

In contrast, when the initial actor was of a dissimilar age to the participant, alpha suppression during *observation 2* was significantly lower (M = 64.79%) compared to imitation (M = 55.68%), t(29) = 5.01, p < .001, d = .91, but did not differ from *observation 1* (M = 64.50%), (t <-.21, p > .83). Alpha suppression was also higher during the imitation period (M = 55.68%) compared to *observation 1* (M = 64.50%), t(29) = 4.44, p < .001, d = .81.

To follow up on the marginal group*task interaction in the alpha frequency band (p = .059), independent sample t-tests and paired sample t-tests were run to explore the effect of task in each group and the difference between groups during each task. Comparisons between task levels in the control group revealed that alpha suppression during *observation 2* (M = 65.48%) was significantly lower compared to *observation 1* (M = 61.51%), t(15) = -2.32, p = .035, d = -0.58, and imitation (M = 55.82%), t(15) = 4.93, p < .001 , d = 1.23. Furthermore, alpha suppression during imitation was significantly higher (M = 55.82%) than during *observation 1* (M = 61.51%), t(15) = 3.12, p = .007, d = .78.

In the ASD group, comparisons between task levels revealed that alpha suppression during *observation 2* (M = 62.69%), as in the control group, was significantly lower compared to during imitation (M = 47.37%), t(13) = 3.87, p = .002 , d = 1.03; however, it did not reach significance when compared to *observation 1* (t < -0.95, p >.36). Furthermore, alpha suppression of imitation was significantly higher (M = 47.37%) compared to *observation 1* (M = 61.17%), t(13) = 4.68, p < .001 , d = 1.25. Alpha suppression did not differ between the control and ASD groups on any of the three task levels (ts < 1.31, ps > .20).

Was MNs functioning influenced by IQ and hand imitation skills?

Correlational analyses revealed insignificant association between alpha suppression and intelligence quotient, *rs* < .38, *ps* > .12. Similarly, there was no significant association between alpha suppression and behavioural imitation, *rs* < .33, *ps* > .18.

Low beta band (12-20Hz)

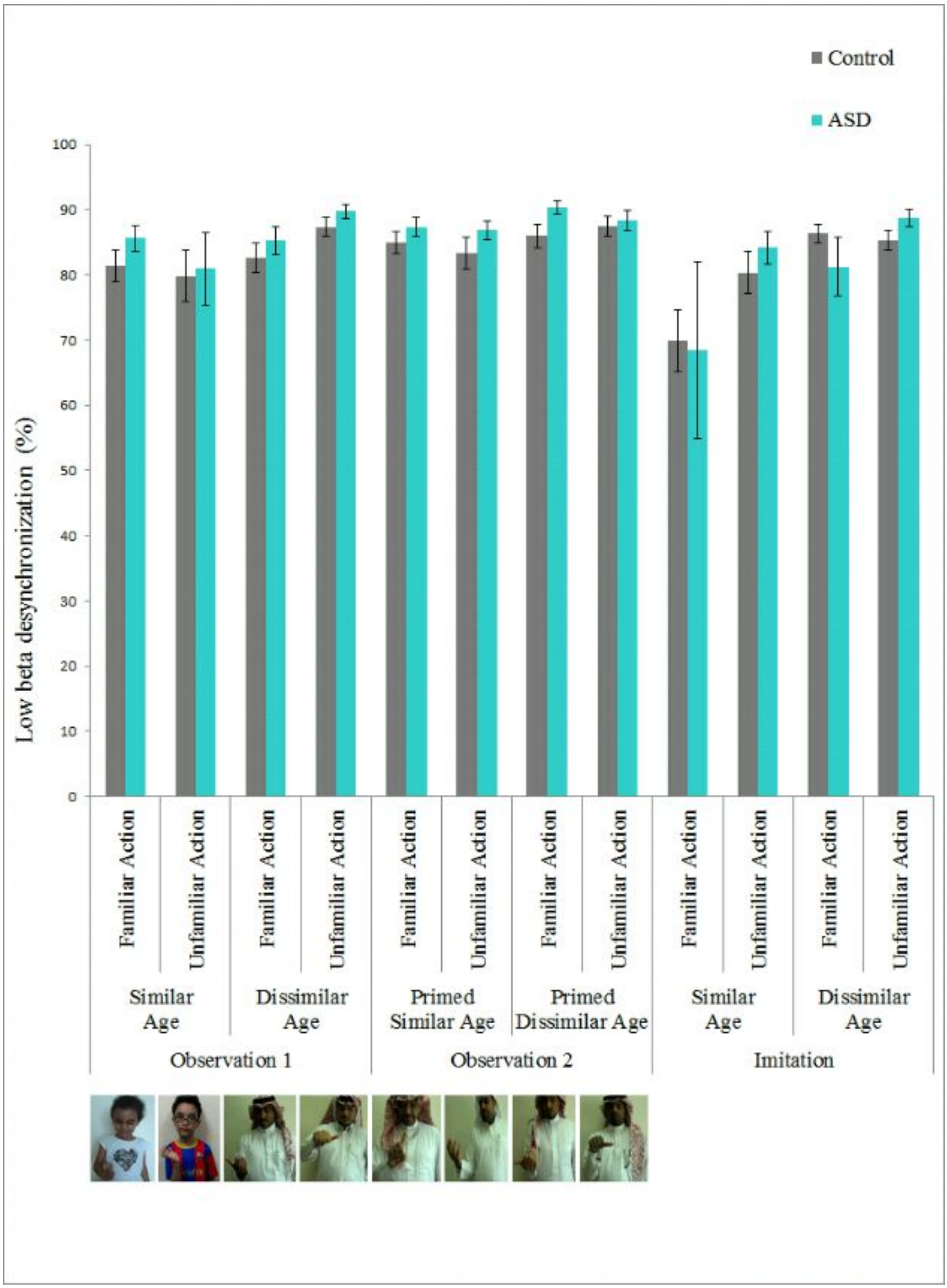


Figure 21 The bar chart represents the percentage of event-related changes in low beta power for control

and ASD groups across the 12 conditions. Error bars represent the standard error of the mean.

A four-way mixed 2 x 2 x 2 x 3 ANOVA was conducted to examine the effects of age (similar/dissimilar), action (familiar/unfamiliar) and task (*observation 1*/*observation 2*/imitation) as within-subject factors, and group (control/ASD) as a between-subject factor on low beta suppression[3]. Analysis revealed a number of significant effects which are summarised in Table 4.4.

	All data		
	df	F-value	p-value
Group	1,28	0.46	0.50
Age	1,28	13.72	0.001***
Action	1,28	5.45	.027*
Task	1.3,36.6	7.86	0.005**
Group*Age	1,28	0.08	0.77
Group*Action	1,28	0.32	0.57
Group*Task	2,56	0.42	0.65
Age*Action	1,28	0.07	0.78
Age*Task	1.4,39.1	3.03	0.07
Action*Task	1.1,30.9	3.70	0.06
Group*Age*Action	1,28	0.02	0.88
Group*Age*Task	2,56	0.06	0.94
Group*Action*Task	2,56	0.93	0.39
Age*Action*Task	1.2,32.3	3.13	0.08
Group*Age*Action*Task	2,56	0.19	0.82

* for p<.05, ** for p<.01 and *** for p<.001.

Table 11 Mixed ANOVA for low beta suppression across age, action, and task.

Is MNs modulated by action familiarity?

A main effect of action $F(1, 28) = 5.45$, $p = .027$, $\eta_p^2 = .16$, revealed an increased level of low beta suppression for a familiar action ($M = 82.44\%$) compared to an unfamiliar action ($M = 85.20\%$).

Is MNs activity modulated by age similarity? And does priming by age similarity facilitate action understanding?

A main effect of age, $F(1, 28) = 13.72$, $p = .001$, $_p\eta^2 = .32$, demonstrated an increased level of low beta suppression for similar aged actors ($M = 81.09\%$) compared to dissimilar aged actors ($M = 86.56\%$). In addition, paired sample t-tests were conducted to explore the two-way marginal interaction between age similarity and task. Exploring the two different age levels at each task level demonstrated that low beta suppression of a similar aged actor compared to a dissimilar aged actor was significantly higher during all task levels: *observation 1* (similar age: $M = 81.85\%$, dissimilar age: $M = 86.15\%$), $t(29) = -2.72$, $p = .011$, $d = -.49$, *observation 2* (similar age: $M = 85.51\%$, dissimilar age: $M = 87.96\%$), $t(29) = -3.39$, $p = .002$, $d = -0.61$, and during imitation trials (similar age: $M = 75.67\%$, dissimilar age: $M = 85.42\%$), $t(29) = -2.78$, $p = .009$, $d = -0.50$.

Furthermore, exploring the three task levels at each age level revealed that when the initial actor was a similar age to the participant, low beta suppression during *observation 2* was significantly lower ($M = 85.51\%$) than during *observation 1* ($M = 81.85\%$), $t(29) = -2.08$, $p = .046$, $d = -.37$, and imitation ($M = 75.67\%$), $t(29) = 2.68$, $p = .012$, $d = .48$. Low beta suppression was also higher during the imitation period ($M = 75.67\%$) compared to *observation 1* ($M = 81.85\%$), $t(29) = 2.10$, $p = .044$, $d = .38$. In contrast, when the initial actor was a dissimilar age to the participant, low beta suppression during *observation 2* was significantly lower ($M = 87.6\%$) compared to observation 1 ($M = 86.15\%$), $t(29) = -2.46$, $p = .020$, $d = -.45$, and imitation ($M = 85.42\%$), $t(29) = 2.17$, $p = .038$, $d = .39$. However, low beta suppression during *observation 1* elicited no significance compared to the imitation period ($t < .69$, $p = .49$).

Was MNs functioning influenced by IQ and hand imitation skills?

Correlational analyses revealed insignificant association between low beta suppression and intelligence quotient, $rs < -.29$, $ps > .24$. Similarly, there was no significant association between low beta suppression and behavioural imitation, $rs < .37$, $ps > .14$.

Theta frequency band (5.5-7.5Hz)

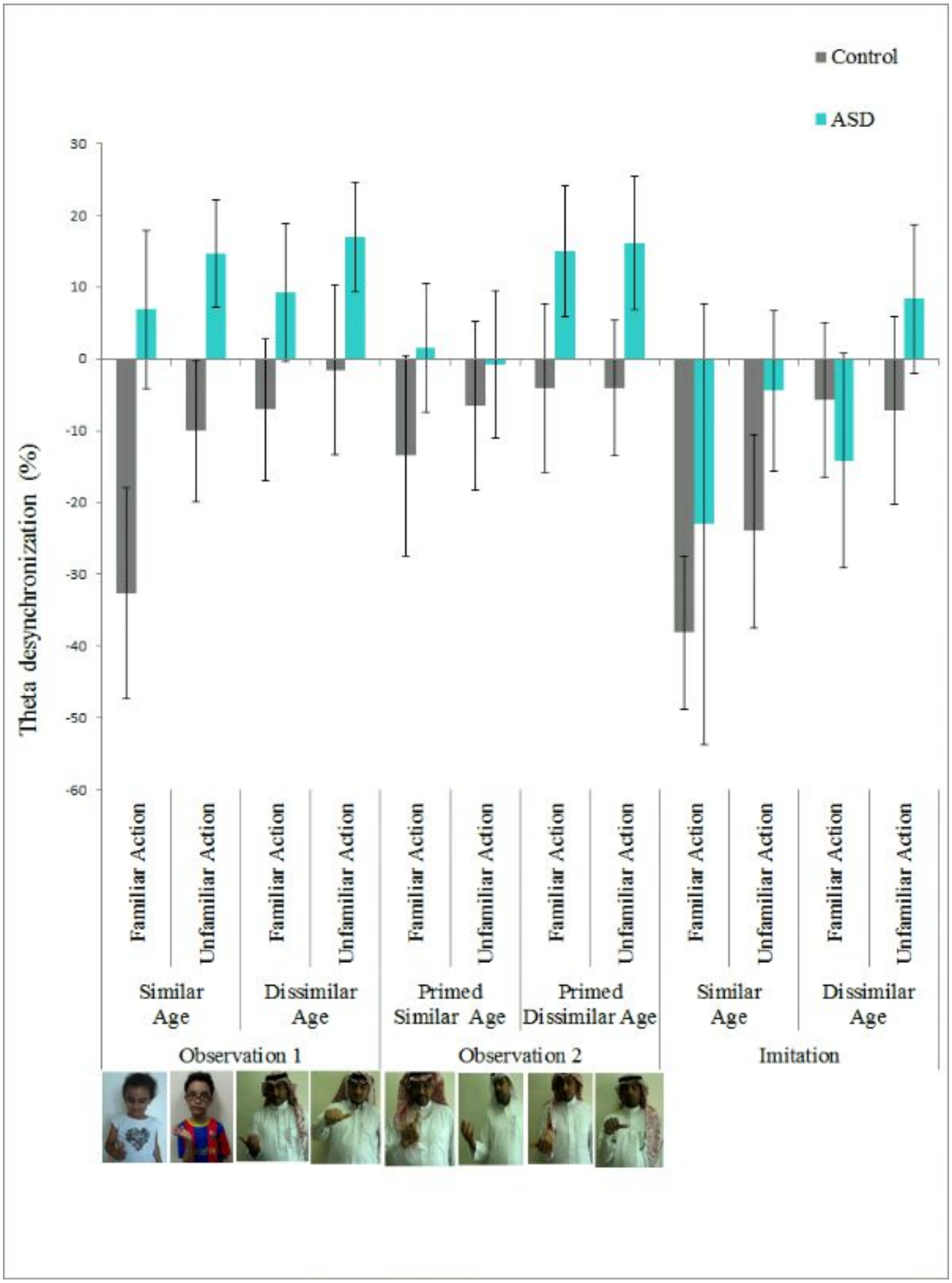


Figure 22 The bar chart represents the percentage of event-related changes in theta power for control and ASD groups across the 12 conditions. Error bars represent the standard error of the mean.

A four-way mixed 2 x 2 x 2 x 3 ANOVA was conducted to examine the effects of age (similar/ dissimilar), action (familiar/ unfamiliar) and task (*observation 1*/ *observation 2*/ imitation) as within-subject factors, and group (control/ ASD) as a between-subject factor on theta suppression[4]. Results revealed a number of significant effects, which are summarised in Table 4.5.

	All data		
	df	F-value	p-value
Group	1,28	1.35	0.25
Age	1,28	15.35	0.001***
Action	1,28	7.26	.012*
Task	1.5,43.2	8.56	0.002**
Group*Age	1,28	1.00	0.32
Group*Action	1,28	0.04	0.84
Group*Task	2,56	1.87	0.16
Age*Action	1,28	0.89	0.35
Age*Task	1.5,43.2	0.77	0.43
Action*Task	1.7,47.1	1.23	0.29
Group*Age*Action	1,28	1.89	0.17
Group*Age*Task	2,56	1.83	0.17
Group*Action*Task	2,56	0.97	0.38
Age*Action*Task	2,56	0.09	0.90
Group*Age*Action*Task	2,56	0.05	0.95

* for p<.05, ** for p<.01 and *** for p<.001.

Table 12 Mixed ANOVA for theta suppression across age, action, and task.

Is MNs modulated by age similarity?

A main effect of age, $F(1, 28) = 15.35$, $p = .001$, $_p \eta^2 = .35$, demonstrated an increased level of theta suppression for similar aged actors ($M = -10.81\%$) compared to dissimilar aged actors ($M = 1.84\%$).

Is MNs modulated aby action familiarity?

A main effect of action, $F(1, 28) = 7.26$, $p = .012$, $_p \eta^2 = .20$, revealed an increased level of theta suppression for a familiar action ($M = -8.77\%$) compared to an unfamiliar action ($M = -.19\%$).

Was MNs functioning influenced by IQ and hand imitation skills?

Correlational analyses revealed insignificant association between theta suppression and intelligence quotient, $rs < -.46$, $ps > .06$. Similarly, there was no significant association between theta suppression and behavioural imitation, $rs < .19$, $ps > .45$.

4.4 DISCUSSION

The current experiment set out two key aims at the outset. The first aim was to examine whether children with or without ASD show evidence of enhanced action understanding when observing a similar-aged person (child) performing a hand gesture, compared to observing a dissimilar-aged person (adult) performing the same gesture. With regards to this first question, the data revealed some important findings. Firstly, participants appeared to demonstrate great engagement with the same age actors, reflected by significantly increased neural desynchronization with the unfamiliar child models compared to the unfamiliar adult models, across all frequency bands.

Secondly, the effectiveness of observing a similar aged actor compared to a dissimilar aged actor was validated by the significantly increased number of correct imitations of actions when they had first been performed by a child actor compared to actions that were first performed by an adult actor. Additionally, results showed that the reverse was also true: children performed significantly fewer incorrect imitations of actions when they had first been performed by a child actor compared to when they were first performed by an adult actor.

The present data appear to be in agreement with previous research in supporting the claim that typically developing children show a same age preference, compared to adults or older siblings. For example, studies that employed the preferential looking paradigm found that childrens 'responses to similar-aged 'peers 'were significant, relative to the adult model (Sanefuji & Ohgami, 2011). In addition, behavioural data have shown that infants match peers 'actions (Hay et al., 1983), interact more with unfamiliar, but same age, peers than unfamiliar adults (Lewis et al., 1975) and have more reciprocal social interactions with similar aged peers than with older siblings (Vandell & Wilson, 1987).

Moreover, similar facilitation effects have been found in children with ASD, emphasizing the efficacy of peers in improving academic achievement (Utley et al. 1997), behavioural changes and social skills (McConnell, 2002). Further studies have shown improvements in imaginative play and levels of activity are correlated with the level of peer involvement (e.g., Kern & Aldridge, 2006).

The data also align well with previous research whose findings suggest an extended ' peer 'effect in prompting imitation. Specifically, Ryalls and colleagues found that the correct sequence of actions were recalled more accurately when the demonstrator was a same-age 'peer 'than when he/ she was an adult (Ryalls et al., 2000). This converges with results reported by Hanna and Meltzoff (1993), whose study aimed to examine peer imitation in toddlers. Their findings demonstrated that children were more likely to replicate actions performed by their peers, even in the absence of the peer 's behaviour.

Remarkably, only actions that had been demonstrated by same age peers, were retained after a 48hr delay, and were used in different contexts. Together, these data infer a lasting behavioural facilitation effect for similar age imitation in learning and development, and as suggested by Hanna and Meltzoff (1993), offers the intriguing possibility of transfer of learning across contexts. This raises the question of whether a similar effect could be found in deferred imitation (i.e. the ability to copy a previously witnessed action when that action is absent from the current visual environment: Courage & Howe, 2002). The current data, derived from a situation in which children re-enacted a hand action immediately following an on-screen video demonstration (but without concurrent perceptual input), suggest that effects of age similarity do act upon

deferred imitation. As such, they fit with previous research that reported more deferred imitation when children were asked to imitate a 2-year-old demonstrator, compared to an adult demonstrator (McCall et al., 1977).

Explaining the present findings within an integrated framework of social cognitive accounts suggests that the selective neural activations that children demonstrated toward observed peers mirror the notion that not all presented models will be imitated equally; the observer, however, will be more influenced by those who symbolise engaging qualities (Bandura, 1977).

This is convergent with the proposal of Epstein (1966) in showing that the characteristics of an observed model (e.g., age, ethnic status) influence the degree to which social attitudes and behaviour will be produced by others. Indeed, research evidence has shown that upon perceiving others as potential social partners, children start to increasingly integrate their activities with unfamiliar peers, and imitate their actions (Maudry & Nekula, 1939; Harlow, 1969), which allows them to elaborate a social engagement with an unfamiliar 'peer', compared to a familiar 'mother' (Eckerman et al., 1975). According to Meltzoff (1990), this synchronises the 'like-me' view, in which peer preference stems from childrens' ability to recognize others as being similar to the self, which in turn requires linking an observed action to the self to understand simulations of others' actions and mental states.

In addition to these views, it is worth highlighting an alternative view, which links age-facilitated peer imitation to cognitive effort. It has been proposed by Eckerman and colleagues that peers' actions are more likely to be copied than actions produced by an adult model, perhaps because they are more easily encoded since children are more likely to execute these actions in the same way as their partner performs them (Eckerman et al., 1975). Presenting a cognitive task as imitating a same-aged model, with a matched plan-of-performing, is less demanding on memory, and thus appears to require less cognitive effort.

This view seems to be in line with earlier suggestions by Festinger (1954) in his social comparison account, by showing that people have a tendency to elect reference figures

who are similar in terms of capability, and to disregard those who are too different from themselves.

When analysed separately, the effect and interactions of age similarity on the number of correct imitations did not yield equivalent findings to EEG data. The control group revealed no significant effect or interactions; as for the ASD group, the behavioural performance of the children did not significantly differ under the function of action familiarity, when the action was performed by a child actor. However, in the case of an adult actor, function of action familiarity was revealed in demonstrating more correct imitation for familiar action. In contrast, the effect of age appeared only as a main effect in the EEG data and analysis of incorrect imitations, showing that age similarity had a comparable effect on both groups within the context of these measures.

The second key aim in this experiment was to examine whether similar-age facilitation effects can be used to prime qualitative changes in behaviour when observing a dissimilar-aged person performing a hand gesture, especially in children with ASD. The data reported here is based on significant interaction of age*task in the alpha band, and marginal interaction of age*task in the low beta band. Specifically, in the alpha frequency band an age*task interaction emerged, showing that alpha suppression was significantly higher when participants were observing a similar aged actor compared to when they were observing a dissimilar aged model during observation 1 and during the imitation period, but did not differ between similar and dissimilar aged actors during observation 2.

This suggests that previous exposure to a similar-aged model did not facilitate action understanding when that same action was subsequently performed by a dissimilar aged model. Notably, the fact that group did not appear in a three-way interaction with age and task suggests that neither group experienced this facilitation effect, and thus the control and ASD children experienced the effect of age similarity across the tasks in a comparable way.

An additional aim of this experiment was to add to the current body of work investigating the developmental course of the frequency of neural oscillations over

childhood by examining the MNs activity in the theta frequency band (5.5 –7.5Hz). There are two main points to consider. Firstly, with reference to the influence of familiarity and similarity improving action understanding, we note that the facilitation effects of a similar aged actor and familiar actions were found to be consistently significant across the three EEG frequency bands. This implies that there was no major inconsistency between the neural activity in the three frequency bands, and supports the value of employing complementary analyses of activity in alpha, low beta and theta ranges.

Secondly, our findings revealed reasonable desynchronization over imitation periods across alpha, low beta and particularly the theta band; this is a finding that was not demonstrated in the work of Lepage and Théoret (2006), in either theta ranges. The theta band was proposed to be an equivalent biomarker of MNs activation in children, similar to the mu band in adults (Stroganova et al., 1999). This was established in Cochin et al. (2001), whose sample comprised children with an average age of 5.2 years.

Their findings demonstrated that observation of biological movement was associated with a significant desynchronization in theta 1 and theta 2 in fronto-temporal and central regions compared with nonhuman movement. This synchronised the data of Lepage and Théoret (2006), but as they failed to reflect the same effect during the execution condition, the argument that they reflect MNs activity is not supported by their data.

Finally, we replicated findings from Experiments 1 and 2 in demonstrating a significant effect for familiar versus unfamiliar hand actions. Here, children were able to perform significantly more correct imitations when they were imitating a familiar action compared to an unfamiliar action. In the ASD group, this action effect interacted with age such that the familiar action advantage was only evident when children were imitating an action that was first performed by a dissimilar aged actor, and not a similar aged actor. Moreover, analysis of the EEG frequency data revealed that both the control and ASD participants demonstrated significantly increased neural desynchronization with familiar hand actions compared to unfamiliar hand actions, across all frequency bands, which is consistent with previous work in this thesis (Chapters 2 and 3).

As for possible limitations in this experiment, it has been indicated earlier in this chapter (see part 4.3.1) that fifteen participants from the total experimental population did not consent to the use of video recording during the imitation task, which meant that analysis of behavioural data was conducted on half the total sample while the electrophysiological analysis was conducted on the full sample.

This raises two important considerations: firstly, the amount of suppression during the imitation period may not be fully explained by the available behavioural findings, as deciding whether the suppression was generated precisely by correct or incorrect hand actions for missing participants was not possible. In particular, the available behavioural data of the control participants, which comprised a limited number of participants, elicited no main effect of interaction for imitating peers; nonetheless, the available EEG data, which comprised all participants, demonstrated significant alpha and low beta suppression for imitating similar-aged models, compared to dissimilar-aged models. Secondly, what is suggested by our behavioural data may not be a statistically large enough sample to be generalised to the relevant wider populations, so any conclusions must be drawn carefully.

4.5 CONCLUSION

In summary, the present neural and behavioural data seem in agreement with our proposal for the significant facilitation capacity of simulating and imitating a similar-aged model, relative to a dissimilar-aged model in TD and ASD children. However, our second question, regarding whether the capacity of simulating a dissimilar-aged model will be improved if it was first primed by a similar-aged model, received no support from the alpha and low beta bands.

Through the use of EEG methodology to detect MNs activity, this experiment provided th e first electrophysiological support for previous behavioural studies that have suggested great effects of similar aged 'peers' in typically developed children (Sanefuji et al., 2000) and ASD children (Chan et al., 2009; McConnell, 2002). Here, we extend our findings to examine MNs activity in the theta band, which demonstrated neural suppression for imitation periods, which has not been established previously in the literature (Lepage &

Théoret, 2006).

Footnote

[1] Analysis of group differences in the baseline condition did not reach significance for the alpha frequency band ($t < -.65$, $p > .51$), low beta frequency band ($t < -1.62$, $p > .11$) or theta ($t < -1.35$, $p > .18$).

[2] when we conducted analyses on the alpha frequency band in the sub-samples, the interaction between group and action (full sample: $F = 4.68$, $p = .039$, $_p\eta^2 = .14$/ sub-sample: $F = 1.20$, $p = .28$, $_p\eta^2 = .07$), age and task (full sample: $F = 7.74$, $p = .001$, $_p\eta^2 = .21$/ sub-sample: $F = 6.26$, $p = .005$, $_p\eta^2 = .29$) and group and task interaction (full sample: $F = 2.97$, $p = .059$, $_p\eta^2 = .09$/ sub-sample: $F = 1.02$, $p = .37$, $_p\eta^2 = .06$) yielded the same range of small effect size.

[3] When we conducted analyses on the low beta frequency band in the sub-samples, the interaction between age and task (full sample: $F = 3.03$, $p = .056$, $_p\eta^2 = .09$/ sub-sample: $F = 3.06$, $p = .06$, $_p\eta^2 = .17$) yielded the same range of small effect size.

[4] When we conducted analyses on the theta frequency band in the sub-samples, the main effect of age (full sample: $F = 15.35$, $p = .001$, $_p\eta^2 = .35$/ sub-sample: $F = 8.85$, $p = .009$, $_p\eta^2 = .37$) and action (full sample: $F = 7.26$, $p = .012$, $_p\eta^2 = .20$/ sub-sample: $F = 2.09$, $p = .16$, $_p\eta^2 = .12$) yielded the same range of small effect size. Therefore, there seems to be no significant difference between the value of full-sample and sub-sample analysis in the three frequency band.

5 The effect of similar ethnicity and action familiarity on facilitating action understanding and imitation: investigating mirror neurons in children with ASD

5.1 5.1 EXPERIMENT 4

Numerous studies, concerned with ethnicity or 'in-group bias', are focused on two major domains: face recognition (Katz and Downey, 2002; Sangrigoli & DeSchonen, 2004; Bar-Haim et al., 2006) and memory (e.g. effect of own-race bias (ORB) (Dunham et al., 2011; Meissner & Brigham, 2001). However, few studies have investigated whether the ethnicity of the model during action observation, or action observation and imitation has an effect (Losin et al., 2013; Liew et al., 2010; Molnar-Szakacs et al., 2007; Désy & Théoret, 2007) and none investigated these effects in children. Furthermore, the majority of studies utilised fMRI and TMS and one was an MEP study. Nonetheless, to our knowledge, no research has yet investigated the neural activation elicited by MNs in looking at the effect of ethnic model similarity in children with ASD.

In the current experiment the same experimental paradigm as Experiment 2 was utilized, attempting to investigate two unanswered questions:

1) *Do children with or without ASD, during observation of a similar-ethnic model (Saudi) performing familiar communicative gestures, elicit greater neural activity of MNs compared to observing a dissimilar ethnic model (European) performing the same gesture.*
2) *Does incorporating a prime similar ethnic model subsequently elicit qualitative MNs neural activation for observing a dissimilar ethnic model performing communicative hand gestures in ASD children?*

Accordingly, two groups of children, ASD and control, watched 4 pairs of video that depicted either a model from the participants' ethnicity (Saudi), or a model from a foreign ethnicity (European) with whom they have limited contact. After each video pair, participants were asked to imitate the observed action. A video recording of the behavioural performance was also taken and used for further analysis. Based on the available literature the following predictions were made:

1) Children with ASD will demonstrate a decreased level of MNs activation relative to that in TD children
2) Children with ASD will demonstrate fewer correct imitative performances relative to TD children.
3) Both children with ASD and TD children will demonstrate a decreased level of MNs activation when observing an unfamiliar action relative to observing a familiar action.
4) Both children with ASD and TD children will demonstrate an increased level of MNs activation when observing a similar ethnic model, relative to a dissimilar ethnic model.
5) The level of MNs activation for observing a similar-ethnic model performing a familiar action will be higher relative to an unfamiliar action in TD and ASD children
6) The neural activation of a dissimilar ethnic model will be improved, if that action was primed by a similar ethnic model.

It is also our intention to investigate, in more depth, the theta band (5.5-7.5Hz). Specifically we expect neural activation by the theta band to yield equivalent neural activity as the alpha and low beta bands.

5.2 METHODS

5.2.1 Participants

A group of 13 control children and 13 children diagnosed with ASD ranging in age from 4 years to 5 years/1 month were used for the study. The ASD participants were recruited from the Autism Research and Treatment Center at King Khaled Hospital in Riyadh, Saudi Arabia. The control participants were recruited from selected nurseries in Riyadh, Saudi Arabia. All participants had normal or corrected-to-normal vision. Autistic participants who had comorbid neurological conditions or full scale IQ < 80 were excluded.

	Control group	ASD group	
Full sample	(*n*= 13)	(*n*= 13)	
Age (*M*/*SD*)	4.5 ± 0.3	4.6 ± 0.3	$t(24) = -.10, p < .91$
Gender	6M/7F	5M/8F	
Handedness	11RH/2LH	12RH/1LH	
Ethnicity (% Saudi)	100%	100%	
Sub-sample	(*n*= 8)	(*n*= 9)	
Age (*M*/*SD*)	4.5 ± 0.3	4.6 ± 0.3	$t(15) = -.15, p = .88$
Gender	3M/5F	2M/6F	
Handedness	8RH	9RH	
Ethnicity (% Saudi)	100%	100%	

ASD, autism spectrum disorder; F, female; M, male; RH, right-handed; LH, left-handed

Table 13 Descriptive characteristics of the full and sub-sample.

Descriptive characteristics of the current sample are summarised in table 5.1. Participants were either reimbursed for their participation at a rate of £20 per 30 minutes or rewarded with toys of a similar value. All participants' parents or legal guardians gave informed, signed consent. Permission to conduct the current study was granted by the Research Ethics Committee of the School of Psychology at the University of Kent, in collaboration with the Autism Research and Treatment Center at King Khaled Hospital. The research was conducted in accordance with the ethical standards of the British Psychological Society.

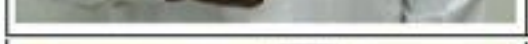
5.2.2 Visual stimuli

Four critical types of visual stimuli were created for the current experiment: (I) a person of similar ethnicity performing a familiar action, (II) a person of similar ethnicity performing an unfamiliar action, (III) a person of dissimilar ethnicity performing a familiar action, and (IV) a person of dissimilar ethnicity performing an unfamiliar action.

'Similar ethnic person' stimuli depicted unknown middle-aged males from the child's ethnic group (i.e. Saudi Arabian) performing an action, while 'dissimilar ethnic person' stimuli depicted unknown middle-aged males from a different ethnic group (i.e. European) performing an action. Familiar actions involved the performance of one of two repetitive familiar hand actions: either a 'come here' sign (by moving the index finger in a beckoning action), or a 'bye' sign (by waving an open hand). Unfamiliar actions involved the performance of one of two repetitive unfamiliar (meaningless)

hand actions: either opening and closing the right hand vertically, or making a fist with the four fingers and moving the thumb in a rotating movement. See Figure 5.1 for still images of these video stimuli in each of the four conditions.

Each video clip lasted for 80 seconds and all were silent, coloured video clips depicting the actor against a plain white background. Thus, ethnic similarity and action familiarity were manipulated in a fully crossed design, such that each similar ethnic actor, dissimilar ethnic actor, and each action (familiar and unfamiliar) was seen only once during the entire experimental session.

Observing 1	Observing 2	Imitation
 Similar Ethnicity/Familiar Action	 Dissimilar Ethnicity/Familiar Action	
Similar Ethnicity/Unfamiliar Action	Dissimilar Ethnicity/Unfamiliar Action	
 Dissimilar Ethnicity/Familiar Action	 Dissimilar Ethnicity/Familiar Action	
 Dissimilar Ethnicity/Unfamiliar Action	 Dissimilar Ethnicity/Unfamiliar Action	

and imitation: age similarity

Figure 23 Four example experimental trials showing the four video stimuli combinations. Each trial consists of two observation periods (observation 1 & 2) followed by one imitation period, each lasting 80 seconds. The first video clip of each trial depicted one of the four conditions described above, crossing both ethnic similarity and action familiarity. The second video clip in each pair depicted the same familiar/unfamiliar action, but this time it was always performed by a person of dissimilar ethnicity.

As in previous experiments, each of these experimental stimuli were analysed in comparison to a baseline visual stimulus condition: white visual noise. This white visual noise depicted a unified, silent, white visual noise video clip lasting for 30 seconds.

Figure 24 Still image of white visual noise used in the 'baseline condition'.

5.2.3 Procedures

Clinical assessment

	Control group	ASD group	t p Cohen's d
Full sample			
VIQ	58.7 ± 3.90	56.38 ± 3.64	$t(24) = 1.14, p = .26, d = .46$
PIQ	55.84 ± 4.25	54.69 ± 6.34	$t(24) = .54, p = .59, d = .22$
FSIQ	113.84 ± 7.79	111.07 ± 8.05	$t(24) = -.89, p = .38, d = -.36$
ADI-R:			
Reciprocal Social Interaction	6.1 ± 2.13	18.1 ± 1.91	$t(24) = -16.20, p < .001, d = -6.61$
Communication	4.2 ± 1.96	13 ± 1.22	$t(24) = -13.65, p < .001, d = -5.57$
Restricted, Repetitive, and Stereotyped Patterns of Behavior	0.2 ± .13	4.1 ± .08	$t(24) = -17.26, p < .001, d = -7.04$
Sub-sample			
VIQ	59.5 ± 2.92	56.22 ± 4.05	$t(15) = -.13, p = .07, d = -.06$
PIQ	57.37 + 1.68	56.44 + 5.63	$t(15) = .44, p = .66, d = .22$
FSIQ	116.87 ± 3.72	112.66 ± 7.48	$t(15) = 1.43, p = .17, d = .73$
ADI-R:			
Reciprocal Social Interaction	5.8 ± 2.29	18.1 ± 1.69	$t(15) = -16.61, p < .001, d = -8.57$
Communication	4.2 + 2.18	12.7 + 1.39	$t(15) = -9.70, p < .001, d = -5.00$
Restricted, Repetitive, and Stereotyped Patterns of Behavior	0.2 ± .10	4.2 ± .08	$t(15) = -13.38, p < .001, d = -6.90$

ADI-R, Autism Diagnostic Interview–Revised (ADI-R; Lord, Rutter, & Le Couteur, 1994)
FSIQ, full-scale intelligence quotient; average subtests: information,comprehension,arithmetic, similaritie, ,vocabulary,digit span, picture completion, picture arrangement, sblock design, object assembly, and coding (WISC; Ismail & Malika, 1974)

Table 14 Descriptive characteristics of the clinical assessments

Initially, participants in the control group were assessed by the Egyptian version of the Wechsler Intelligence Scale for Children (WISC) (Ismail & Malika, 1974). ASD participants' diagnoses were confirmed by clinical evaluations based on DSM-IV criteria as well as the Autism Diagnostic Interview –Revised (ADI-R; Lord et al., 1994).

EEG data acquisition

During the main experiment, the experimenter prepared and tested each participant individually. Preparation for the EEG recording was carried out as described in Experiment 2. Videos were presented on a 16-inch computer screen within comfortable viewing distance. Participants initially viewed the video of white visual noise (baseline condition) lasting for 30 seconds. This baseline period was followed by the four experimental trials, which presented participants with the visual stimuli described above, in a counterbalanced order. On each trial, participants viewed two consecutive video clips, each lasting 80 seconds. The first video clip depicted one of the four conditions described above, crossing both ethnic similarity and action familiarity. The second video clip in each pair depicted the same familiar/unfamiliar action, but this time it was always performed by a dissimilar ethnic person.

This allowed us to examine whether the similarity cues available in the first video influenced action understanding of later repetitions of that action. Immediately after each pair of videos, participants were instructed to imitate the observed hand action for 80 seconds. With prior permission from the childrens' parent, participants' hand actions were recorded throughout the imitation period for later analysis of behavioural performance. Participants were invited to take short breaks between experimental videos to ensure they were alert and prepared for each recording phase. The entire EEG recording period for Experiment 4 lasted approximately 30 minutes.

EEG data were collected from three electrodes over the sensorimotor cortex, from C3, Cz and C4 electrodes, and from the left and right mastoids, positioned according to the international 10-20 system. Impedance levels were lowered to at least 10 kΩ in all electrodes. The EEG signal was acquired using BIOPAC system (MP150), and Acknowledge software, as in Experiment 1. EEG data were recorded against a linked mastoids reference at a sampling rate of 1000 Hz. EEG data were collected for all observation and imitation periods.

EEG data preparation and statistical analysis

EEG data were analysed using Vision Analyzer 2 (Brain Products). Firstly, the continuous EEG signal for each participant was filtered using a 40Hz low-pass cut-off and 0.5Hz high-pass cut-off. The first and last 10 seconds of each 80 second period of continuous EEG were removed. Each 60 seconds for each condition was then divided into epochs of 2 seconds, with 50% overlap. Using a semi-automatic artefact rejection method, segments containing artefacts, such as muscle movement or drift, were identified and removed.

Fast Fourier transform was then performed on the data using a 10% Hanning window. Averaged power data of alpha (8 –12 Hz), low beta (12 –20 Hz) and theta (5.5-7.5 Hz) frequency bands was obtained, and ERD was calculated, as described in Experiment 1. IBM SPSS version 20 software was used to perform ANOVAs on the EEG data comparing the between-participants factor, group (control/ ASD) with the repeated-measures factors, ethnicity (similar/ dissimilar), action (familiar/ unfamiliar) and task

(observation 1/ observation 2/ imitation) averaged across electrodes (C3, Cz, C4).

Given that corresponding behavioural data were not available for the full sample, EEG data was analysed primarily on a full sample and a sub-sample who had only completed the behavioural and EEG measures. Note that degrees of freedom were corrected using Greenhouse-Geisser when Mauchly's test of Sphericity indicated that sphericity had been violated.

5.3 RESULTS

5.3.1 Behavioural results

As in Experiment 1, behavioural analyses were conducted to examine children's explicit ability to imitate observed actions in each condition. These analyses were conducted by hand-coding the video recordings of each participant performing the imitation actions, in each condition. The number of times that children performed the correct action during each 80-second imitation period was counted, along with the number of incorrect actions performed in the same period.

Seven participants from the total experimental population did not consent to the use of video recording during the task. Therefore, the behavioural analyses were conducted on a smaller set of eight participants in the control group and nine participants in the ASD group.

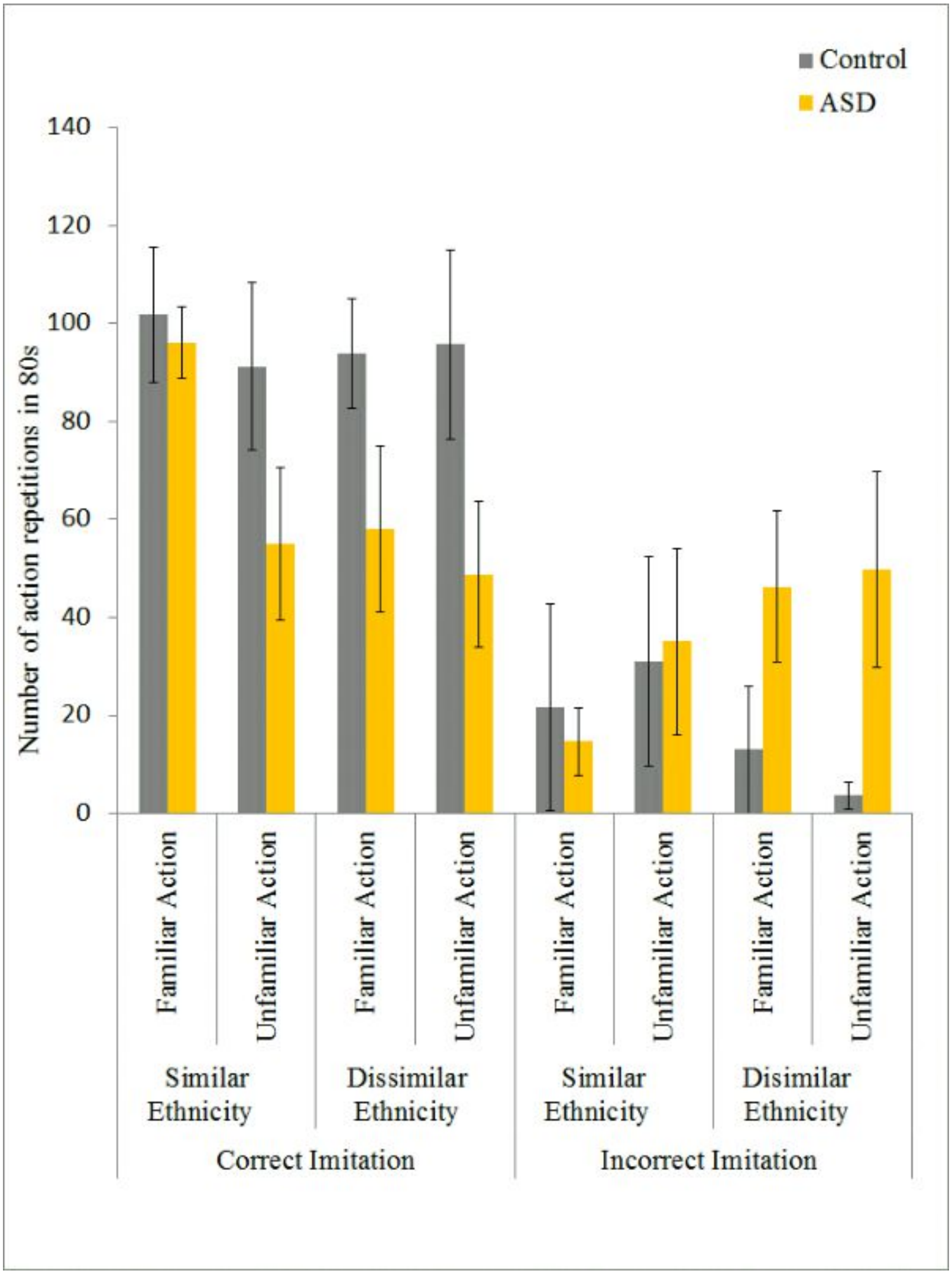


Figure 25 Mean number of correct and incorrect imitations for control and ASD groups for each of the four imitation conditions. Error bars represent the standard error of the mean.

Is imitating communicative gestures impaired in ASD?

A three-way mixed 2 x 2 x 2 ANOVA was conducted to examine the effects of ethnicity (similar/dissimilar) and action (familiar/unfamiliar) as within-subject factors, and group (control/ASD) as a between-subject factor on the number of correct action imitations. As in the previous studies a main effect of group, $F(1, 15) = 5.99$, $p = .027$, $_p\eta^2 = .29$, demonstrated an increased number of correct imitations in the control group ($M = 95.68$), compared to the ASD group ($M = 64.50$).

Is imitating familiar communicative gesture facilitate the number of correct actions?

A main effect of action, $F(1, 15) = 6.73$, $p = .020$, $_p\eta^2 = .31$, showed an increased number of correct imitations for imitating a familiar action ($M = 87.49$), compared to an unfamiliar action ($M = 72.69$).

Does imitating a communicative gesture performed by a similar ethnic model facilitate the number of correct actions?

The main effect of ethnicity did not reach significance ($F < 1.26$, $p = .27$), and neither did any of the interactions (Fs < 3.34, ps $> .08$).

A similar three-way mixed 2 x 2 x 2 ANOVA was conducted to examine the effects of ethnicity (similar/dissimilar) and action (familiar/unfamiliar) as within-subject factors, and group (control/ASD) as a between-subject factor on the number of incorrect action imitations. Results revealed a marginally significant interaction between group*ethnicity, $F(1, 15) = 4.28$, $p = .056$, $_p\eta^2 = .22$. To explore this two-way interaction further, independent sample t-tests were run to test the between-group differences for each ethnic similarity condition.

This revealed that there was no significant difference in the number of incorrect imitations between the control and ASD groups when participants were imitating an action that had first been performed by an actor from a similar ethnic group, ($t < .36$, $p > .72$). However, when participants were imitating an action that had first been performed by an actor from a dissimilar ethnic group, ASD participants ($M = 48.00$) performed significantly more incorrect imitations than control participants ($M = 8.31$), $t(11) = -2.51$, $p = .028$, $d = -2.4$[1)]. Furthermore, paired sample t-tests showed no significant effect

of ethnicity in either the control group ($t < 2.04, p > .08$) or the ASD group ($t < -1.26, p > .24$).

None of the remaining effects or interactions reached significance ($Fs < .44, ps > .52$).

5.3.2 Electroencephalographic results

As in Experiment 3, ERD was calculated in three frequency bands: alpha (8-12Hz), low beta (12-20Hz) and theta (5.5-7.5Hz).

Alpha frequency band (8-12Hz)

[1)] Since Levene's test indicated unequal variances, degrees of freedom were adjusted from 15 to 11.

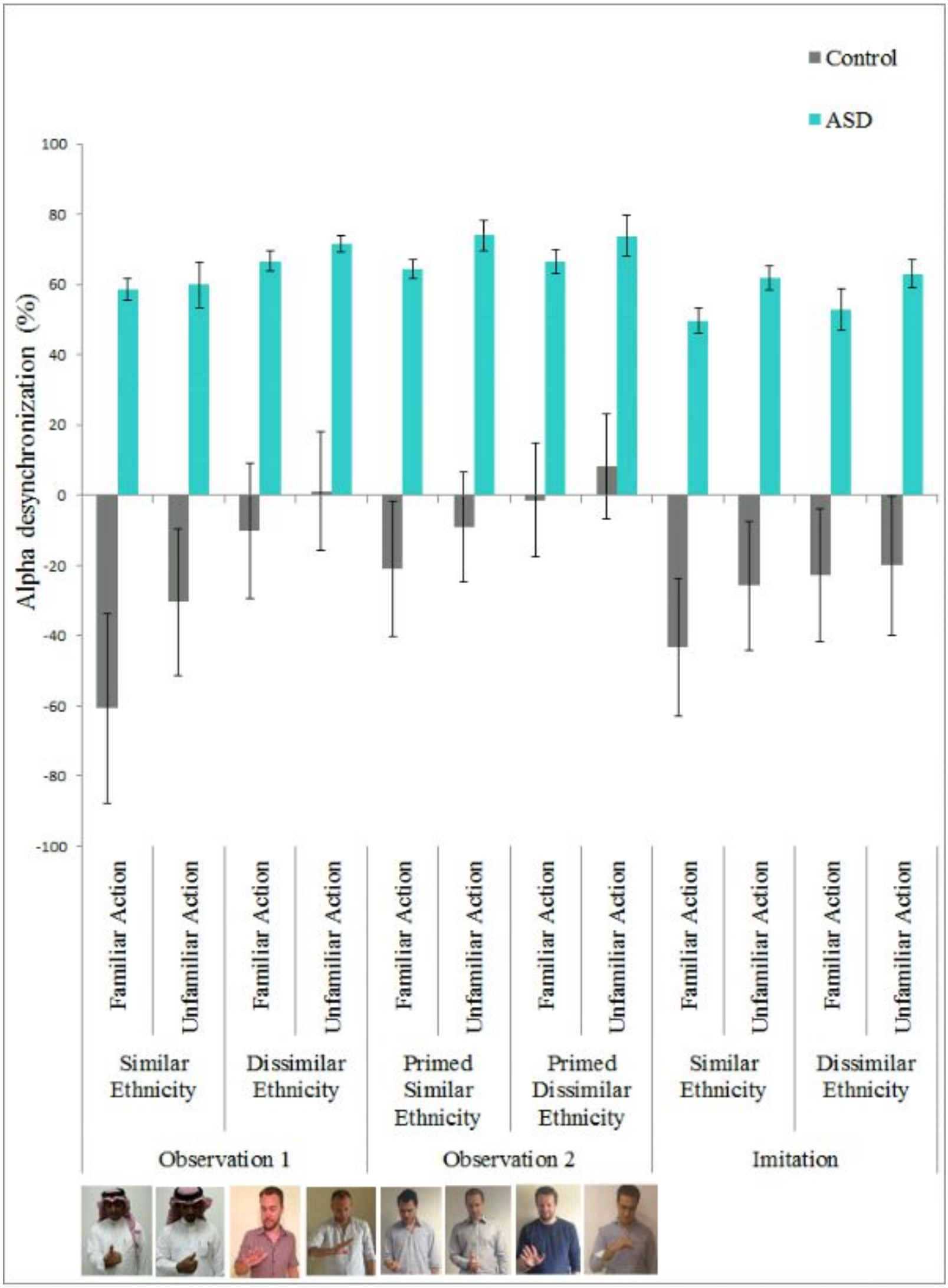


Figure 26 The bar chart represents the percentage of event-related changes in alpha power for control and ASD groups across 12 conditions. Error bars represent the standard error of the mean.

A four-way mixed 2 x 2 x 2 x 3 ANOVA was conducted to examine the effects of ethnicity

(similar/ dissimilar), action (familiar/ unfamiliar), and task (*observation 1*/ *observation 2*/ imitation) as within-subject factors, and group (control/ ASD) as a between-subject factor on alpha suppression[2]. Results revealed a number of significant effects and interactions which are shown in Table 5.2.

	All data			Control group			ASD group		
	df	F-value	p-value	df	F-value	p-value	df	F-value	p-value
Group	1,24	20.12	0.001***	-	-	-	-	-	-
Ethnicity	1,24	25.87	0.001***	1,12	23.46	0.001**	1,12	2.42	0.14
Action	1,24	30.62	0.001***	1,12	16.83	0.001**	1,12	25.35	0.001***
Task	1.6,37.6	23.30	0.001***	2,24	14.61	0.001***	2,24	17.05	0.001***
Group*Ethnicity	1,24	16.63	0.001***	-	-	-	-	-	-
Group*Action	1,24	4.88	0.037*	-	-	-	-	-	-
Group*Task	2,48	6.42	0.003**	-	-	-	-	-	-
Ethnicity*Action	1,24	2.57	0.12	1,12	3.44	0.08	1,12	0	0.98
Ethnicity*Task	2,48	7.71	0.001***	2,24	4.62	0.020*	2,24	3.89	.035*
Action*Task	1.6,38.9	0.61	0.51	2,24	1.63	0.22	1.2,14.7	1.65	0.21
Group*Ethnicity*Action	1,24	2.52	0.13	-	-	-	-	-	-
Group*Ethnicity*Task	2,48	1.27	0.29	-	-	-	-	-	-
Group*Action*Task	2,48	2.65	0.08	-	-	-	-	-	-
Ethnicity*Action*Task	2,48	0.22	0.79	2,24	0.87	0.43	1.4,16.8	0.55	0.52
Group*Ethnicity*Action*Task	2,48	1.36	0.27	-	-	-	-	-	-

* for p<.05, ** for p<.01 and *** for p<.001.

Table 15 Mixed & separate ANOVAs for alpha suppression across ethnicity, action, and task.

Is MNs activity impaired in ASD?

A main effect of group, $F(1, 24) = 20.11$, $p < .001$, $_p\eta^2 = .47$, demonstrated an increased level of alpha suppression in the control group ($M = -19.58\%$), compared to the ASD group ($M = 63.62\%$).

Is MNs activity modulated by action familiarity?

A main effect of action, $F(1, 24) = 30.62$, $p < .001$, $_p\eta^2 = .56$, demonstrated an increased level of alpha suppression for a familiar action ($M = 17.06\%$) compared to an unfamiliar action ($M = 26.97\%$). Analysis of the control group showed a main effect of action, $F(1, 12) = 16.82$, $p < .001$, $_p\eta^2 = .58$, demonstrated an increased level of alpha suppression for a familiar action ($M = -26.51\%$) compared to an unfamiliar action ($M = -12.64\%$). Similar to the control group, analysis of the ASD group showed a main effect of action $F(1, 12) = 25.34$, $p < .001$, $_p\eta^2 = .68$, demonstrated an increased level of alpha suppression for a familiar action ($M = 60.65\%$) compared to an unfamiliar action ($M = 66.60\%$).

Is MNs activity modulated by similar ethnicity of the model? And is priming by similar ethnicity of the model facilitate action understanding?

A main effect of ethnicity, $F(1, 24) = 25.87$, $p < .001$, $_p\eta^2 = .52$, demonstrated an increased level of alpha suppression for similar ethnic actors ($M = 15.31\%$) compared to dissimilar ethnic actors ($M = 28.73\%$). Analysis of the control group showed that a main effect of ethnicity, $F(1, 12) = 23.46$, $p < .001$, $_p\eta^2 = .66$, demonstrated an increased level of alpha suppression for similar ethnic actors ($M = -31.67\%$) compared to dissimilar ethnic actors ($M = -7.48\%$).

As for significant interaction, planned comparisons (paired sample t-tests), collapsed across action, were conducted to explore the two-way interaction between the two levels of ethnicity with the three levels of task for the control group. Exploring the two different ethnicity levels, at the same task level, demonstrated that the alpha suppression of observing a similar ethnic model was significantly higher ($M = -35.43$) relative to observing a dissimilar ethnic model during *observation 1* ($M = -14.64$), $t(12) = -2.97$, $p = .012$, $d = -0.8$. Similarly, the alpha suppression during *observation 2*, primed

by a similar ethnic model, was significantly higher (M = -11.17) relative to *observation 2* when primed by a dissimilar ethnic model (M = -.40), $t(12)$ = -2.79, p = .016, d = -0.8. Furthermore, the alpha suppression of imitating a similar ethnic model was significantly higher (M = -32.93) relative to imitating a dissimilar ethnic model (M = -22.88), $t(12)$ = -2.61, p = .022, d = -0.7.

Exploring each of the ethnicity levels, across the three task levels, revealed that the comparisons of a similar ethnic model demonstrated that the alpha suppression of *observation 1* was significantly higher (M = -35.4) relative to *observation 2* (M = -11.17), $t(12)$ = -3.28, p = .007, d= -0.9. In addition, the alpha suppression during *observation 2* was significantly lower (M = -11.2) relative to the imitation conditions (M = -33), $t(12)$ = 4.4, p < .001, d= 1.2; however, the comparison of *observation 1* and imitation did not elicit significance (t < -0.41, p > .68).

Comparisons of a dissimilar ethnic model, across the three task levels, revealed a similar trend to the similar ethnic model interaction. Mainly, the alpha suppression of *observation 2* was significantly lower (M = -0.40) relative to *observation 1* (M = 14.64), $t(12)$ = -2.54, p =.026, d= -0.7, and the imitating condition (M = -22.88), $t(12)$ = 5.18, p < .001, d= 1.4. However, the comparison of *observation 1* and imitation did not elicit significance (t < 1.86, p > .08).

Similar to the control, planned comparisons (paired sample t-tests), collapsed across action, were conducted to explore the two-way interaction between the two levels of ethnicity with the three levels of task for the ASD group. Exploring the two different ethnicity levels, at the same task level, demonstrated the alpha suppression of observing a similar ethnic model did not elicit a significant difference relative to observing a dissimilar ethnic model during *observation 1* or *observation 2* (Fs < 1.91, ps > .08). However, the alpha suppression of imitating a similar ethnic model was significantly higher (M = 53.72) relative to imitating a dissimilar ethnic model (M = 64.06), $t(12)$ = -3.20, p = .008, d = -0.9.

Exploring each of the ethnicity levels, across the three task levels, revealed that the comparisons of a similar ethnic model demonstrated that the alpha suppression during *observation 1* was significantly higher ($M = 62.66$) relative to *observation 2* ($M = 65.75$), $t(12) = -2.45$, $p = .031$, $d = -0.7$, and the alpha suppression during *observation 2* was significantly lower ($M = 65.6$) relative to imitation conditions ($M = 53.72$), $t(12) = 3.64$, $p = .003$, $d = 1$. Furthermore, the alpha suppression during *observation 1* was also significantly lower ($M = 62.66$) relative to the imitation condition ($M = 53.72$), $t(12) = 3.35$, $p = .006$, $d = .9$. Comparisons of a dissimilar ethnic model, across the three task levels, revealed that the alpha suppression during *observation 1* did not elicit significance relative to *observation 2* or imitation (Fs < -1.91, ps > .08); however, the alpha suppression during *observation 2* was significantly lower ($M = 69.98$) relative to the imitation condition ($M = 64.06$), $t(12) = 2.28$, $p = .041$, $d = .6$.

Independent sample t-tests were run to explore the differences between the group effect of group*ethnicity*task interaction.

As for similar ethnic person, comparisons of *observation 1* demonstrated that alpha suppression of the control group ($M = -35.43\%$) was significantly higher compared to the ASD group ($M = 62.66\%$), $t(24) = -4.34$, $p < .001$, $d = 1.70$. Similarly, comparisons of *observation 2* demonstrated that alpha suppression of the control group ($M = -11.17\%$) was significantly higher compared to the ASD group ($M = 65.56\%$), $t(24) = -4.28$, $p < .001$, $d = 1.68$. In addition, comparison of imitation demonstrated that alpha suppression of the control group ($M = -32.93\%$) was significantly higher compared to the ASD group ($M = 53.72\%$), $t(24) = -4.58$, $p < .001$, $d = 1.79$. As for dissimilar ethnic person, comparisons of *observation 1* demonstrated that alpha suppression of the control group ($M = -14.64\%$) was significantly higher compared to the ASD group ($M = 56.75\%$), $t(24) = -4.24$, $p < .001$, $d = 1.66$. Similarly, comparisons of *observation 2* demonstrated that alpha suppression of the control group ($M = -.40\%$) was significantly higher compared to the ASD group ($M = 69.98\%$), $t(24) = -4.44$, $p < .001$, $d = 1.74$. In addition, comparison of imitation demonstrated that alpha suppression of the control group ($M = -22.88\%$) was significantly higher compared to the ASD group ($M = 64.06\%$), $t(24) = -4.57$, $p < .001$, $d = 1.79$.

Was MNs functioning influenced by IQ and hand imitation skills?

Correlational analyses revealed insignificant association between alpha suppression and intelligence quotient, *rs* < -.29, *ps*> .25. However, there was significant association between alpha suppression during both the imitation condition, $r = -.49$, $p = .043$, and the observation condition, $r = -.47$, $p = .053$, and behavioural imitation. Both revealed moderate correlation.

Low beta band (12-20Hz)

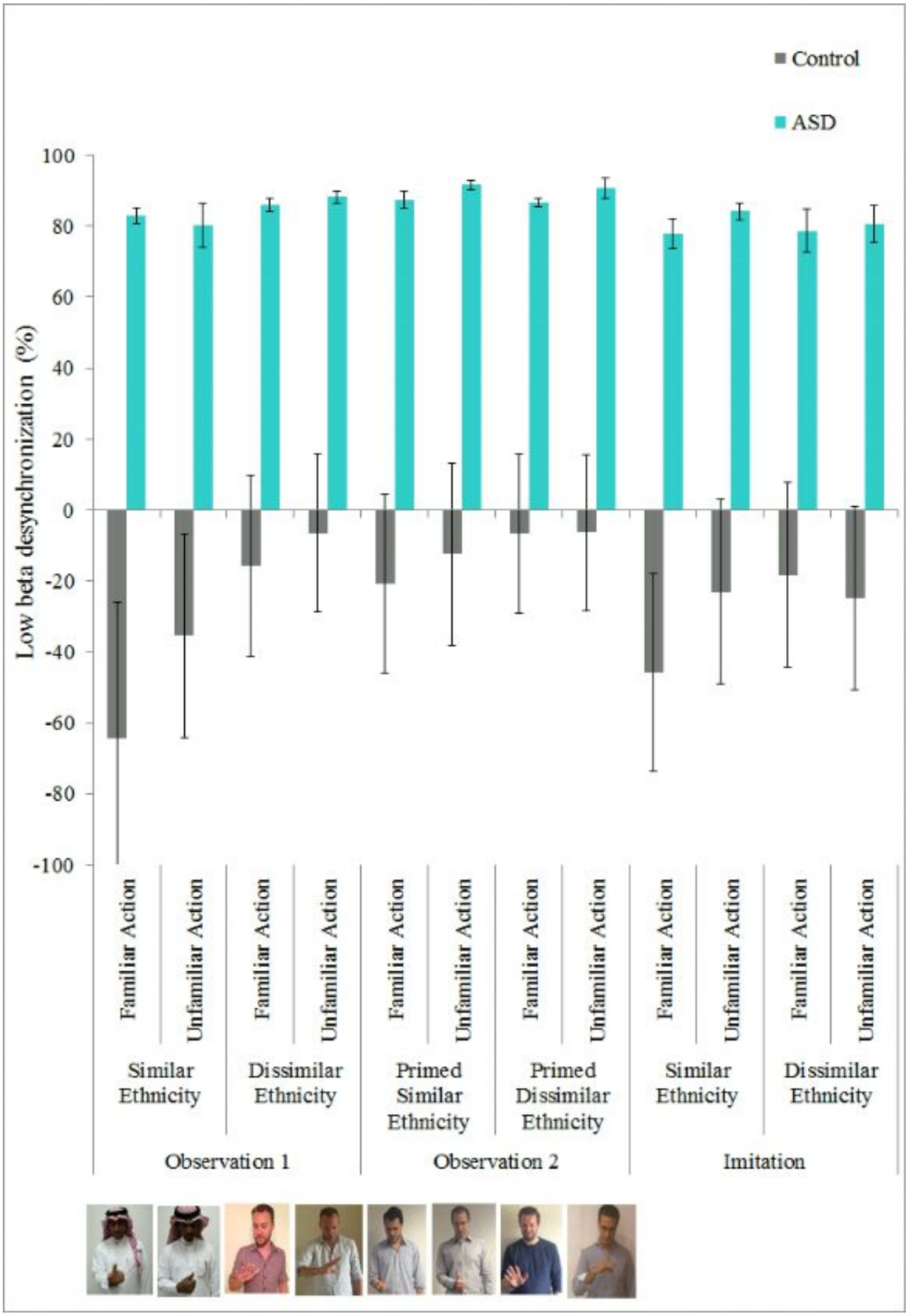

Control
ASD
Low beta desynchronization (%)
100
80
60
40
20
0
-20
-40
-60
-80
-100
Familiar Action
Unfamiliar Action
Familiar Action
Unfamiliar Action
Familiar Action
Unfamiliar Action
Familiar Action
Unfamiliar Action
Familiar Action
Unfamiliar Action
Familiar Action
Unfamiliar Action
Similar Ethnicity
Dissimilar Ethnicity
Primed Similar Ethnicity
Primed Dissimilar Ethnicity
Similar Ethnicity
Dissimilar Ethnicity
Observation 1
Observation 2
Imitation

Figure 27 The bar chart represents the percentage of event-related changes in low beta power for control and ASD groups across 12 conditions. Error bars represent the standard error of the mean.

A four-way mixed 2 x 2 x 2 x 3 ANOVA, was conducted to examine the effects of ethnicity (similar/ dissimilar), action (familiar/ unfamiliar) and task (*observation 1*/ *observation 2*/ imitation) as within-subject factors, and group (control/ ASD) as a between-subject factor on low beta suppression[3]. Results revealed a number of significant effects and interactions which are represented in Table 5.3.

	All data			Control group			ASD group		
	df	F-value	p-value	df	F-value	p-value	df	F-value	p-value
Group	1,24	18.17	0.001***	-	-	-	-	-	-
Ethnicity	1,24	7.75	.010**	1,12	7.86	.016**	1,12	0.04	0.83
Action	1,24	8.95	.006**	1,12	6.51	.025*	1,12	9.01	.011**
Task	2,48	8.85	0.001***	2,24	5.69	.009**	2,24	6.76	0.005**
Group*Ethnicity	1,24	7.26	.013**	-	-	-	-	-	-
Group*Action	1,24	4.19	0.052*	-	-	-	-	-	-
Group*Task	2,48	2.75	0.07	-	-	-	-	-	
Ethnicity*Action	1,24	3.35	0.08	1,12	3.45	0.08	1,12	0.001	0.98
Ethnicity*Task	1.6,38.2	4.45	.026*	2,24	3.05	0.06	1.1,13.5	1.96	0.18
Action*Task	1.2,28.8	0.38	0.57	1.2,13.9	0.66	0.45	1.2,15	0.45	0.55
Group*Ethnicity*Action	1,24	3.39	0.07	-	-	-	-	-	-
Group*Ethnicity*Task	2,48	1.43	0.25	-	-	-	-	-	-
Group*Action*Task	2,48	0.89	0.41	-	-	-	-	-	-
Ethnicity*Action*Task	1.3,32.2	0.51	0.53	1.3,15.2	0.46	0.55	2,24	0.52	0.59
Group*Ethnicity*Action*Task	2,48	0.43	0.66	-	-	-	-	-	-

* for p<.05, ** for p<.01 and *** for p<.001.

Table 16 Mixed & separate ANOVAs for low beta suppression across ethnicity, action and task

Is MNs activity impaired in ASD?

A main effect of group, $F(1, 24) = 18.16$, $p < .001$, $_p\eta^2 = .43$, demonstrated an increased level of low beta suppression in the control group ($M = -23.34\%$), compared to the ASD group ($M = 84.60\%$).

Is MNs modulated by action familiarity?

A main effect of action, $F(1, 24) = 8.94$, $p = .006$, $_p\eta^2 = .28$, demonstrated an increased level of low beta suppression for a familiar action ($M = 27.50\%$), compared to an unfamiliar action ($M = 33.74\%$). Analysis of the control group showed a main effect of action, $F(1, 12) = 6.51$, $p = .025$, $_p\eta^2 = .35$, revealing a greater suppression for a familiar action condition ($M = -28.61$) compared to an unfamiliar action ($M = -18.08$). Similarly, analysis of the ASD group showed a main effect of action, $F(1, 12) = 9.00$, $p = .011$, $_p\eta^2 = .43$, revealing greater suppression for a familiar action condition ($M = 83.61$) compared to an unfamiliar action ($M = 85.58$).

Is MNs modulated by similar ethnicity of the model?

A main effect of ethnicity, $F(1, 24) = 7.75$, $p = .010$, $_p\eta^2 = .24$, demonstrated an increased level of low beta suppression for similar ethnic actors ($M = 25.39\%$) compared to dissimilar ethnic actors ($M = 35.85\%$). Analysis of the control group showed a main effect of ethnicity, $F(1, 12) = 7.86$, $p = .016$, $_p\eta^2 = .40$, revealing a greater suppression for a similar ethnicity condition ($M = -33.65$) compared to a dissimilar ethnicity condition ($M = -13.04$).

Was MNs functioning influenced by IQ and hand imitation skills?

Correlational analyses revealed insignificant association between low beta suppression and intelligence quotient, $rs < -.39$, $ps > .11$. However, there was significant association between low beta suppression during both the imitation condition, $r = -.46$, $p = .059$, and the observation condition, $r = -.49$, $p = .046$, and behavioural imitation. Both revealed moderate correlation.

Theta frequency band (5.5-7.5Hz)

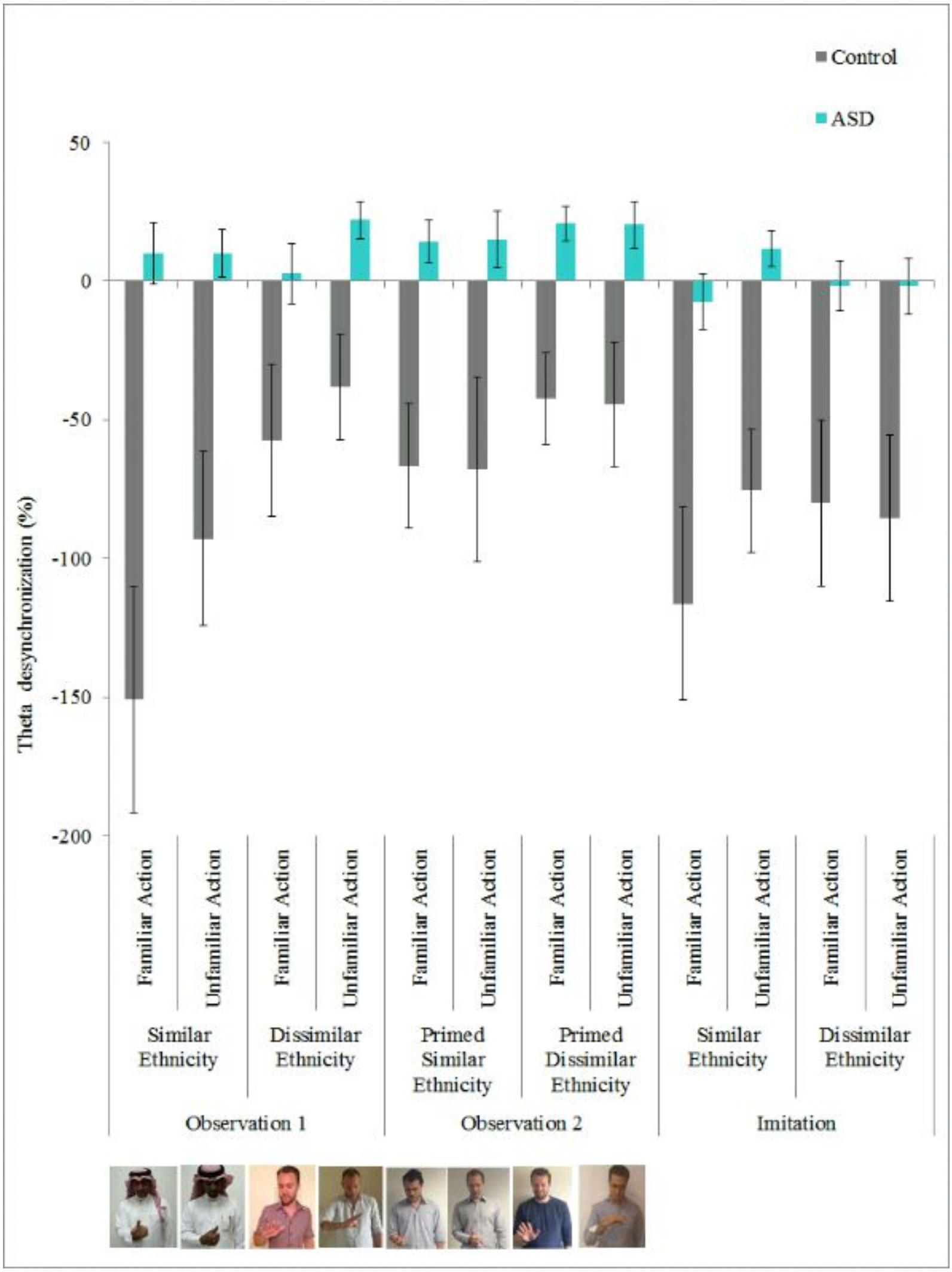


Figure 28 The bar chart represents the percentage of event-related changes in theta power for control and ASD groups across 12 conditions. Error bars represent the standard error of the mean.

A four-way mixed 2 x 2 x 2 x 3 ANOVA, was conducted to examine the effects of ethnicity (similar/ dissimilar), action (familiar/ unfamiliar) and task (observation/ imitation) as within-subject factors, and group (control/ ASD) as a between-subject factor on theta suppression[4]. Results revealed a number of significant effects and interactions which are shown in Table 5.4.

	All data			Control group			ASD group		
	df	F-value	p-value	df	F-value	p-value	df	F-value	p-value
Group	1,24	10.53	0.003**	-	-	-	-	-	-
Ethnicity	1,24	16.73	0.001***	1,12	21.89	0.001***	1,12	0.01	0.96
Action	1,24	7.08	.014**	1,12	5.37	.039*	1,12	1.77	0.21
Task	2,48	15.45	0.001***	2,24	10.36	0.001***	2,24	9.09	0.001***
Group*Ethnicity	1,24	17.14	0.001***	-	-	-	-	-	-
Group*Action	1,24	2.42	0.13	-	-	-	-	-	-
Group*Task	2,48	4.94	.011**	-	-	-	-	-	-
Ethnicity*Action	1,24	6.07	.021*	1,12	7.03	.021*	1,12	0.01	0.93
Ethnicity*Task	2,48	5.96	.005**	2,24	5.36	.012**	2,24	2.04	0.14
Action*Task	2,48	3.12	0.053*	2,24	1.91	0.17	2,24	2.30	0.12
Group*Ethnicity*Action	1,24	5.73	.025*	-	-	-	-	-	-
Group*Ethnicity*Task	2,48	3.98	.025*	-	-	-	-	-	-
Group*Action*Task	2,48	0.79	0.46	-	-	-	-	-	-
Ethnicity*Action*Task	2,48	1.36	0.26	2,24	0.91	0.41	2,24	4.58	.021*
Group*Ethnicity*Action*Task	2,48	1.23	0.30	-	-	-	-	-	-

* for p<.05, ** for p<.01 and *** for p<.001.

Table 17 Mixed & separate ANOVAs for theta suppression across ethnicity, action, and task.

Is MNs activity impaired in ASD?

A main effect of group, $F(1, 24) = 10.53$, $p = .003$, $_p\eta^2 = .31$, demonstrated an increased level of theta suppression in the control group ($M = -76.43\%$), compared to the ASD group ($M = 9.66\%$).

Is MNs activity modulated by action familiarity?

A main effect of action, $F(1, 24) = 7.07$, $p = .014$, $_p\eta^2 = .23$, demonstrated an increased level of theta suppression for a familiar action ($M = -39.11\%$), compared to an unfamiliar action ($M = -27.65\%$). Analysis of the control group showed a main effect of action, $F(1, 12) = 5.36$, $p = .039$, $_p\eta^2 = .31$, which demonstrated an increased level of theta suppression for a familiar action relative to an unfamiliar action.

Is MNs activity modulated by actor ethnicity?

A main effect of ethnicity, $F(1, 24) = 16.72$, $p < .001$, $_p\eta^2 = .41$, demonstrated an increased level of theta suppression for similar ethnic actors ($M = -42.56\%$), compared to dissimilar ethnic actors ($M = -24.20\%$). Analysis of the control group showed a main effect of ethnicity, $F(1, 12) = 21.89$, $p < .001$, $_p\eta^2 = .65$ which demonstrated an increased level of theta suppression for a similar ethnic model compared to a dissimilar ethnic model.

Does priming with a similar ethnic model facilitate action understanding in control and ASD?

Planned comparisons (paired sample t-tests) were conducted to explore the two-way interaction between ethnicity and task in the control group. Theta suppression was significantly higher while participants were observing a similar ethnic model ($M = -104.10\%$) relative to observing a dissimilar ethnic model during - *observation 1* ($M = -65.49\%$), $t(12) = -2.28$, $p < .042$, $d = -0.6$. In contrast, none of the ethnicity comparisons elicited an acceptable level of significance for *observation 2* or imitation (ts < -1.53, ps $> .15$).

Furthermore, when the action had been primed by a similar ethnic model, theta suppression was significantly lower during *observation 2* ($M = -54.32\%$) relative to *observation 1* ($M = -104.10\%$), $t(12) = -2.81$, $p < .016$, $d = -0.8$, and imitation ($M = -$

98.09%), $t(12) = -3.14$, $p < .008$, $d = .9$. Theta suppression did not differ between *observation 1* and imitation periods (ts < -.54, ps > .59). Similarly, when the action had been primed by a dissimilar ethnic model, theta suppression was significantly lower during *observation 2* (M = -56.08%) relative to the imitation period (M = -80.47%), $t(12) = 2.19$, $p < .049$, $d = .6$. The remaining comparisons did not elicit significant effects (ts < -.75, ps > .46).

Independent sample t-tests were run to explore the differences between the group effect of group*ethnicity*task interaction. As for similar ethnic person, comparisons of *observation 1* of a similar ethnic person demonstrated that theta suppression of the control group (M = -104.10%) was significantly higher compared to the ASD group (M = 6.39%), $t(24) = -3.17$, $p = .004$, $d = 1.24$. Similarly, comparisons of *observation 2* of similar ethnic person demonstrated that theta suppression of the control group (M = -54.32%) was significantly higher compared to the ASD group (M = 17.45%), $t(24) = -3.51$, $p = .002$, $d = 1.38$. In addition, comparison of imitation of similar ethnic person demonstrated that theta suppression of the control group (M = -98.09%) was significantly higher compared to the ASD group (M = -1.99%), $t(24) = -3.07$, $p = .005$, $d = 1.20$. As for dissimilar ethnic person, comparisons of *observation 1* of a dissimilar ethnic person demonstrated that theta suppression of the control group (M = -65.49%) was significantly higher compared to the ASD group (M = 16.11%), $t(24) = -3.20$, $p = .004$, $d = 1.25$. Similarly, comparisons of *observation 2* of dissimilar ethnic person demonstrated that theta suppression of the control group (M = -56.08%) was significantly higher compared to the ASD group (M = 13.13%), $t(24) = -2.42$, $p = .023$, $d = .95$. In addition, comparison of imitation of dissimilar ethnic person demonstrated that theta suppression of the control group (M = -80.47%) was significantly higher compared to the ASD group (M = 6.88%), $t(24) = -3.39$, $p = .002$, $d = 1.33$.

As for ASD, planned comparisons (paired sample t-tests), were conducted to explore the three-way interaction between ethnicity, action and task in the ASD group. During *observation 1*, for similar ethnicity, results revealed that none of the comparisons elicit

significance (*t*s < -.01, *p*s > .99).

In contrast for dissimilar ethnicity, comparisons revealed that theta suppression during a familiar action was significantly higher (*M* = 2.77%) relative to an unfamiliar action (*M* = 22.14%), *t*(12) = -2.18, *p* < .050, *d* = -0.60. Furthermore, when the action was familiar, the comparison did not reached significance (*t*s < .66, *p*s > .51). In contrast, when the action was unfamiliar, the suppression of similar ethnic model was significantly higher (*M* = 10.08%) relative to dissimilar ethnic model (*M* = 22.14%), *t*(12) = -2.85, *p* < .014, *d* = -0.79.

During *observation 2*, interaction of ethnicity and action revealed that none of the comparisons reached significance (*t* < -1.75, *p* > .10).

During imitation, for similar ethnicity, comparisons revealed that theta suppression was significantly higher for a familiar action (*M* = -2.27%) relative to an unfamiliar action (*M* = 15.60%), *t*(12) = -2.79, *p* < .016, *d* = -0.77. In contrast, for dissimilar ethnicity, comparison revealed that the theta suppressions during a familiar action and an unfamiliar action did not reach significance (*t* < .01, *p* > .98).

Furthermore, when the action was familiar, the theta suppression with a similar ethnic model was higher (*M* = -2.27%) relative to a dissimilar ethnic model, but it did not reach significance (*M* = -1.71%), (*t* < -.06, *p* > .94). However, when the action was unfamiliar, the theta suppression with a similar ethnic model was significantly lower (*M* = 15.60%) relative to a dissimilar ethnic model (*M* = -1.84%), *t*(12) = 3.30, *p* = .006, *d* = .09.

Does the similarity of actor ethnicity have an effect on motor resonance in the control?

Planned comparisons (paired sample t-tests), collapsed across task, were conducted to explore the two-way interaction between ethnicity and action in the control group. For similar ethnicity, comparisons revealed that theta suppression was significantly higher for a familiar action (*M* = -111.10%) compared to an unfamiliar action (*M* = -78.71%), *t*(12) = -2.69, *p* = .019, *d* = -0.7.

In contrast, for dissimilar ethnicity, comparisons revealed that theta suppression did not differ significantly between a familiar action (M = -59.91%) and an unfamiliar action (M = -56%), t = -.65, p = .52. Furthermore, when the action was familiar, theta suppression was significantly higher when the initial actor was of similar ethnicity (M = -111.10%) compared to when the initial actor was of dissimilar ethnicity (M = -59.91%), $t(12)$ = -4.48, p < .001, d = -1.2. Similarly, when the action was unfamiliar, theta suppression was significantly higher when the initial actor was of similar ethnicity (M = -78.7%) compared to when the initial actor was of dissimilar ethnicity (M = -55.99%), $t(12)$ = -3.15, p < .008, d = -0.9.

Independent sample t-tests were run to explore the differences between the group effect of group*ethnicity*action interaction. Comparisons of similar ethnic person performing familiar action demonstrated that theta suppression of the control group (M = -111.10%) was significantly higher compared to the ASD group (M = 7.29%), $t(24)$ = -3.69, p = .001, d = 1.44. Similarly, comparisons of similar ethnic person performing unfamiliar action demonstrated that theta suppression of the control group (M = -78.71%) was significantly higher compared to the ASD group (M = 12.25%), $t(24)$ = -3.24, p = .001, d = 1.27. In addition, comparisons of dissimilar ethnic person performing familiar action demonstrated that theta suppression of the control group (M = -59.91%) was significantly higher compared to the ASD group (M = 7.27%), $t(24)$ = -2.67, p = .013, d = 1.04. Similarly, comparison of dissimilar ethnic person performing unfamiliar action demonstrated that theta suppression of the control group (M = -55.99%) was significantly higher compared to the ASD group (M = 11.82%), $t(24)$ = -2.86, p = .008, d = 1.12.

Was MNs function influenced by IQ and behavioural skills?

Correlational analyses revealed insignificant association between theta suppression and intelligence quotient, rs < -.43, ps > .08. Similarly, there was no significant association between theta suppression and behavioural imitation, rs < -.25, ps > .31.

5.4 DISCUSSION

The aim of this experiment was firstly to answer: Do children with or without ASD during observation of a similar-ethnic model (Saudi) performing familiar communicative gestures, elicit greater neural activity of MNs compared to when observing a dissimilar-ethnic model (European) performing the same gesture. With regards to the first question, the data revealed some important findings.

For the control group, the effect of a similar ethnic model and a familiar action were clearly revealed by the significant neural differences across the experimental conditions. However, this relationship was not replicated in the behavioural data as no significant effects were observed for similar ethnicity or action familiarity.

As for the ASD group, neural data across the three bands were inconsistent in supporting the effect of ethnicity. As for alpha suppression, interaction of group*ethnicity elicited no difference between similar and dissimilar ethnicity; however, a similar interaction in the low beta band revealed greater low beta suppression for similar ethnicity compared to dissimilar ethnicity. Furthermore, theta suppression rather revealed within-data inconsistency in ethnicity*action*task interaction; similar ethnicity seemed to appear to be significant with an unfamiliar action during observation 1, and dissimilar ethnicity seemed to appear to be significant with an unfamiliar action during imitation. In this context, there is no precise evidence to reject or accept the claim. For that reason, we looked in each frequency band for possible further statistical operations.

As for the alpha frequency band, we followed up on the marginal interaction group*action*task further, to inspect the separated ANOVAs for each group. Analysis of the ASD group revealed three major findings; firstly, the main effect of ethnicity was not significant; secondly, ethnicity*task interaction revealed a significant difference for similar ethnicity compared to dissimilar ethnicity in imitation conditions only; thirdly, the higher alpha suppression for observing a similar ethnic model compared to observing a dissimilar ethnic model, in ethnicity*task interaction, was, by the result,

driven by the control group.

As for the low beta frequency band, we explored the marginal interaction group*ethnicity*action, with follow up ANOVAs for each group. Analysis of separated ANOVAs revealed that the main effect of ethnicity was significant in the control group; neither the main effect of ethnicity nor other interactions reached significance in the ASD group.

As for the theta frequency band, our interest here was not limited to inspecting whether the neural activation established a significant difference between ethnicities; our main interest was in exploring the consistency of theta desynchronization, with that of alpha and low beta, in this age population. Accordingly, it is important to remark that ethnicity and action manifested as components of the three-way interaction in the theta band. Furthermore, exploring this interaction revealed that the suppression of the theta frequency band did not fully correspond with that of alpha and low beta bands. Specifically, in the theta band, no significant effects for familiar actions were observed and no effects for the ethnicity of the model. Unlike the other bands, neural activation of theta data did not demonstrate significant differences between observing familiar gestures relative to unfamiliar gestures, but did demonstrate significant neural activation for imitating a dissimilar ethnic model performing an unfamiliar gesture, in contrast to a similar ethnic model.

To understand the source of these inconsistent outcomes, the three-way interaction was broken down into two-way interactions: ethnicity*task, action*task, and ethnicity*action. For ethnicity and task interaction, none of the similar and dissimilar ethnicity comparisons was significant at any of the task levels - a finding consistent with the other bands; however, neural activation with people of familiar ethnicity at the third task level (imitation) was exceptionally high compared to that of the second task level (observation 2). As for action*task, none of the familiar and unfamiliar gesture comparisons were significant at any of the task levels. In addition, neural activation with unfamiliar gestures at the third task level (imitation) was exceptionally high compared

to that of level 1 (observation 1) and level 2 (observation 2). Furthermore, ethnicity and action interaction revealed no significant comparisons.

It seems, initially, that there is some inconsistency in the neural activation between the theta band and the other two bands. Firstly and critically, it appears that the source of disagreement stems from imitation suppression, which was not limited to the theta band. ASD participants demonstrated significant neural activation for imitating a similar ethnic model in the alpha band as well. In fact, having significant neural activation in the imitation condition, for either ethnicity, does not necessarily mean that the elicited neural activity was generated by correct actions, due to the limited corresponding behavioural data. Secondly, although the interaction between action and task elicited no significant effect for familiar gestures, the mean values for the neural activation of observing familiar gestures was higher at observations 1 and 2, compared to unfamiliar gestures.

These analyses lead us to conclude that the ASD group show no detectable difference between observing a similar ethnic model and a dissimilar ethnic model, across the alpha, low beta, and theta frequency bands. Furthermore, significant neural activation with similar ethnic models during the imitation condition was not significant enough to validate the variance between ethnicities.

As testing our second hypothesis was subject to demonstrating significant neural sensitivity to a similar ethnic model, relative to a dissimilar ethnic model, current data arguably suggests no effect of ethnicity on action understanding or imitation in children with ASD.

Explaining these data in the light of the development of this effect revealed that infants are born with the ability and tendency to attend to the details of human faces (Meltzoff & Moore, 1977). When they are primarily exposed to one ethnicity, they learn to recognise those racial features best (Bar-Halm et al., 2006), building prototypical models of faces based on those they are exposed to (Kelly et al., 2005). In this regard, a specific

ethnicity modulation appears to stem from contact with other races' 'familiarity' (Meissner et al., 2005).

The notion of 'contact' or 'configural-featural hypothesis' (Meissner & Brigham, 2001) literally implies that those who have been raised in mixed ethnic communities, or in a foreign country, where they experience early exposure to other ethnicities, should represent, by practice, experience and exposure 'familiarity,' reasonable capability for face processing and intention understanding. Hence this reflects how much interaction one has dedicated to members of other races.

Support for this argument is mirrored by researchers focusing on the effect of culture and ethnicity change on neurocognitive processes. Losin et al. (2012) illustrated that American adults of European, African, or Chinese racial backgrounds, who tried to imitate actors making meaningless gestures, had greater neural activation of frontal, parietal, and occipital areas as measured by fMRI, than when trying to imitate people of the same race performing the same gestures. This seems in line with Elfenbein and Ambady (2002) who demonstrated that a same race bias was shown in visual preference tasks by Caucasian-Israeli infants living in a primarily Caucasian population, as well as by African-Ethiopian infants living in a primarily African population.

In comparison, African-Israeli infants living in a primarily Caucasian environment did not show a same race bias. These findings are consistent with a meta-analysis that reported increased accuracy on emotion recognition tasks when expressed and recognised by members of the same national, ethnic, or regional group. In contrast, decreased accuracy was reported when cultural groups had regular exposure to one another. This supports the finding of another meta-analysis in which 'contact' was found to mediate similar race preference; hence suggesting that individuals from integrated populations (i.e., mixed ethnicities) are less likely to show a same ethnicity preference (Meissner & Brigham, 2001).

One possible concern may arise through questioning the independence of action

familiarity effect, in isolation of the effect of similar ethnicity, as this directly concerns the usefulness of similar ethnicity involvement. In other words, was it possible for the Saudi children to operate the motor resonance effectively, despite the actor ś ethnicity? This, statistically, should result in significant difference when putting the ódissimilar ethnicity ʾunder control, and manipulating the familiarity of the action. It would not be the case (p = .52), however, when putting the śimilar ethnicity ʾunder control and manipulating the familiarity of action that the significant difference would emerge (p = .019).

This suggests that undergoing a cognitive task that requires the participant to infer a meaning, intention or a goal for a familiar sign or communicative gesture is affected by ethnic familiarity and motor resonance, which seem to have reciprocal effect (Molnar-Szakacs et al., 2007). Hence, if the demonstrator is from the observers ʾethnic group, the child will find it easier to simulate the actor. Conversely, when the demonstrator and child were of different ethnicities, the participants found it harder to simulate the actor.

This finding concurs with Molnar-Szakacs et al., (2007), in which the Euro-American participants were less likely to simulate a Nicaraguan actor while performing American emblems. In addition, data from the current study seem in parallel with the proposal of the Reading the Mind in the Eyes task (Adams et al., 2009). When asked to interpret the mental states conveyed by photographs of eyes, adult Native Japanese and Caucasian American participants performed better when the eyes were of the same ethnicity compared to the óther ʾethnicities condition. FMRI imaging also revealed greater ʻ bilateral posterior superior temporal sulci ʾrecruitment in the same ethnicity condition than in the others.

The current experiment has two caveats to consider; firstly, present findings demonstrated significant effects for similar ethnic members, as there was a clear contrast between Saudis and Europeans on the basis of skin colour, physiognomy and wearable costumes; however, the outcomes of employing two ethnic groups with minimal contrast (e.g. Middle Eastern and Iranian) might draw a different conclusion.

Therefore, any conclusions drawn from the current data within a different context must be evaluated carefully. In addition, based on the contrast identified, determining which stimulus component was responsible for the ethnicity effect has not been mediated in the current experiment. Secondly, as has been outlined in chapter 4 (see section 4.4), due to the small sample of behavioural data, any conclusions must be drawn carefully.

5.5 CONCLUSION

Current results represent initial investigation of MNs in similar ethnicity effects on communicative gestures 'processing, an effect, to our knowledge, not previously studied in an ASD population. Earlier work, conducted by Molnar-Szakacs and colleagues (2007) targeted the imprint of culture on the neural system for action representation and understanding. In their transcranial magnetic stimulation (TMS), they measured CSE in adult Euro-American participants while they watched a Euro-American or Nicaraguan actor perform both culturally familiar and foreign emblems.

Consistent with the findings of experiments 1, 2, and 3, an effect of familiar action in the control and ASD groups was clearly revealed by the significance differences across experimental conditions in their EEG profile. One of the important findings revealed by the present experiment, was that preschool TD children, as was the case for adults (Molnar-Szakacs et al, 2007), showed that familiar action perception was modulated by the ethnicity of the actor. Specifically, observing an actor from a different ethnicity performing a familiar action seems to negatively affect motor resonance.

As for the effect of ethnicity, control children demonstrated consistent significant potentiality, across the three bands when engaging with an adult member from a similar ethnic group, compared with an adult member from a different ethnic group. Nonetheless, regardless of the ethnicity, they behaviourally produced a similar imitative ability. As for the ASD group, similar ethnicity seemed less likely to ease social engagement. However, an indication by TD children in the theta result suggested reasonable potentiality for simulating unfamiliar action when performed by a member of a similar ethnic group, relative to an out-group, suggesting the positive influence of a

similar ethnic member in learning novel actions.

Our data, in general, yielded consistent findings across the EEG bands; exceptionally, neural suppression of the theta band in the ASD group was not in line with those of the Alpha and low beta bands, in establishing neural differences for action familiarity.

Footnote

[1] Analysis of group differences in the baseline condition did not reach significance for the alpha frequency band ($t < -.59, p > .55$), low beta frequency band ($t < -1.63, p > .11$) or theta ($t < -1.54, p > .13$).

[2] When we conducted analyses on the alpha frequency band in the sub-samples, interaction between ethnicity and task for the control group (full sample: $F = 4.61, p = .020, {}_p\eta^2 = .27$/ sub-sample: $F = 5.44, p = .018, {}_p\eta^2 = .43$) and the ASD group (full sample: $F = 3.88, p = .035, {}_p\eta^2 = .24$/ sub-sample: $F = 3.75, p = .046, {}_p\eta^2 = .31$), remained consistently significant; however, although effect size of the sub-sample analysis appeared to be larger in the value, both effect sizes still to be within small range.

[3] When we conducted analyses on the low beta frequency band in the sub-samples, the main effect of group (full sample: $F = 18.16, p < .001, {}_p\eta^2 = .43$/ sub-sample: $F = 23.01, p < .001, {}_p\eta^2 = .60$), remained consistently significant; however, the effect size associated with main effect in the full-sample analysis was small, whereas the effect size associated with main effect in the sub-sample was moderate, which indicated a stronger effect.. The main effect of action (full sample: $F = 8.94, p = .006, {}_p\eta^2 = .28$/ sub-sample: $F = 12.20, p = .003, {}_p\eta^2 = .44$) remained consistently significant and yielded the same small range of effect size. The main effect of ethnicity (full sample: $F = 7.75, p < .010, {}_p\eta^2 = .24$/ sub-sample: $F = 4.80, p = .045, {}_p\eta^2 = .24$) remained consistently significant and yielded the same high range of effect size.

[4] When we conducted analyses on the theta frequency band in the sub-samples, the main effect of group (full sample: $F = 10.53, p = .003, {}_p\eta^2 = .31$/ sub-sample: $F = 9.70, p = .007, {}_p\eta^2 = .39$) remained consistently significant and yielded the same range of small effect size. The main effect of action (full sample: $F = 7.07, p = .014, {}_p\eta^2 = .23$/ sub-sample: $F = 3.39, p = .08, {}_p\eta^2 = .18$) showed that the sub-sample is marginal; given that the effect size associated with the main effect in the sub-sample analysis was within the same range of the effect size associated with the main effect in the full-sample analysis, the lack of significance in the sub-sample analyses was likely to be due to a reduction in power, rather than a methodological difficulty with the full-sample analyses. The main effect of ethnicity (full sample: $F = 16.72, p < .001, {}_p\eta^2 = .41$/ sub-sample: $F = 9.88, p = .007, {}_p\eta^2 = .39$) showed that the effect size of sub-sample analysis remained consistently significant and yielded the same range of small effect size.

As for the control group, the main effect of ethnicity (full sample: $F = 21.89, p < .001, {}_p\eta^2 = .65$/ sub-sample: $F = 10.25, p = .015, {}_p\eta^2 = .59$) remained consistently significant. Although the effect size associated with the main effect in the sub-sample analysis was smaller than that of the full-sample analysis, both were still within moderate range. The main effect of action (full sample: $F = 5.36, p = .039, {}_p\eta^2 = .31$/ sub-sample: $F = 2.54, p = .15, {}_p\eta^2 = .26$) showed that the sub-sample is not significant; given that the effect size associated with the main effect in the sub-sample analysis was within the same range of the effect size associated with the main effect in the full-sample analysis (small effect size), the lack of significance in the sub-sample analyses was likely to be due to a reduction in power, rather than a methodological difficulty with the full-sample analyses. In addition, interaction between ethnicity and action for the control group (full sample: $F = 7.02, p = .021, {}_p\eta^2 = .36$/ sub-sample: $F = 4.47, p = .07, {}_p\eta^2 = .39$) showed that the sub-sample is marginal;

given that the effect size associated with the interaction in the sub-sample analysis was within the same range of the effect size associated with the interaction in the full-sample analysis, the lack of significance in the sub-sample analyses was likely to be due to a reduction in power, rather than a methodological difficulty with the full-sample analyses. The interaction between ethnicity, action and task for the ASD group (full sample: F= 4.57, p = .021, $_p$ ŋ²= .27/ sub-sample: F= 1.70, p = .21, $_p$ ŋ²= .17) remained consistently within the same range of effect size.

. Interaction between ethnicity and task for the control group (full sample: F= 5.35, p = .012, $_p$ ŋ²= .30/ sub-sample: F= 4.88, p = .025, $_p$ ŋ²= .41) remained consistently significant. Although the effect size associated with the interaction in the sub-sample analysis was higher than that of the full-sample analysis, both were still within small range.

6 Discussion

In the current thesis, across four EEG experiments, we manipulated four critical factors that are likely to influence the degree to which individuals with ASD can understand and imitate other people's actions: familiarity of the motor action, familiarity of the person, similarity of the model's age and similarity of the model's ethnicity. The broad aim was to understand the influence of each factor on action understanding, and subsequently, on imitation ability in children between the ages of 3 and 5 years old with and without ASD.

In addition, upon demonstrating a significant influence, we aimed to investigate the possibility of utilising each factor as a priming stimulus, which might then facilitate action understanding of later unfamiliar and dissimilar models. Here, we utilised two categories of motor actions; culturally familiar communicative hand gestures (Nydell, 2002, p57), and meaningless hand gestures. We combined two methods of investigation to assess action understanding and imitation: desynchronization of EEG frequency bands to detect the neural activation of central MNs, and video recording for off-line, imitation performance analysis.

6.1 Effect of group

Here, the general trend of group effect, as illustrated by present EEG data across chapters 2, 3, and 5, showed significant main effect of group whereby the TD children elicited significantly higher alpha, low beta, and theta suppression relative to the ASD group. The single exceptional case appears in chapter 4 as effect of group was not significant across the three frequency bands.

Although present behavioural analyses were conducted on a small sample set across all experiments, the general trend of group effect was significant in demonstrating significant higher numbers of correct action imitations in TD children relative to ASD children; although the effect of group in Experiment 1 did not reach significance, it showed a trend towards an increased number of correct action imitations in the control versus the ASD group. On the other hand, in Chapter 3 - Experiment 2, the effect of group was not limited to correct imitation, effect of group was significant in incorrect

action imitations, which revealed significantly higher incorrect action imitations in the ASD group relative to the TD group. In reporting the significant effect of group, our data are consistent with (Bernier et al., 2007; Oberman et al., 2005; Théoret et al., 2005).

Full sample analysis of Experiment 3 demonstrated insignificant group effect with a small effect size, which is likely to indicate unbroken MNs functioning among ASD participants. Nonetheless, the variance between the full set of EEG data and the small set of behavioural data raises uncertainty about the MNs functioning and their role in imitation; thus, the need for sub-sample analysis was essential for equivalent comparison. It is worth noting that sub-sample analysis showed that the mean of EEG suppression in the control group become consistently higher across the three frequency bands than the mean of EEG suppression in the ASD group; however, the effect of group remains within small effect size range.

The absence of group difference of EEG data raises two possible arguments; first, MNs connectivity or activity might function normally and, thus, it would be plausible that impaired imitative performance was the result of different defects. Indeed, all ASD participants -except for 1- and all control participants -except for 2- deformed the same sample that participated in Experiment 4. Hence, although the main effect of group in Experiment 4 was consistently significant across the three EEG frequency bands, it does not seem statistically plausible that the MNs of ASD group were functioning normally. Second, MNs connectivity or activity is functioning abnormally; nonetheless, MNs seem to be activated when someone can define themselves with others. Across the literature which experimentally appraises the effect of some social characteristics (e.g. person familiarity) on ASD's neurophysiological profile, there seem to be circumstances in which MNs activity in ASD was statistically insignificant relative to typically matched group (Oberman, et al., 2008). Within this context, it seems to be hard to build a definite conclusion regarding age similarity effect on EEG attenuation at this stage, due to the lack of similar empirical evidence.

The stream of this argument leads logically to question the role of MNs in imitation; in particular in the case of both insignificant and significant effect of group on behavioural performance in Experiment 1. The inconsistent behavioural data raises three possible

arguments: first, as for the insignificant effect of group on behavioural performance, accepting that the behavioural imitation was preserved in the ASD sample, this will statistically contradict the behavioural data of Experiment 2 in which the same ASD sample was recruited to participate, and in which the mean of their correct behavioural performance was significantly lower while the mean of their incorrect behavioural performance was significantly higher than the control group. Second, accepting that the MNs have no role in imitation, this will statistically contradict the EEG data of current experiments, and, theoretically, the previous literature. The activation of MNs during a certain task is evidence of their involvement. No matter the range or the type of action, MNs are shown to be recruited during imitation of simple finger movements (Iacoboni, et al., 1999), and complex motor acts (Buccino, et al., 2004) and, therefore, no MNs activity should be detected during imitation conditions if MNs have no role; which apparently is not the case, and, thus, this seems also an incongruous speculation. Third, accepting that the MNs have a role in imitation, but imitation is not solely governed by MNs activity, this synchronises our finding and fits well with contradictory literature. It touched upon the claim that MNs are a necessary condition for imitation, but not sufficient (Rizzolatti and Sinigaglia, 2006). An individual still requires a control system to govern the mirror mechanism, in which the detectable actions are properly reproduced as a performance. Without a control system, an individual would compulsively replicate every single possible motor action. For this reason, the frontal lobe, which involves inhibition and executive function skills, is largely assumed to be involved in any actions in which the individual makes a decision about imitating or disregarding the action. Therefore, there seems to be a complex system underlying the capacity to imitate. Further support for this account is reflected by group differences in regions that do not literally correspond to MNs areas, which were established by evidence from a number of brain structure studies. These regions include the cerebellum (Brambilla, et al., 2003; Toal, et al., 2010), the fusiform (Toal, et al., 2010; Duerden, et al., 2012) and the cingulate and insula (Duerden, et al., 2012), but not in IFG or IPL.

The involvement of various cognitive mechanisms and other brain regions in imitation, other than MNs, would explain the variation in imitation among ASD samples; however, it remains unclear as to why the ASD participants of Experiment 1 & 2 would present

variant behavioural performance. The key difference between the two experiments was methodological; the task. Literature shows that the task is one of the evident factors that directly affect the imitation (Mundy, et al., 1986). In Experiment 1, each single observation condition was immediately followed by imitation condition and, thus, the behavioural performance was a result of observing stimuli that have direct and explicit characteristics (e. g. familiar person/ familiar action). Whereas in Experiment 2, each single observation condition (*observation1*) –which procedurally mimics the design of Experiment 1- was followed by a second observation condition (*observation 2*) which consistently depicts a fixed characteristic (e.g. unfamiliar person).

6.2 Effect of action familiarity

Our EEG data consistently showed that both children with ASD and TD children demonstrated significant effect of a familiar action across all four experiments, in the alpha, low beta, and theta bands. These were in agreement with current behavioural data; in particular, children with ASD and TD children revealed a significantly higher number of correct hand actions for imitating a familiar action relative to an unfamiliar action. In addition, children with ASD revealed significantly more incorrect hand actions for imitating an unfamiliar action relative to a familiar action. Our EEG data seem to synchronise those of Calvo-Merino et al. (2005) and Marshall et al. (2009) in showing that the observer's experience with observed motor actions has a major role in the amount of neural activation.

6.3 Effect of person familiarity

In chapters 2 and 3, the effect of person familiarity, was manipulated by comparing actions performed by the child's own parent versus a stranger (another child's parent), and showed significant differences across experimental conditions. In particular, TD children demonstrated an effect of familiar person, evident by significant alpha and low beta suppression for a familiar person, across both experiments. However, the behavioural data were not consistent, as the effect of person familiarity appears in Experiment 2 only.

The ASD children demonstrated an effect of familiar person, evident in significant alpha and low beta suppression for a familiar person, across both experiments as well. In addition, the behavioural findings revealed consistently similar trends in which the most correct imitation actions were elicited when imitating a familiar person, and the highest number of incorrect imitation actions was elicited when imitating an unfamiliar person.

These results are consistent with the previous literature in validating the critical effects of familiarity in facilitating action understanding and imitation in ASD (Wolff & Barlow, 1979; Oberman et al., 2008). Nonetheless, the theoretical meaning of Oberman et al.'s (2008) data were limited by the simple experimental design, in which the video stimuli depicted changeable agents (stranger, mother, and self) performing the same intransitive hand action (i.e. opening and closing hand; see Figure 6.1). Hence the intransitive hand action was repeatedly presented and therefore it was not possible to decide whether MNs were modulated as a result of observing a familiar agent (i.e. 'my hand'), or a familiar action (i.e. 'this is my performed action').

Figure 29 In Oberman et al.'s design, each of the three video stimuli depicted different agents (stranger, guardian or mother and self), repeatedly performing the same intransitive hand action (opening-closing hand).

In this thesis, the experimental design was purposefully built to investigate the effects of action familiarity and person familiarity independently, along with their interactions. Therefore, we employed two different familiar actions and two different unfamiliar

actions, along with different stranger adults that appear in each trial. To this end, we were able to ensure that familiarity of action was not due to it being presented twice. In addition, the strangeness of the actor was maintained across trials as each one depicted a different strange adult.

Results showed that both control and ASD children generally demonstrated the highest level of alpha and low beta suppression when they were observing a familiar person (i.e. their parent) performing a familiar action. However, children with ASD, compared to control children, failed to demonstrate similar suppression when observing an unfamiliar person (i.e. a stranger) when they were performing a familiar action. Furthermore, children with ASD demonstrated less alpha and low beta suppression when they were observing a familiar person performing a meaningless (unfamiliar) action compared to a familiar action; these were two effects that could not be observed by Oberman and colleagues' work (2008).

These effects seem to raise two major suggestions: firstly, the facilitated effect of person familiarity allows children with ASD, while observing their parent, to operate motor resonance for meaningful gestures effectively. Secondly, despite the fact that imitating intransitive hand actions is impaired in ASD children compared to transitive actions (object-directed action), the meaning that could be yielded by this intransitive action plays a role in the level of MNs activation even with the great effect demonstrated by having a familiar actor. This was directly shown by the reduction of MNs activation when observing a familiar person performing a meaningless action.

6.4 Effect of person age similarity

In Chapter 4, participants in both groups appeared to demonstrate increased engagement with the same aged actors, as reflected by significantly increased neural desynchronization when observing the unfamiliar child models compared to the unfamiliar adult models, across all frequency bands. In addition, behavioural analyses of the imitation period showed that children were able to perform more correct imitations when imitating an action performed firstly by a similar aged actor compared to an action first performed by a dissimilar aged actor, and also, significantly more correct

imitations when they were imitating a familiar action compared to an unfamiliar action.

In the ASD group, this action effect interacted with age such that the familiar action advantage was only evident when children were imitating an action that was first performed by a dissimilar aged actor, and not a similar aged actor (where performance was already good). Interestingly, previous exposure to a similar aged model did not facilitate action understanding when that same action was subsequently performed by a dissimilar aged model. This result was drawn from the interaction of age*task interaction in the alpha band and low beta band, as the theta band demonstrated no significant interaction. Explaining the present findings within an integrated framework of social cognitive accounts suggests that the selective neural activations that children demonstrated towards their peers mirror the notion that not all presented models will be imitated equally; the observer will be more influenced by those people who symbolise engaging qualities (Bandura, 1977).

This is convergent with the proposal of Epstein (1966) in showing that the characteristics of an observed model (e.g., age, ethnic status) influence the degree to which social attitudes and behaviour will be produced by others. Indeed, research evidence has shown that upon perceiving others as potential social partners, children start to increasingly integrate their own activities with unfamiliar peers, and imitate their actions (Maudry & Nekula, 1939; Harlow, 1969), which allows them to elaborate a social engagement with an unfamiliar (but same-aged) peer, compared to a familiar 'mother'(Eckerman et al., 1975). According to Meltzoff (1990), this synchronises with the 'like-me' view, in which peer preference stems from children's ability to recognize others as being similar to the self, which in turn requires linking an observed action to the self to understand simulations of others' actions and mental states.

6.5 Effect of person in terms of ethnic similarity

Imitation data provided the major supportive evidence for the existence of MNs in humans, however, such imitation behaviour was not produced by the macaque monkey (Rizzolatti & Craighero, 2004), which narrows the initial empirical speculations about MNs' functions in action understanding. Nevertheless, Hickok and Hauser (2010)

identified two forms of imitations which are in the repertoire of macaques: observational learning and cultural transmission.

For instance, adult macaques can replicate a sequence of movements (e.g. pressing buttons to select certain type of pictures) by watching another macaque performing the same movements (Subiaul et al., 2004). They were also shown to perform stone-handling behaviour, which has been shown to spread through populations. This is a unique socially transmitted behaviour in Japanese macaques because it occurs in the absence of any tangible direct benefit (Heyes, 1996).

In Chapter 5 (Experiment 4), we investigated whether TD and ASD children would elicit greater neural activity of MNs when observing an action performed by a person of similar ethnicity (Saudi) compared to a person of dissimilar ethnicity (European), and whether this bias would impact on their imitation performance.

One of the important findings revealed in Experiment 4 was that in preschool TD children, familiar action perception was modulated by the ethnicity of the actor, as has been shown previously in adults (Molnar-Szakacs et al., 2007), Specifically, Molnar-Szakacs et al. found that observing an actor from a different ethnicity performing a familiar emblem action negatively affects the motor resonance, reflected in lower neural activation (see Figure 6.2).

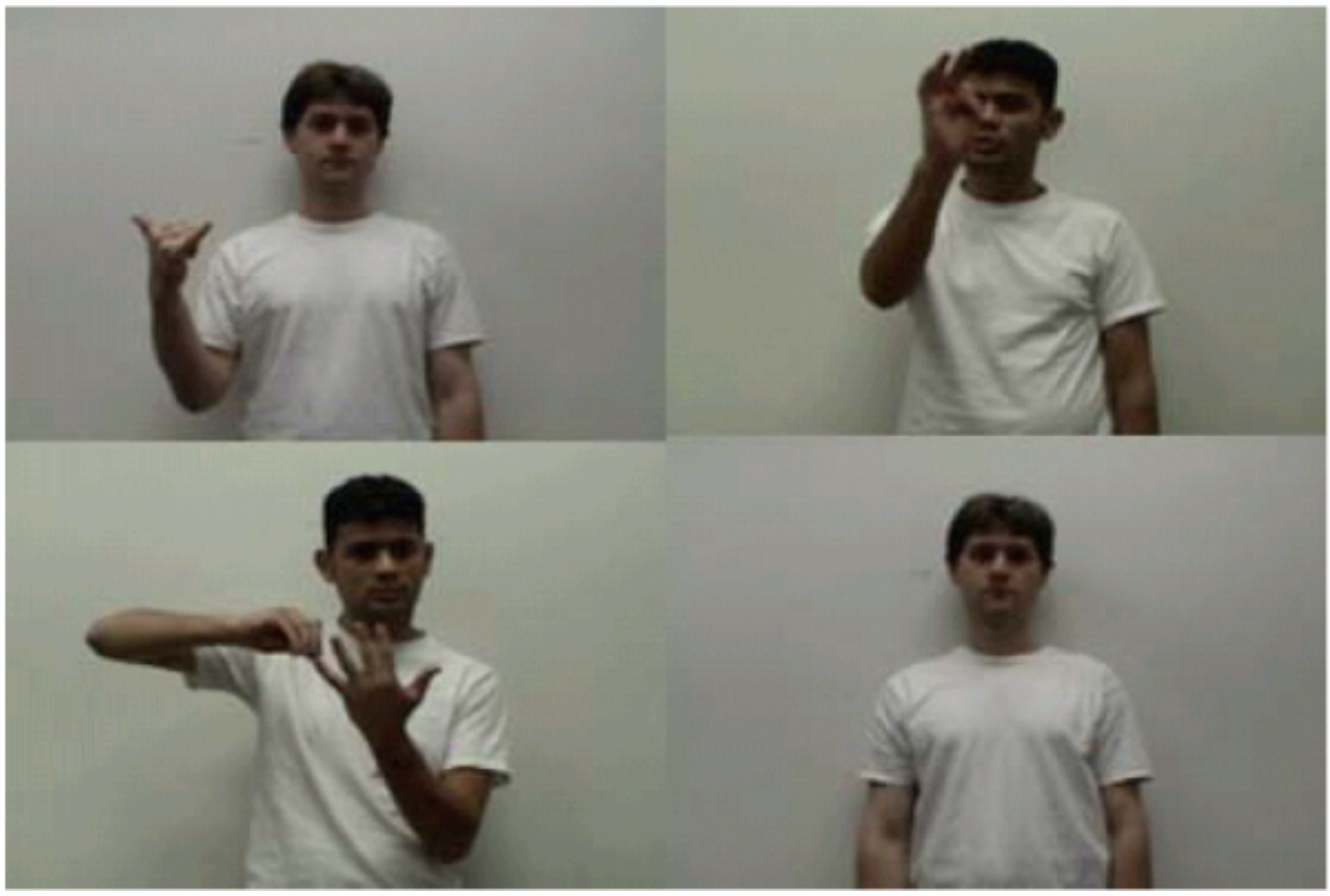


Figure 30 Molnar-Szakacs et al. (2007) demonstrated that observing a Nicaraguan actor performing Euro-American emblems affects motor reasonance. Motor resonance thus appears to be modulated by cultural factors.

As for effect of ethnicity in Experiment 4, TD children demonstrated consistent significant suppression across the three EEG bands when observing and imitating the behaviour of an adult from a similar ethnic group, compared to an adult from a different ethnic group. Moreover, EEG suppression in the theta band suggested that TD children are facilitated in simulating an unfamiliar action when it has been performed by a member of a similar ethnic group, relative to a member of a dissimilar ethnic group, suggesting the positive influence of a similar ethnic member in learning novel actions.

Nonetheless, behavioural analysis of imitation showed that TD children produced similar imitation ability, regardless of the initial actor ś ethnicity. In the ASD group, similar ethnicity seemed less likely to ease social engagement. In particular, as for the alpha frequency band, we followed up on the marginal interaction group*action*task further to inspect the separated ANOVAs for each group.

Analysis of the ASD group revealed three main findings: firstly, the main effect of

ethnicity was not significant; secondly, ethnicity*task interaction revealed a significant difference for similar ethnicity compared to dissimilar ethnicity in imitation conditions only; thirdly, the higher alpha suppression for observing similar ethnic models compared to observing dissimilar ethnic models, in ethnicity*task interaction, was, by the result, driven by the control group. As for the low beta frequency band, we also explored the marginal interaction group*ethnicity*action which revealed that neither the main effect of ethnicity, nor other interactions reached significance in the ASD group. As for the theta frequency band, the result revealed two important findings: firstly, the main effect of ethnicity did not reach significance; secondly, the three-way interaction in ethnicity*action*task revealed no significant effect for similar ethnicity.

Explaining these data in the light of the development of this effect draws upon evidence that infants are born with the ability and tendency to attend to the details of human faces (Meltzoff & Moore, 1977). When they are primarily exposed to one ethnicity, children learn to recognise those racial features best (Bar-Halm et al., 2006), building prototypical models of faces based on those they are regularly exposed to (Kelly et al., 2005). In this regard, a specific ethnicity modulation appears to stem from contact with other races 'familiarity '(Meissner et al., 2005). The notion of 'contact 'or 'configural-featural hypothesis '(Meissner & Brigham, 2001) implies that individuals who are raised in mixed ethnic communities, or in a foreign country where they experience early exposure to people from other ethnicities are, in turn, more familiar with these types of face and, hence, possess a reasonable capability for face processing and intention understanding. This ethnicity effect then reflects how much interaction one has dedicated to other race members. This in-group effect, as reflected by our TD data, did not play a role in evoking MNs in ASD, which may relate to previous literature in arguing a lower tendency to attend to human faces (Swettenham et al., 1998), which necessarily affected their recognition of racial features.

In summary, as discussed in Chapter 1, the theoretical notion of the 'broken mirror neurons 'theory in ASD (Ramachandran & Oberman, 2006), and the corresponding deficiency in their capacity to simulate the mental states of others, arises from a failure to simulate an observed agent as they perform actions that have simple social meanings. The role of person familiarity, age and ethnic similarity emerge through a plausible

capacity of ASD individuals to: firstly, 'simulate' an unknown actor performing a communicative hand action that depicts social inference, when this action was primed by a familiar model; Secondly, to simulate 'peer' models while performing a communicative hand action.

The improved capacity for action understanding in individuals with ASD when familiar/ similar actors were used, implies defects in their neural encoding. Nonetheless, it also seems to imply that even faulty MNs can be triggered under specific circumstances and when more sensitive recording techniques are employed. However, the experiments presented here provided no statistical evidence to suggest that simply repeating observation of an unfamiliar (meaningless) or familiar hand action with an unfamiliar actor results in enhanced capacity to simulate the actor.

6.6 Links with previous EEG studies of MNs and ASD

An important point arises regarding how the present work relates to the existing literature. There are seven major studies that have investigated MNs in ASD using EEG: Oberman et al., 2005; Bernier et al., 2007; Martineau et al., 2008; Oberman et al., 2008; Raymaekers et al., 2009; Fan et al., 2010; Bernier et al., 2013. In the following discussion, we will interpret the findings reported within this thesis in the light of plausible elements of divergence or agreement with previous studies. This discussion will focus on three main dimensions: EEG attenuation during observation, EEG attenuation during imitation and lastly, behavioural imitation performance.

In Oberman et al., (2005), EEG activity was recorded while ASD and TD participants were presented with two non-biological motion stimuli and one biological motion stimulus, which depicted a meaningless intransitive hand action (i.e. opening-closing hand), as well as during a period of action execution. Results showed that the ASD group, compared to the TD group, demonstrated intact mu (8-13Hz) suppression during execution of an action, but showed significantly less mu suppression during action observation. Similarly, Bernier et al., (2007) compared the EEG attenuation and behavioural performance of an adult ASD sample with TD adults in four action conditions: resting, observing, executing, and imitating.

In agreement with Oberman et al., ASD participants demonstrated significantly increased mu rhythm (8-13Hz) suppression during execution, compared to the observation condition. In addition, their behavioural performance revealed significantly poorer imitation performance compared to control adults in the imitation condition. Compared to Oberman et al. (2005) and Bernier et al. (2007), our ASD group did not consistently[1] demonstrate higher alpha (8-12Hz) suppression during execution conditions compared to observation conditions. Sample characterization is assumed to contribute to the inconsistent findings between these studies (Jones & Klin, 2009), where a large age range seems difficult to reconcile with the heterogeneity of ASD.

This implies that one of the key differences in the execution condition between our work and Oberman et al.'s is the maturity of imitation skills. As such, the effort to imitate in our pre-school age sample, with a small age range (3-5 years) and average age of 4.4 years, is unlikely to be equivalent to that required by the wide range of older children and adults tested in Oberman et al., which ranged from 6 to 46 years and an average age of 16.6 years. Nonetheless, a number of findings have revealed higher suppression for execution compared to observation conditions, even in a young sample with ASD.

For example, Raymaekers et al. (2009) tested a sample of individuals with high functioning ASD, which consisted of children between 8 and 13 years, and aged/ IQ matched controls, on an experiment based on Oberman et al.'s (2005) paradigm. Similar to the results reported here, their findings differed from Oberman et al. (2005) as control and ASD children both demonstrated significant mu suppression during execution and observation of the hand action. However, in line with Oberman et al., both groups demonstrated higher suppression during action execution than observation. This suggests that variation in sample characteristics is unlikely to be the sole cause of lower mu suppression during imitation. Instead it is possible that during these action execution periods children might not have been moving their hand continuously, which resulted in low suppression.

Our sample might arguably be more comparable to those of Martineau et al. (2008), whose ASD sample consisted of children between 5 and 7 years, who were compared to

TD children matched in gender and age. In their experiment, three EEG ranges were used: theta 1 (3 –5.5Hz), theta 2 (5.5 –7.5Hz) and alpha 1(7.5 –10.5 Hz) suppression were recorded during observation of videos showing non-human actions, intransitive human action or still scenes of non-human stimuli. Control children demonstrated desynchronization in the motor cerebral cortex and the frontal and temporal areas, solely during observation of human actions; however, no such desynchronisation was found in ASD children.

However, unlike our experiments, this study did not record the imitation performance of the children with ASD, thus we cannot compare the behavioural performance or changes in mu rhythm between observation and imitation periods in this and our experiments. Nevertheless, they did include a biological motion condition that consisted of an intransitive action (a leg action). Researchers have hypothesized that children with ASD, compared to TD children, will fail to show mu suppression when observing biological motion due to MNs dysfunction. Here, we may question why the activation of observing an action in ASD could be different to that in TD children? An important suggestion was offered by Press et al. (2010); they argued that the nature of the task might have a role in evoking lower MNs activation, even if the system itself is intact. Specifically, in experimental paradigms where ASD participants are not required to produce any imitation performance, the attentional effort that is required during passive observation is less likely to reflect the optimum level of their attention. In that regard, our data - even during the observation periods - might not be comparable with their findings due to methodological differences.

On the other hand, it is worth noting that despite the plausibility of Press and colleagues' argument, they provide no explanation regarding why differences in levels of attention do not lead to comparable effects in TD children. If the attention of TD children could be affected by the nature of the task, then it is possible that the absence of a group difference is mediated by the same factor.

Another major point to consider is that the diagnostic methods and tools vary greatly amongst studies. In the experiments reported here, we employed the Revised Autism Diagnostic Interview (ADI-R, Lord et al., 1994), which is similar to many previous

studies (Bernier et al., 2007; Fan et al., 2010; Martineau et al., 2008; Raymaekers et al., 2009). Our ASD sample all met the criteria of the ADI-R, but most importantly, they had all already been formally diagnosed with an ASD by a clinician, based on DSM-IV criteria (American Psychiatric Association, 2000).

In addition to differences in diagnostic criteria, details of symptoms and severity are not usually provided (e.g. Oberman et al., 2005), which restricts the comparisons and justification of variance between studies. This raises an important point regarding the degree to which imitation abilities themselves might be linked to MNs activity in ASD. For example, in an EEG study by Fan et al. (2010), mu rhythm (8-13Hz) was measured in ASD participants who ranged in age from 11-26 year old, with a matched control group, while they were executing a transitive hand action, observing the transitive hand action, or observing moving dots. The findings revealed no significant difference in mu attenuation between the groups during observation or imitation of transitive hand actions. However, behavioural data revealed poor imitation performance in ASD participants, despite the intact mu attenuation. The researchers inferred that this provides evidence against the abnormality proposal of MNs in ASD populations.

Our correlational analyses were partially in line with those of Bernier et al. (2007) in showing a correlation between mu suppression and imitation performance. Our correlational analyses from different perspective were partially in line with those of Oberman et al. (2008). In their study, though the ASD group showed impairment in imitation, this impairment was not significantly associated with the EEG suppression. In addition, our correlational analyses were not in line with those of Bernier et al. (2013) in showing a correlation between mu suppression (observation condition) and imitation performance (facial but not hand). Our correlational analyses were also not in line with those of Raymaekers et al. (2009) in which their data showed correlation between mu suppression and intelligence.

Although Fan et al's findings align with Bernier et al. (2007), whose adult ASD sample demonstrated poor performance in the imitation task but intact mu suppression during imitation, Bernier et al. (2007) reported a correlation between mu suppression and imitation competence, which was not found in Fan and colleagues' data. These

differences, which could be found in our data, relate to the debate that was raised by Warreyn et al. (2013) in questioning the link between the strength of the mu attenuation and the level of social communicative abilities, such as language and imitation. Indeed, imitation is not one of the ASD diagnostic criteria, and thus the imitative skills of ASD individuals are not consistently determined or related to MNs activity. This individual variation in imitation skills among individuals with ASD might therefore contribute to inconsistent findings across studies.

This individual variation account is supported by Bernier et al. (2013), whose recent EEG study investigated mu rhythm (8-13Hz) in 6 years old ASD participants with impaired cognitive abilities, compared to matched TD children, during observation and execution of goal-directed hand actions. The findings revealed that, regardless of the group membership of the participants, reduced or absent MNs activity during the observation condition was associated with impaired imitation skills. Their finding thus appears to marginalize the effect of population categories and put greater emphasis on general imitation skills. However, these results should be taken with caution since a quarter of the ASD sample in Bernier et al.'s study had participated in a two-year intervention of imitation training, which is likely to be responsible (at least in part) for the absence of a between-group difference.

The difference between transitive and intransitive actions also seems to be relevant. Fan et al. (2010) demonstrated preserved mu suppression in individuals with ASD when observing a hand performing a transitive action; a finding that was similarly reported by Bernier et al. (2007, 2013). This contrasts with Martineau et al. (2008) and Oberman et al. (2005) whose stimuli depicted a hand performing an intransitive action. The capability of children with ASD to imitate or produce intransitive actions, compared to other types of actions (e.g. transitive), is under debate for many reasons. Firstly, intransitive gestures include wide ranges of hand actions (e.g. communicative, pantomime, emblem, arbitrary meaningless gestures). Some of these categories differ from others as they either include semantic meaning (e.g. communicative), cultural inference (emblems), or social communicative content (indicating) - a skill that is highlighted as impaired in the ASD profile.

Hence, the mechanism that underlies a specific intransitive gesture, in which the impaired imitation or production was identified, does not necessarily underlie other gestures that might be processed by a similar mechanism or capacity and therefore may not be equally impaired. This proposal has been supported by numerous lines of evidence. For example, Mundy et al. (1986) argued that despite the fact that *affiliation*, *indicating* and *requesting* are all categorized as non-verbal social communication acts, they vary greatly in many ways. For instance, behaviours involved in requesting and indicating differ from affiliation in terms of attentional demands, which seem to be dyadic (self and others) in the first category and triadic in the latter category (self, others, and objects).

In addition, the coordinating capacity for affiliation is generally higher than for indicating and requesting as it involves an object. The second argument for difficulty in intransitive actions in ASD builds on the fact that children with ASD show a visual preference for objects within social contexts (Klin et al., 2002). This observation was supported by Vivanti and contributors when they employed eye-tracking methods to investigate the pattern of visual attention while 8-15 year-old individuals with ASD were observing and imitating object-directed actions and meaningless actions (Vivanti et al., 2008). Results showed that participants with ASD demonstrated two important findings. Firstly, they spent less time looking at the demonstrator's face. Secondly, only those who spent more time looking at the action region produced accurate imitations of the meaningless gesture stimuli.

The third argument for difficulty in intransitive actions in ASD relates to the context in which the task is presented, which rules out variance in imitative performance. Ingersoll (2008) aimed to examine the validity of this hypothesis by investigating the capacity of children with ASD to imitate transitive actions with toys, after observing an experimenter performing the actions within two different contexts. The first context was an 'elicited condition', wherein the participant was directly instructed to imitate the observed action, and the second context condition was a 'spontaneous condition', wherein the experimenter imitated the participant's play actions and then instructed him to observe him, allowing time for the participant to imitate him back. In contrast to the control participants who imitated equally well in both conditions, the imitation

performance of ASD participants was significantly less overall and significantly worse in the spontaneous condition.

Therefore, independent of the action form and its meaning to the ASD participant, impaired imitation skills in spontaneous settings appear to be accounted for by impaired reciprocal social interaction, as found by McDuffie et al. (2007). The fourth argument for difficulty in intransitive actions in ASD stems from the correlation that has been found between 'gestures' and 'language'. Sigman and Ungerer (1984) found that children with ASD, compared to TD and developmentally-delayed children, manifested specific impairments in vocal and gestural imitation, which correlated with receptive language for all groups of children. Furthermore, vocal imitation correlated with expressive language in TD and ASD children. Given that communication impairment is one of the clinical symptoms of an ASD diagnosis, impairment in 'gestural performance' is therefore more likely to be displayed compared to other categories of imitation.

Within the same scope, the inconsistent findings regarding an imitation deficit in individuals with ASD may be driven by the fact that this developmental deficit is highly featured with repetitive and stereotyped behaviours and 'écholalia', which is an automatic repetition of vocalization (Ganos, Ogrzal, Schnitzler, & Münchau, 2012). While these clinical features appear to ease copying other peoples' speech and behaviour, they also seem to emphasize the notion of underlying neural malfunctions. In theory, in order for mirror neurons to simulate an action, a controlled inhibitory system is required, which is likely to lead to specific uncontrolled repetitive behaviours when this system is impaired.

6.7 Methodological considerations and limitations

In the experiments presented in this thesis, sensorimotor mu rhythm was analysed within two frequency bands: alpha (8 –12 Hz) and low beta (12 –20 Hz), as advocated by Hari (2006), and also in the theta band (5.5-7.5 Hz) for Experiments 3 and 4. The use of mu desynchronization in the lower frequencies (8-13 Hz) as an index of MNs activity has been well established in previous research (e.g., Altschuler et al., 2000; Hari et al., 2000; McFarland et al., 2000; Muthukumaraswamy et al., 2004; Muthukumaraswamy &

Johnson, 2004; Oberman, et al., 2007; Pineda & Hecht, 2009). However, more recent research has indicated that the mu activity could also be detected in the low beta range (12 –20 Hz) desynchronization (Puzzo et al., 2010).

Although mu rhythm desynchronization is viewed as the main determinant of MNs activity, the possibility of interference with classical alpha oscillations has concerned many researchers. However, some scholars (e.g. Hari et al., 1997; Pfurtscheller, 1989) have marginalized these interference effects based on various considerations; for instance, the cortical sources of alpha activity, based on MEG data, have been found to be generated around the parieto occipital sulcus, while the sensorimotor alpha rhythms were found to be maximal along the somatosensory cortex.

In addition, mu rhythm activity is more likely to present an anterior focus with some inter-hemispheric asymmetry, whereas alpha oscillations typically have a more posterior and bilateral distribution. Previous research (e.g. Oberman et al., 2005) has reported that no mediation of the mu band power by posterior alpha activity and no electrode sites, other than those over the sensorimotor cortex, showed a similar pattern of suppression in the mu frequency band to observed and executed actions in ASD (Reymeakers et al., 2009).

Therefore, it is improbable that recording from sensorimotor scalp areas could be confounded by anterior and posterior alpha activity. Furthermore, mu rhythm power is suppressed by motor activity, while alpha is modulated by visual stimulation. Thus, in the current experiments, the first and last 10 seconds of each 80 seconds of EEG recording were removed prior to analysis to eliminate the effects of alpha modulations due to the onset of visual registration. In addition, most importantly, occipital alpha is known to generate in eye-closed phase (Palva & Palva, 2007), which does not seem to correspond with our current stimuli as all participants kept their eyes open.

As our sample comprised young children, electrophysiological recording during observation conditions were arguably affected by body movements; nonetheless, participants were continuously monitored to avoid excessive movements. On the another hand, research has reported that mu rhythm suppression during action

observation is a centrally-controlled phenomenon, thus, movement in the body, if it occurred, would not account for mu rhythm suppression in observation conditions (Muthukumaraswamy et al., 2004; Muthukumaraswamy & Johnson, 2004; Raymeakers et al., 2009). In future research, one EMG channel could be placed on the participant's hand to monitor biological movement during the observation periods and to perhaps exclude other possible sources of movement.

Finally, it is important to note that while the participants' visual view was kept clear of any external stimuli in this and previous studies, there is no definite way of fully ensuring that children were attending and processing the stimuli effectively. For instance, participants at any time might focus their attention away from the moving hand on another part of the screen (or off-screen), which could result in reduced levels of ERD. In future research, the use of simultaneous eye-tracking recording could provide vital information about participant's anticipation of actions, as well as enabling a valid assessment of whether participants were attending to the hand action at all times.

Given the evidence presented above showing that methodological variances play a major role in the direction of findings, some precautions must be acknowledged regarding the generalizability of the effects reported in this thesis. Firstly, the clinical sample in the studies here were solely composed of high-functioning autistic children, and both the control and clinical samples spanned a narrow age range (3-5 years old), meaning that the findings have limited generalizability to lower-functioning individuals with different severities of social ability, or individuals across a wider range of ages (e.g. adults). Secondly, there has been a suggestion, based on a number of fMRI studies (e.g. Caspers et al., 2010; Keysers et al., 2010; Wicker et al., 2003), that a broader MNs network exists which incorporates the premotor cortex, somatosensory and possibly anterior insula. The research conducted within this thesis limited its investigations to the sensorimotor cortex. Thus it is possible that, by focusing on this specialised brain area, we have missed some underlying MNs activity. Thirdly, while our data suggested greater modulation for observing familiar versus unfamiliar actions, this effect was established based on 'communicative hand gestures'. Therefore, extra investigations are required to establish whether the same effects are present for other forms of intransitive actions.

Furthermore, a number of empirical studies (see Kelly et al., 2005; Wright & Sladden, 2003) have shown a gender bias in face recognition. Investigating a gender-bias in social understanding was not of interest here (and in fact, all our adult actors were males) for three vital reasons. Firstly, the philosophy behind the specific use of male actors in the current stimuli was that they would be presented to Saudi participants for whom watching male actors is socially and culturally accepted, which would be different in a different cultural context.

Secondly, although research on cultures in which women wear headscarfs, as in Saudi culture, shows an internal-feature advantage, in face processing over external-features (Megreya, & Bindemann, 2009; Megreya, Memon, & Havard, 2011), this advantage is not clear when related to our clinical population. Thirdly, the current video stimuli included the upper part of the models 'body (i.e. from the waist up), thus recognition was not limited to models 'faces.

A specific caveat to consider with regards to Experiment 4 is that the models from different ethnicities (i.e. Saudis and Europeans) represented clear differences based on low-level factors, such as skin colour, physiognomy and clothing (including head-dresses). It might be interesting, therefore, in future research to examine the outcome of employing two ethnic groups with minimal contrast (e.g. Middle Eastern and Iranian), which might lead to different conclusions. In addition, based on the numerous visual differences between the models from different ethnicities in the current study, it was not possible to determine which stimuli component(s) was responsible for the ethnicity effects we found.

6.8 Key contributions of the current work

In summary, the work presented here responds to the lack of fMRI and EEG evidence of MN activity in early childhood, which was mirrored in requests for further scientific investigations (Bertenthal & Longo, 2007; Lepage & Théoret, 2007). The present data are derived from the first study of MNs in Saudi Arabian ASD and TD child populations. Furthermore, this research and novel paradigm extends existing work by Oberman et al. (2008), in demonstrating a significantly facilitated capacity to 'simulate 'an unfamiliar

person (i.e. in *observation 2*, Experiment 2), when performing a familiar or unfamiliar communicative gesture, when these conditions were primed by a familiar person (i.e. the child ś parent).

This thesis also presents the first experiments to look at the effect of ethnic similarity and age similarity on MN activation in ASD children. Thus, our data bridge the plethora of behavioural literature emphasizing the role of ṕeers ’in social reciprocity, academic learning, language acquisition, and current theories of MNs. Finally, the four experiments allowed us to replicate the effect of familiar actions in ASD populations, and utilising a mixed design, showed how action familiarity is mediated by person familiarity and similarity.

7 References

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