IMITATION OF ATYPICAL BIOLOGICAL MOTION IN AUTISM SPECTRUM DISORDERS

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Contents

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Arte et Labore

II Table Legends

III Figure Legends

IV Abbreviations

Short	Term
AD	Asperger's Disorder
ADHD	Attention Deficit Hyperactivity Disorder
ADOS	Autism Diagnostic Observation Schedule
APA	American Psychiatric Association
DLPFC	Dorsolateral Prefrontal Cortex
DSM-V	Diagnostic and Statistical Manual of Mental Disorders
EEG	Electroencephalography
fMRI	Functional Magnetic Resonance Imaging
GOADI	Goal-Directed Imitation
HFA	High Functioning Autism
IFG	Inferior Frontal Gyrus
IPL	Inferior Parietal Lobule
IPMC	Inferior Premotor Cortex
LFA	Low Functioning Autism
MC	Motor Cortex
MEG	Magnetoencephalography
MEP	Motor Evoked Potentials
MFG	Middle Frontal Gyrus
mPFC	Medial Prefrontal Cortex
PDD	Pervasive Development Disorder
PET	Positron Emission Tomography
PMC	Premotor Cortex
RBS-R	Repetitive Behaviour Scale-Revised
STORM	Social Top-Down Response Modulation
SPL	Superior Parietal Lobule
STS	Superior Temporal Sulcus
TMS	Transcranial Magnetic Stimulation
TPJ	Temporo-Parietal Junction
WAS-II	Wechsler Abbreviated Scale of Intelligence

V Publications and Presentations

Hayes, S. J., Andrew, M., Elliott, D., Bennett, S. J., Gowen E. (2016). Low-fidelity imitation of atypical biological kinematics in autism spectrum disorders is modulated by self-generated selective attention. *Journal of Autism and Developmental Disorders*. *46*(2). 502-513. doi: 10.1007/s10803-015-2588-1.

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VI Thesis Abstract

The aim of the present thesis was to examine imitation of biological motion in adults with autism spectrum disorders. Using a novel behavioural protocol, adults with autism and matched neurotypical control adults imitated models that displayed distinctly different, but biological believable kinematics. In *Chapter Two* it was observed that adults with autism exhibited low-fidelity imitation of *atypical* biological motion. In *Chapter Three* it was observed that when selective-attention instructions were provided, although eye movements recorded during action-observation was similar to controls, imitation of *atypical* biological motion was still impaired. In *Chapter Four* across three experiments it was shown that adults with autism exhibit reasonably high-fidelity imitation of *atypical* biological motion. This was achieved by presenting the to-be-imitated biological models in a fixed presentation structure which is known to facilitate greater integration and consolidation of sensorimotor information. This suggestion was supported by a further study where firstly participants were required to complete a secondary motor task during the inter-trial delay, and when the presentation structure was randomised (similar to *Chapters Two* and *Three*) resulting in low-fidelity imitation of *atypical* biological motion. These findings across the present thesis will be discussed in light of a critical evaluation with respect to current literature on imitation in autism, as well as implications for theoretical accounts of impaired imitation in autism and related sensorimotor control processes. Future considerations and translational research will be discussed, with the intention of offering prospective social rehabilitation protocols in autism.

1 Review of Imitation of Biological Motion Kinematics in Autism Spectrum Disorders

1.1 Aim of the Chapter

The following introductory chapter outlines the rational and aims of this thesis. There will first be an overview of literature pertaining to imitation, which includes reference to the different definitions and types, associated models, and a description of the underlying neural structures. This will be followed by comment on the nature of stimuli imitated and at this point, an overview of literature examining the imitation abilities of individuals with autism spectrum disorders will be provided. Finally, there will be an appraisal of the current theories as well as the sensorimotor processes associated with imitation in autism spectrum disorders, after that the individual aims of the chapters will be provided.

1.2 Imitation

Copying other people shapes evolutionary and cultural development, and in particular the acquisition of novel actions. This process is known as imitation when it involves copying novel bodily features associated with a movement performed by a human model (e.g., using the left foot, and movement dynamics, to kick the ball into the box) (Thorndyke, 1898; Heyes, 2001; Want & Harris, 2002; Whiten, McGuigan, Marshall-Pescini, & Hopper, 2009). Imitation behaviours begin very early in life (Carpenter, Akhtar, & Tomasello, 1998). For instance, 42 hours after birth new-born infants have been shown to mirror (i.e., imitate) simple actions of others such as facial expressions (e.g., lip smacking) and hand gestures (e.g., pointing) (Meltzoff & Moore, 1977; 1983; 1997). To successfully imitate an individual translates visual information observed (i.e., action-observation) from a

human action (i.e., biological motion) into a sensorimotor representation that contains the outcome-goal (i.e., touching the ear) and the form (i.e., limb velocity) to achieve said outcome-goal (Hobson & Lee, 1999). The sensorimotor representation serves as a motor-plan and is mapped onto the motor system for motor-execution, as well as providing the expected consequences of the movement required for motor control (Flanagan & Wing, 1997). During motor-execution the expected sensory consequences are compared to the actual sensory (i.e., visual, proprioceptive) input, such that any resulting inconsistencies can be minimised by online adjustments throughout the movement (Kilner, Friston, & Frith, 2007; Burke, Tobler, Baddeley, & Schultz, 2010). Following motor-execution, the sensorimotor representation is consolidated based on further processing of afferent and efferent sensorimotor information (Wolpert, Ghahramani, & Jordan, 1995; Wolpert, Diedrichsen, & Flanagan, 2011). This motor process is recurrent on a trial-by-trial basis through repeated exposures to the model combined with physical attempts at imitating the model, where error is reduced as the observer adapts their movement to be more like the model (Miller & Dollard, 1941; Sheffield, 1951; Carroll & Bandura, 1982). Higher-order (cognitive/attentional) and lower-level (visuomotor) mechanisms are involved in these processes (Bandura, 1977; Byrne & Russon, 1998), which are embedded within a system linking perception with action (Prinz, 1997; Brass & Heyes, 2005).

1.2.1 Types of Imitation

An individual may be able to produce one type of imitation, yet may have impairments in another type (Hamilton, 2008). It is therefore necessary at this point to distinguish between different types of imitation, as each encompasses varying

functions and accordingly have different underlying processes. The subsections below will briefly discuss different forms of imitation.

1.2.1.1 Spontaneous Imitation

This form of imitation occurs without any premeditation or external stimulus, rather they involve non-specific prompts such as a demonstrator playing with a toy then handing it to the observer, saying "*you can play*". Spontaneous imitation is stereotypically examined using systematic naturalistic observations and parent questionnaires both measuring rates (i.e., how many times the individual imitates the actions of the observer) of imitation.

1.2.1.2 Elicited Imitation

Somewhat the opposite to spontaneous imitation, elicited imitation occurs with explicit instructions to imitate the actions. For example, a demonstrator shows the observer and action and then says "*now you can do it*" or "*your turn*". Elicited imitation is normally examined using accuracy measures, where the observer's actions are compared to that of the demonstrators and provided a score based on these comparisons (e.g., providing a score of two for fully correct imitation, one for partially correct imitation, and zero for incorrect imitation). These to-be-imitated actions are characterised by the presence and/or absence of an object (i.e., actions on objects versus gestures; Vivanti, Nadig, Ozonoff & Rogers, 2008), whether the actions are directed towards a goal (i.e., visual targets) or not (meaningful versus non-meaningful actions; Wild, Poliakoff, Jerrison, & Gowen, 2012), and whether these actions are simple or complex (i.e., single versus sequential actions; Rogers, Bennetto, McEvoy & Pennington, 1996).

1.2.1.3 Emulation

Emulation occurs as a function of imitating the goal of an action (i.e., action end-point) but not the form (i.e., velocity of the limb) to achieve the goal (Tomasello, Kruger, & Ratner, 1993). For example, when children imitated the contralateral hand gestures (e.g., touching the ear and/or dots on a table) of an experimenter sat facing them, they reached for the correct object yet preferred to use the ipsilateral limb. This preference was diminished when then hand movements were directed at space rather than physical objects (Bekkering, Wöhlschlager, & Gattis, 2000; Gleissner, Bekkering, & Meltzoff, 2000; Wöhlschlager, Gattis, & Bekkering, 2003). These hand errors suggested that perception-action coupling is directed by goals inferred by the imitation, such as the physical object at which an action is directed (i.e., a particular ear) and the agent of that action (i.e., a particular limb) (Bekkering et al., 2000).

1.2.1.4 Automatic Imitation

Frequently referred to as 'mimicry', automatic imitation occurs when an observer spontaneously and unintentionally matches the action (e.g., raising the index finger; yawn) of a model (Heyes, 2011). For example, when required to execute finger movements (tapping; lifting of the index finger) in response to a video stimulus of compatible (i.e., same) or incompatible (i.e., different) finger movements, responses were initiated faster when the stimulus was compatible (i.e., when a finger lifting response was made in the presence of an finger lifting stimulus) rather than incompatible (i.e., when a finger lifting response was made in the presence of a finger tapping stimulus) (Brass, Bekkering, & Prinz, 2001). The finding of shorter movement responses when the response effector is compatible

with the observed effector, compared to incompatible, is often referred to as levels of automatic imitation (Heyes, Bird, Johnson, & Haggard, 2005). Previous work has shown similar levels of automatic imitation in individuals with and without autism spectrum disorders (e.g., Press, Richardson, & Bird, 2010; Sowden, Koehne, Catmur, Dziobek & Bird, 2016) which will be discussed in more detail later.

1.2.1.5 True Imitation

Often referred to as 'hierarchical imitation' (Byrne & Russon, 1998) or simply 'imitation' (Whiten & Ham, 1992), here an observer imitates the goal of an action, as well as the form to achieve the goal. For instance, an observer will imitate the upper limb kinematics (i.e., velocity) displayed by the demonstrator in order to achieve the goal of drawing a shape on a digital graphics tablet (Williams, Casey, Braadbaart, Culmer, & Mon-Williams, 2014). It is known that this particular type of imitation places a large emphasis on both the goal and the form to achieve the goal (Vivanti & Hamilton, 2014).

1.2.1.6 Imitation in the Current Thesis

These different forms of imitation contain different levels of intricacy and require attention to different aspects of an observed action. The key distinction between 'automatic' and 'true' imitation is that the former is an involuntary process that leads to an observer nonconsciously copying certain movement properties displayed by a model. This requires said movements (e.g., finger tapping) to be already stored within the observer's sensorimotor repertoire (Heyes, 2011). In contrast, true imitation is a voluntary/explicit process where on a trial-by-trial basis, a movement pattern is copied that is not already stored in the observer's

sensorimotor repertoire (Vogt et al., 2007). In the current thesis, true imitation will be examined as it is central to development in the broader context of motor behaviour in autism spectrum disorders. For example, true imitation underpins the acquisition of everyday sensorimotor skills such as writing with a pen, or tying shoes laces, or riding a bicycle.

1.2.2 Imitation for Social Cognition

In addition to being an influential facilitator in the acquisition of novel sensorimotor behaviours, imitation also serves a social function, as studies have found imitative abilities to be correlated to socio-cognitive skills (Meltzoff & Decety, 2003) such as language (Bates et al., 1988), play (Fiese, 1990), joint attention (Carpenter, Nagall, & Tomasello, 1998), and measures of Theory of Mind (Perra, Williams, Whiten, Fraser, Benzie, & Perrett, 2008).When individuals are unaware that they are being imitated, they report increased levels of closeness (Kühn, Müller, van Baaren, Wietzker, Dijksterhuis, & Brass, 2010), altruistic behaviour (van Baaren, Holland, Steenaert, & van Knippenberg, 2003), trust (Bailenson & Yee, 2005), and a positive social attitude (Lakin & Chartrand, 2003). Through investigation of the 'chameleon effect' (i.e., nonconscious imitation of postures, facial expressions, gestures and behaviours of another during social interaction), Chartrand and Bargh (1999) reported that when working with another on a task (i.e., description of a photograph) participants unintentionally matched their own body positions (e.g., arms crossed) to that of the partner. Furthermore, increased levels of affection were reported by participants whose actions (e.g., posture) were unintentionally imitated by another during social interaction. From these findings, it was suggested the chameleon effect is underpinned by a mechanism

that links perception with action (Prinz, 1997). In addition to these findings, it has also been shown that having a pro-social attitude can positively influence on imitation. For instance, when required to arrange five words such that they formed a grammatically correct sentence containing pro-social (e.g., friend; team) or anti-social (e.g., obstinate; distrust) words, individuals who arranged pro-social sentences demonstrated significantly higher levels of automatic imitation compared to individuals who arranged anti-social words (Leighton, Bird, Orsini, & Heyes, 2010; Cook & Bird, 2011). These findings are a result of imitation being bi-directionally associated with positive social interaction, and is a key component of building positive social relationships (Lakin & Chartrand, 2003).

1.2.3 Neural Models of Imitation

Although the current thesis is written from a behavioural-psychological perspective, it is important to highlight the neurophysiological underpinnings of imitation, as certain influential accounts of associated with imitation in autism spectrum disorders are underpinned by differences in how the visual motor processes operate and are controlled during imitation. For example, neurophysiological studies using Functional Magnetic Resonance Imaging (fMRI) and/or Transcranial Magnetic Stimulation (TMS), have shown similar responses within the human brain during action-observation and motor-execution (e.g., Gallese, Fadiga, Fogassi, & Rizzolatti, 1996; Rizzolatti, Fadiga, Fogassi, & Gallese, 1996). Extending upon original work (Di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992) that showed neurons are active in the inferior premotor cortex (IPMC) of the macaque monkey during action-observation of goal-directed actions (e.g., grasping, holding and tearing), Fadiga, Fogassi, Pavesi, and Rizzolatti (1995) stimulated human motor cortex (MC)

using single pulse TMS during: (1) observation of an experimenter grasping an object; (2) observation of same object; (3) observation of an experimenter tracing geometrical figures; and (4) detection of a dimming light. Results showed that motor evoked potentials (MEP; motor evoked potential recorded from peripheral muscles using electromyography (EEG)) were significantly greater during action-observation compared to non-action-observation. In addition, MEPs during action-observation positively correlated with motor-execution of the same actions, indicating a common coding between observed and motor actions (Fadiga et al., 1995). Later work by Buccino et al. (2004) imaged (fMRI) novice participants while imitating guitar chords during four events (action-observation; motor-planning; motor-execution; inter-trial processing). Results indicated a neural circuit that is active throughout all phases of imitation consisting of inferior parietal lobule (IPL) and the posterior part of the inferior frontal gyrus (IFG), plus the adjacent premotor cortex (PMC), which becomes active during action-observation. Then during motor-planning, the middle frontal gyrus (MFG; area 46), dorsal pre-motor cortex, superior parietal lobule and rostral mesial areas additionally become active (Buccino et al., 2004). Activation throughout the specific phases of the imitation process highlights the neural circuit that translates an observed action into a motor action (see also Iacoboni, Woods, Brass, Bekkering, Mazziotta, & Rizzolatti, 1999; Iacoboni et al., 2001; Vogt et al., 2007; Di Dio, Di Cesare, Higuchi, Roberts, Vogt, & Rizzolatti, 2013). This circuit is referred to as the human ‘mirror neuron system’ (for a review see Rizzolatti & Craighero, 2004; Iacoboni, 2005) with the core components being the superior temporal sulcus (STS), inferior frontal gyrus and inferior parietal lobule, which underpins perception to action in imitation (Prinz, 1997; Heyes, 2001; Hamilton, 2015).

1.2.4 Models of Imitation

1.2.4.1 Dual-Route Model

The model was first put forward by Rumiati and Tessari (2002) and predicted that two pathways are operating during imitation. There is a semantic route which is utilised for known, meaningful goal-directed actions, and a direct route which is used for novel actions that do not have a goal (i.e., non-meaningful action). During imitation both systems are operating but depending on the specific context, are modulated in order to achieve the imitation goal. For example, if the action is known (e.g., reaching for a pen), an observer uses the semantic route that relies upon pre-existing sensorimotor representations that are selected and scaled to meet the task demands. Alternatively, if the action is novel and non-meaningful, such as gestures that can be described only in terms of postures (e.g., a hand moving across the forehead), an observer engages a direct route to imitation, which recruits visuomotor mapping processes to code biological motion (i.e., human action) in order to represent the novel movement kinematics (e.g., limb velocity). Follow up work by Tessari and Rumiati (2004) provided support for the predictions of the dual-route model (see also Rumiati et al., 2005; Rumiati & Tessari, 2007; Carmo & Rumiati, 2009; Rumiati, Carmo, & Corradi-Dell'Acqua, 2009). For example, across three experiments participants imitated meaningful (pantomimes of object use) and non-meaningful (similar to meaningful yet they were not recognised) actions where the stimulus was presented in a blocked or random structure. Overall, and consistent with their previous work (Rumiati & Tessari, 2002) imitation was more accurate when imitating meaningful compared to non-meaningful actions. Furthermore, when the stimulus presentation was blocked, participants used the semantic route for

known actions and direct route for novel actions, with greater imitation accuracy exhibited for the former. When stimulus presentation was randomised, there was no difference in imitation accuracy. The authors concluded that imitation was processed though the direct route when the stimulus was unfamiliar within a random presentation, and through the semantic route when the stimulus was more familiar in a blocked presentation structure. Given that participants were constrained for time with reduced cognitive resources, they used the most convenient route for imitation. The current thesis examined true imitation, which involves imitating an action that is not stored within the observer's sensorimotor repertoire and thus would be more in-line with processing though a direct route.

1.2.4.2 Goal-Directed Imitation

It has also been suggested that during imitation (e.g., upper-limb pointing movement) an observer cognitively decomposes an observed movement by representing a hierarchy of goals and sub-goals. This goal hierarchy follows the functionality of the action where the end-point (i.e., final goal of the action) of the movement is given more importance than the form to achieve the goal (i.e., limb selection; limb velocity). This suggestion is now more commonly referred to as to as the goal-directed theory of imitation (GOADI; Bekkering et al., 2000). Recent behavioural (Hayes, Hodges, Scott, Horn, & Williams, 2007; Horn, Williams, Scott, & Hodges, 2007; Hayes, Dutoy, Elliott, Gowen, & Bennett, 2016) and neuroimaging (Grafton & Hamilton, 2007; Hamilton & Grafton, 2007) work examining goal-directed imitation exemplifies this suggested pattern. For instance, in a study examining goal-directed and goal-less imitation, participants observed and subsequently imitated a series of aiming movements that varied in overall speed

(fast; slow), across two conditions where visual targets (dots on table) were either present (i.e., goal-directed) or removed (i.e., goal-less). In-line with the model of goal-directed imitation, it was suggested the end-point (i.e., final goal of the action) of the movement was prioritised, leading to the end-goal of the movement being imitated rather than the form. In contrast, during goal-less imitation, where constraining end-goal (i.e., targets removed) information is removed, visual attention is directed to the form to achieve the goal leading to more accurate imitation of the kinematics. Results showed that when visual targets were removed (i.e., goal-less condition), participants modulated their movement kinematics (i.e., peak velocity was significantly higher in the 'fast' trials) such that they became closer to the model (Wild, Poliakoff, Jerrison, & Gowen, 2010), compared to the goal-directed imitation (i.e., when visual targets were present). Wild and colleagues suggested that different processes mediate imitation of goal-directed and goal-less movements, where accurate imitation of goal-less imitation (i.e., when visual targets were removed) is facilitated by direct visuomotor mapping (e.g., Rumiati & Tessari, 2002), and end-goal imitation occurs through a sematic route (Rumiati et al., 2009) or is positioned higher in the goal hierarchy and prioritised based on goal achievement (e.g., GOADI; Wöhlschlager et al., 2003).

1.2.4.3 Associative Sequence Learning

This was first put forward by Cecilia Heyes (Heyes & Ray, 2000; Heyes, 2001; 2005) and suggested that the development of the mirror neuron system (Rizzolatti & Craighero, 2004) is based to some extent on sensorimotor experience. In other words, these links express mirror neuron system activation allowing action-observation to prime, or develop, motor-execution. Here then, activation of the

motor representation is paired with a corresponding perceptual representation (i.e., observation of the finger) and through experience (i.e., trial-by-trial basis) a bidirectional associative link is created where activation of one representation primes the other. Action-observation of a novel movement (i.e., biological motion) during imitation involves two processes. First, there are horizontal links that use sensory (i.e., visual) representations of actions in a sequence (i.e., sensory 1 activates sensory 2) which enable an observer to acquire what the action looks like. Second, there are vertical links that operate before the novel movement is observed and results in a sensory representation of the action components (i.e., sensory 1) becoming associated with a motor representation of the same component.

Support for the suggestion that Associative Sequence Learning is a general visuomotor mechanism that modulates the development of mirror neuron activity comes from training studies (Heyes et al., 2005; Bird, Brindley, Leighton, & Heyes, 2007; Gillmeister, Catmur, Liepelt, Brass, & Heyes, 2008; Catmur, Walsh, & Heyes, 2009; Cook, Press, Dickinson, & Heyes, 2010; Catmur, Mars, Rushworth, & Heyes, 2011; Cooper, Cook, Dickinson, & Heyes, 2013; Cavello, Heyes, Becchio, Bird, & Catmur, 2014). For example, individuals performed a counter-mirror protocol that required compatible or incompatible sensorimotor training (Catmur, Walsh, & Heyes, 2007). During compatible training, participants executed index-finger movements, whilst simultaneously observing index-finger movements. During incompatible training, participants executed index-finger movements, whilst simultaneously observing little-finger movements. After incompatible training, TMS-induced MEPs recorded from the little finger abductor muscle were greater during observation of index-finger movement compared to a little-finger movement. These findings demonstrate the sensorimotor system was reconfigured during

correlated sensorimotor training, and thus indicate imitation is associated with a general mechanism involving lower-level visuomotor processes that represent biological motion, as opposed to a specialised mechanism that mediate the translation of visual information into a motor action (Meltzoff & Moore, 1997).

1.3 Biological Motion

In the context of a human movement, biological motion refers to the visual-sensory information contained in a movement that describes a particular action (Kozlowski & Cutting, 1977). For instance, an individual can be judged to be walking, jumping, or throwing an object based upon how the arms and legs move in relation each other. Thus, during imitation, and more specifically true imitation, attention is directed towards the biological motion kinematics (e.g., timing and magnitude of velocity of the limb) of the observed demonstrator/model in order to gain a reference of the to-be-imitated action. Importantly, there is evidence that the human mirror neuron system, which underpins imitation, processes biological (i.e., human) and non-biological (i.e., robotic) motion differently (Grossman et al., 2000; Grèzes, Fonlupt, Bertenthal, Delon-Martin, Segebarth, & Decety, 2001). Using Positron Emission Tomography (PET), participants were scanned during observation of a manual grasping action performed by either a human model or a robot model. Results indicated a significant neural response within the premotor cortex, which is responsible for action encoding (Gallese et al., 1996), during observation of a human model only (Tai, Scherfler, Brooks, Sawamoto, & Castiello, 2004). Furthermore, magnetoencephalography (MEG) data recorded during observation of vertical hand movements showed activation was consistent with sensorimotor learning when the

hand movements displayed kinematics that had biological (i.e., human) compared to non-biological (i.e., robotic) velocity profile (Press, Cook, Blakemore, & Kilner, 2011; for a larger review of biological motion and the action observation network, see Press, 2011). These above findings demonstrate that that the human mirror neuron system that underpins imitation is tuned to processes biological motion differently to non-biological (Grossman et al., 2000; Grèzes et al., 2001; Tai et al., 2004; Keysers & Gazzola, 2006).

Biological motion models used in perception studies (e.g., Johansson, 1973) and imitation studies (Horn, Williams, Scott and Hodges, 2005) are typically generated using point light displays. These models were originally created by attaching small light bulbs to the joints of a demonstrator, and actions were recorded in a dark room (Johansson, 1973). With the advancements in technology, reflective markers are attached and recorded using three-dimensional motion capture systems (i.e., Vicon Nexus). In the imitation task across the present programme of work, a single white-dot will be presented (similar to point light displays) as the model. Given that the mirror neuron system is tuned to biological motion (Grossman et al., 2000; Grèzes et al., 2001; Tai et al., 2004) the models will display a biological (i.e., human) velocity profile. These velocity curves are characteristically bell-shaped and are typical (**Figure 1.2b**) of a natural reach-to-grasp action (e.g., reaching for a pen; cup of coffee). Here then, the individual moves the limb slowly in the initial phase of the movement, accelerates through the middle and slows down to accurately grasp the pen (Flash & Hogan, 1985; Elliott, Hansen, Grierson, Lyons, Bennett, & Hayes, 2010). In comparison, the non-biological model will present a constant velocity profile. This velocity model is computer generated and had no deviations in the perpendicular axis (**Figure 1.2b**).

1.4 Autism Spectrum Disorder

Autism spectrum disorder (henceforth referred to as autism across for the remainder of this thesis) is a neurodevelopmental developmental disorder primarily classified by atypicalities in social interaction, verbal and non-verbal communication, as well as a restricted repertoire of interest and activities (American Psychiatric Association (APA), 1994, 2000, 2013). Autism was first identified by Leo Kanner (1943) and Hans Asperger (1944). In 1943, Kanner published a seminal paper entitled 'autistic disturbance of affective contact' where he described eleven cases of children who were unable to establish social relationships with others. He described the autistic child as remote and if they spoke, they used rote-learned phrases or words; and did not just show simple repetitive movements (e.g., flapping of hands) but more elaborate rituals. In each of these cases, individual differences in various characters were identified suggesting that the disorder comprised a syndrome (Kanner, 1943). A year later Asperger published a seminal paper entitled 'autistic psychopathy in childhood'. He described case studies where children showed deficiencies in social interaction as well as behavioural differences including: impairments in nonverbal communication; peculiarities in verbal communication; social adaptation and special interests (Asperger, 1944).

1.4.1 Characteristics of Autism

Building upon the work of Kanner (1943) and Asperger (1944) there are now clearer core characteristics that define autism (American Psychiatric Association, 2013) which are discussed in the subsections below:

1.4.1.1 Social Development

The foremost characteristic that differentiates autism from other developmental disorders is the development of social skills. This unusual social development becomes apparent during childhood, as during social interaction, a toddler with autism typically make less eye contact (i.e., looking at the person face; Senju & Johnson, 2009) and less turn-taking (i.e., waiting for the other person to finish talking before they speak), as well not possessing the ability to use simple movements to express themselves (i.e., pointing at objects). During the ages between 5 and 8 years old, children with autism are less likely to show poor social understanding (Sigman, Dijamco, Gratier, & Rozga, 2004). They are unable to respond to emotions (i.e., responding to someone with a sad face) as well as non-verbal communication (i.e., making gestures). Moreover, though this may be due to difficulty in processing emotions, adults with autism consistently perform worse on tasks involving face and emotional recognition (Bird & Cook, 2013). Though not a core characteristic, the lack of social development often leads to difficulties in forming and maintaining relationships, resulting in increased reports of loneliness in adults with autism (Burgess & Gutstein, 2007).

1.4.1.2 Communication

As early as 12 months old, children with autism exhibit delays in the development of communicative skills. These delays include the onset of babbling (i.e., articulate sounds that are not recognisable words), unusual gestures (e.g., hand gestures), diminished responsiveness (i.e., responding to a stimulus) together with vocal patterns that are not in sync with others. These difficulties continue into 24 and 36 months, as children with autism have also shown frequent and less diverse

babbling, consonants (i.e., basic speech sound) and word combinations, as well as their gestures (i.e., facial expressions; finger pointing) are less integrated with words (Noens, Berckelaer-Onnes, Verpoorten, & van Duijn, 2006). Furthermore, both individuals with autism have shown deficits in language development. For example, although children (aged ~ 8 years) and adolescents (aged ~ 15 years) with autism showed similar performance as matched neurotypicals on basic language tasks (e.g., spelling and vocabulary), both autism groups performed significantly worse than matched typically developing children and adolescents on complex language tasks involving comprehension and figurative language (Williams, Goldstein, & Minshew, 2006).

1.4.1.3 Repetitive Behaviours

The final core characteristic of autism is the display of restricted and repetitive behaviours. In accordance with the Repetitive Behaviour Scale-Revised (RBS-R; Lam & Aman, 2007), these behaviours include those that are stereotyped (constant movements such as hand flapping), compulsive (i.e., actions that have rigid rules intended to reduce anxiety), ritualistic (i.e., unvarying pattern of daily activities e.g., morning routine), and restricted (activities which are limited in variety e.g., playing with the same toy). As well as a stubbornness to change (e.g., refusing to be interrupted; moving furniture in a room) and behaviours that cause injury to themselves (e.g., eye-poking, hand-biting and head-banging). Although none of these repetitive and/or self-injuring behaviours are specific to autism, an elevated pattern of occurrence and severity characterise the disorder (Bodfish, Symons, Parker, & Lewis, 2000).

1.4.2 Diagnosis of Autism

Per the Diagnostic and Statistical Manual of Mental Disorders (DSM-V; American Psychiatric Association, 2013), a diagnosis of autism requires at least six items from three main behavioural symptom categories. Firstly, social interaction difficulties include solidarity, making less frequent eye movements compared to typically developing children, as well as unusual and/or unfitting behaviours (e.g., aloofness; remoteness). Secondly, communication difficulties include a significant delay in language attainment and a failure to comprehend feelings and empathy towards others. Finally, restricted interests and repetitive behaviours include an inflexibility adherence to rituals and stereotyped behaviours (e.g., flapping of hands). Moreover, they require a delay and/or impairments in at least one or more categories including social interaction, language, and symbolic or imaginative play. These behavioural symptoms attributed to the categorisation of autism ordinarily present themselves within early childhood (i.e., between 18 and 24 months old) and persist throughout the lifespan of the individual, though their presentation typically varies throughout development. Due to these wide range of variant symptoms, autism sits under the general category for pervasive development disorder (PDD), which also encompasses those who are identified as having low-functioning autism (LFA) with an intelligence quotient (IQ) score typically below 70 (as well as other types of biological causes), and those who are identified as having high-functioning autism (HFA) with normal (> 70) to high (> 85) IQ scores (Ghaziuddin & Mountain-Kimchi, 2004). In addition, Asperger's disorder (AD) is distinguished from autism spectrum disorder by the absence of significant general delays in language attainment.

1.4.3 Theory of Mind in Autism

One of the earliest and most prominent theory to account for these atypicalities in communication and social interaction in autism, and at one time considered to be the primary reason of the disorder, is the Theory of Mind (Baron-Cohen, Leslie, & Frith, 1985; Baron-Cohen, 1995) or 'mindblindness' (Frith, 2001) hypothesis. Theory of Mind is an ability to make inferences concerning the goals, desires, beliefs, and mental states of another individual (Premack & Woodruff, 1978). For example, if an observer witnesses a person reaching for a biscuit from a tin container labelled 'biscuits', it could be assumed that the person would like a biscuit and believe that there are biscuits in the container, even if the observer is already aware the container is empty. With regards to imitation, if an individual does not possess Theory of Mind and the ability represent the mental states of others, then they would have difficulties to form and manage representation of self and other (i.e., self-other mapping). One of the first studies on Theory of Mind in autism was reported by Baron-Cohen et al. (1985) and studied the "Sally-Anne" false-belief test. In this test, children are presented with a story in which Sally has a basket, and Ann has a box. The story proceeds where Sally puts her marble in the basket and leaves the room. While Sally is away, Ann takes the marble from her basket and put it in the box. Sally returns to look for the marble. The child is then asked "*where will Sally look for her marble?*" If the child points to the previous location of the marble (i.e., basket), they pass by appreciating the doll's now false belief. If, however, they point to the current location they fail by not taking into account Sally's belief. It was reported that 85 % of unimpaired children and 86 % of children with Down's Syndrome answered the false-belief question correctly, compared to only 20 % of

children with autism who answered the false-belief question correctly (Baron-Cohen et al., 1985; see also Baron-Cohen, Leslie, & Frith, 1986).

1.4.4 Prevalence Rates of Autism

In comparison with earlier reports of autism (e.g., Eisenberg & Kanner, 1955), advances in understanding of symptoms, together with diagnostic criteria, have influenced prevalence rates of autism. Prevalence rates monitor the number of known cases reported within one period of time or a span of time. Initially, autism was seen as a relatively uncommon disorder, with prevalence rates of 4 per 10,000 individuals (Rutter, 1978). This estimation has increased noticeably over the years with prevalence rates reported to be 157 per 10,000 in the United Kingdom (Baron-Cohen et al., 2009). This year-on-year increase can be clearly demonstrated by the work of Gurney and colleagues (Gurney, Fritz, Ness, Sievers, Newschaffer, & Shapiro, 2003) who reported an increase of autism prevalence among children aged 6-11 years in the state of Minnesota (USA) from 251 in 1991-1992 to 4094 from 2001-2002 (an increase of 1531 %). As can be seen in **Figure 1.1**, in 2010 the prevalence rate of autism within the United Kingdom was reported to be 39 boys, and 8 girls, per 10,000 children (**Figure 1.1a**; Taylor, Hershel, & MacLaughlin, 2013). Globally, prevalence rates were reported to be 5.8 and 3.0 per 10,000 in boys and girls respectively (**Figure 1.1b**; Baxter, Brugha, Erskine, Scheurer, Vos, & Scott, 2014). Importantly, when interpreting these rates caution must be taken as differences in methodology and diagnostic tests have produced varying results. The increase in awareness of autism, as well as improved diagnostic techniques and reporting practices, has contributed towards an increase in the prevalence rates of autism (Hansen, Schendel, & Parner, 2015).

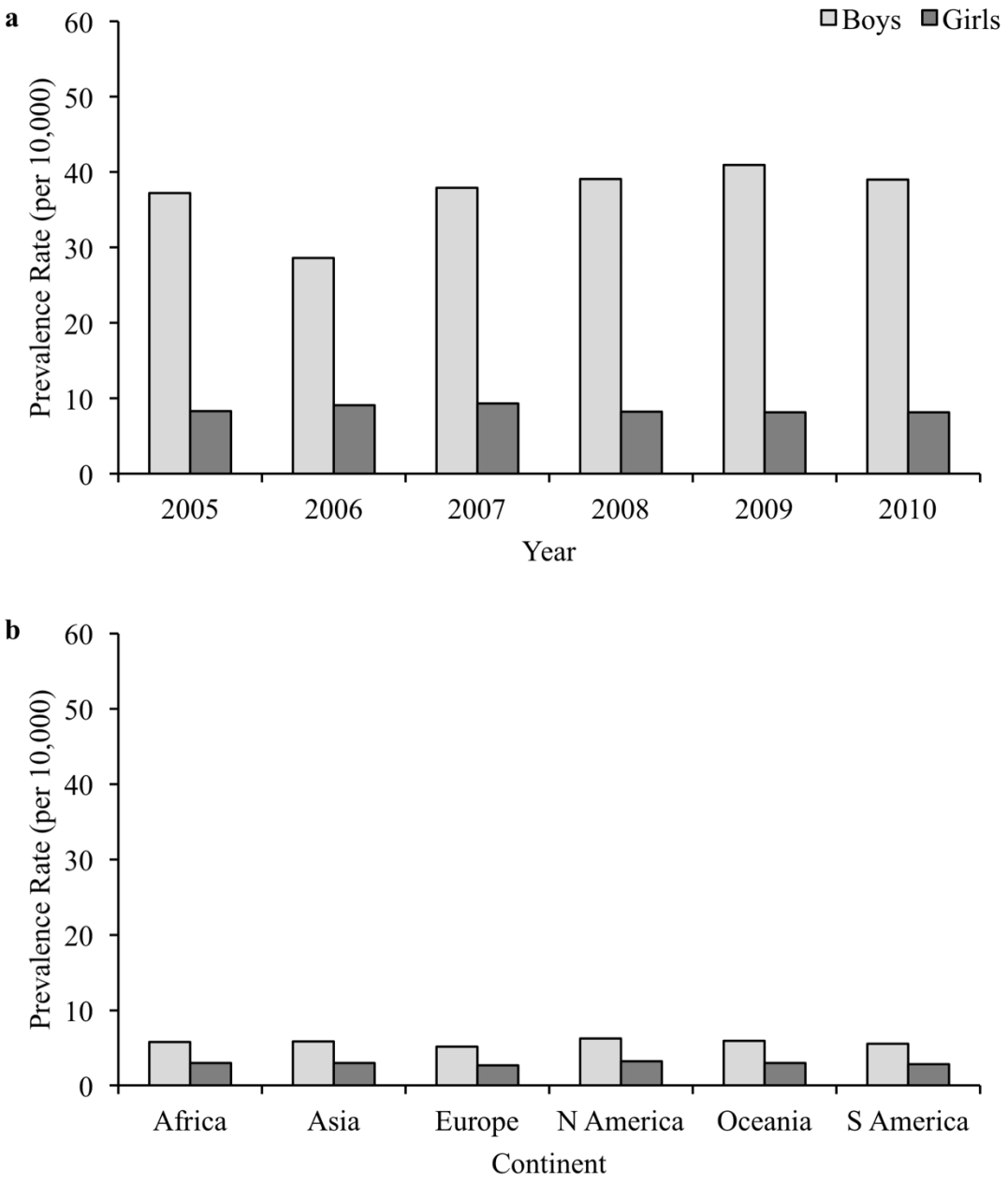

Figure 1.1 (a) Mean prevalence rates of autism in the United Kingdom from 2005-2010 presented as a function of gender and year (adapted from Taylor et al., 2013).

1.5 Imitation in Autism

Given the complexity of characteristics associated with autism and the obvious benefits of imitation for social interaction, imitation abilities in individuals with autism have received considerable examination (Rogers & Pennington, 1991; Smith & Bryson, 1994; Rogers, 1999; Rogers, Hepburn, Stackhouse, & Wehner, 2003; Rogers & Williams, 2006; Hamilton, 2013; Vivanti & Hamilton, 2014; Vivanti & Rogers, 2014). For example, Edwards (2014) performed a meta-analysis of fifty-three studies to examine whether individuals with autism show significant imitation deficits and whether they are specific to autism. A random-effects model showed individuals with autism showed deficits with autism with an average of 0.81 SDs below neurotypical individuals. Furthermore, this observed deficit was specific to autism, as moderator analysis indicated that average Autism Diagnostic Observation Schedule (ADOS; a semi-structured instrument for diagnosing and assessing autism) scores positively correlated with autism imitation abilities. Finally, the manner in which imitation was operationalised affected the size of the imitation differences between individuals with and without autism. However, the study setting, novelty of actions, format of imitation tasks, or the number of actions to imitate were not found to significantly affect the sizes of the imitation differences between individuals with and without autism.

Much of the work examining imitation in individuals with autism has mainly focused on spontaneous and elicited imitation. One of the first studies (DeMyer et al., 1972) that explicitly examined spontaneous imitation abilities in children with autism involved non-specific prompts (e.g., a demonstrator plays with a toy, then hands it to the observer, saying "*you can play*"). It was reported that compared to

neurotypical children, children with autism were better at imitating in contexts that required motor-object imitation (copying the object) than in contexts that required body movements to imitated (e.g., a standing jump). Another method to examine spontaneous imitation in autism is through automatic imitation (i.e., mimicry). Several studies using automatic imitation have found that compared to matched neurotypicals, individuals with autism show intact automatic imitation responses (Leighton, Bird, Charman, & Heyes, 2008; Sowden et al., 2016). For instance, when adults with autism and neurotypical adults were required to perform hand actions following observation of either a human or robotic hand actions, adults without autism showed an automatic imitation effect, which was more profound after observing human compared to robotic actions ('animacy' bias). Importantly, adults with autism also showed a similar automatic imitation effect and greater animacy bias than neurotypical adults (Bird, Leighton, Press, & Heyes, 2007).

Other research examining imitation abilities in autism examined elicited imitation. These to-be-imitated actions are typically characterised by whether they are directed towards a goal (meaningful/familiar) or not (non-meaningful/novel). Studies examining elicited imitation have regularly reported difficulties in imitation of non-meaningful actions in individuals with autism (Stone, Ousley, & Littleford, 1997; Bernier, Dawson, Webb, & Murias, 2007; Vanvuchelen, Roeyers, & De Weerdt, 2007; Rogers, Young, Cook, Giolzetti, & Ozonoff, 2010). For example, Rogers et al. (1996) examined imitation and pantomime in adolescents with autism. Seventeen adolescents with autism were matched (chronological age and verbal IQ) with fifteen typically developing adolescents and completed three tasks. First, in the hand imitation task participants were required to imitate single or sequential hand actions that were either meaningful (familiar e.g., put arms over head, clasp together

and shake) or non-meaningful (novel e.g., extend arm and hand straight out in front of body, with fingers fanned out, and thumb pointed to ceiling). Results indicated that aside from single meaningful actions, neurotypical adolescents demonstrated greater imitation performance (number of perfect scores) compared to autistic adolescents. Second, in a facial imitation task participants were required to imitate single or sequential (three movements consisting of movements from single movements) facial actions that were meaningful (e.g., happy, sad, frightened) or non-meaningful (e.g., tongue protrusion with mouth open). Results demonstrated that typically developing adolescents imitated non-meaningful sequential facial actions with higher accuracy than adolescents with autism. Finally, in a pantomime task, participants were required to pantomime single and sequential meaningful actions with the use of common (e.g., toothbrush) objects. Furthermore, they completed two control tasks where participants imitated using objects in an appropriate way and where they demonstrated the real use of the object without a model. Results showed that typically developing adolescents had greater accuracy when imitating single and sequential actions that did not require the use of an object (Rogers et al., 1996).

In another study that concentrated solely on imitation of non-meaningful actions in individuals with autism, Hobson and Lee (1999) examined whether children with autism had specific problems imitating the style (e.g., harsh movement) in which the action is performed. Sixteen adolescents with autism were matched (chronological age and verbal IQ) with sixteen typically developing adolescents and completed four non-meaningful imitation tasks: (1) Pipe-rack and stick (strumming and stick across the ridges of the pipe-rack three times); (2) frog and roller brow-wiping (laying a synthetic frog on the palm of the hand and wiping

forehead three times); (3) stamp and ink-pad (holding a handle of a stamp, pressing the stamp on the ink-pad, and then transferring it onto a sheet of paper); (4) rolling policeman (pushing a spring mechanism on a toy policeman that stood on wheels such that it moved forward). In each of the tasks, the action was to be performed in either a harsh (i.e., abrupt) or gentle (i.e., elegant) style (except the rolling policeman task which required the policeman to be depressed with either the wrist or index and middle finger). Results indicated that fewer children with autism imitated the style of the action. That is, they were able to perform the same goal-directed action (e.g., move a stick across a wooden pipe-rack), but failed to imitate the style (e.g., gentle or harsh movement to strum the stick across the wooden pipe-rack) with which the action was performed (Hobson & Lee, 1999; see also Hobson & Hobson, 2008).

A number of other more recent studies have confirmed that individuals with autism show a priority towards imitating the goal of the action, over imitating the form to achieving the goal (Cossu et al., 2012; Salowitz et al., 2013). In a study by Hamilton, Brindley and Frith (2007) examining goal-directed imitation and action understanding, autistic children were matched (verbal mental age) with typically developing children and completed four action-representation tasks. For example, when testing Bekkering's goal-directed imitation task (Bekkering et al., 2000), participants sat across from an experimenter and copied their hand movements to a target (dot on a table) using mirror imitation. Results indicated that children with autism displayed similar goal-directed imitation strategies as typically developing children. Here then, when required to imitate the contralateral trials (congruent target; incongruent limb), they chose to imitate the end-point of the action (i.e., target on a table) over the limb selected (i.e., congruent limb) to perform the

movement. Thus, the data provides evidence that both typical and autistic children understand and imitate the goal of an action.

Up until recent years, imitation in autism was quantified using descriptive measures (e.g., Rogers et al., 1996; Hobson & Lee, 1999), where a quantitative form score is used to measure imitation performance (e.g., providing a score of two for fully correct imitation). Though this is a suitable approach, more recently kinematic analysis has been used to examine imitation of biological motion (e.g., Wild et al., 2010; Williams et al., 2014). Kinematic measures have the advantage of being able to quantify exactly what properties of the movement are, or are not, imitated in autism, such as biological motion kinematics. During examination of goal and goal-less imitation (Wild et al., 2012), adults with autism and matched (age, sex and IQ) controls observed a human model perform a series of upper-limb pointing movements that were differentiated by speed (fast, slow). In addition, context was manipulated such that the model aimed to visual-targets (dot on table) or an end-space. According to the goal-directed theory of imitation (Bekkering et al., 2000), when the visual-targets are removed (i.e., end-space) an imitator is likely to focus attention towards imitating the model's movement (i.e., kinematics) as opposed to simply reaching for the goal (i.e., visual-target). Consistent with this theory, the results from Wild et al. (2012) showed only control participants imitated the different movement speeds when targets were removed. In comparison, participants with autism failed to modulate the movement speed in either condition. Moreover, using similar apparatus to that in the current thesis, it has also been shown that autistic individuals have some trouble imitating spatial properties of an action. In a study by Stewart, McIntosh, and Williams (2013), participants were required to imitate actions using a stylus on a graphics tablet performed by a human or non-

human (only the end-point of the movement seen) model. Imitation accuracy was calculated by comparing against a 'ghost control' (the end-point of the movement was replaced by a dot on the screen) condition. Compared to matched (chronological age) controls who exhibited no differences in imitation following observation of human or non-human model, adolescents with autism exhibited differences in action duration and path length in both observation conditions.

To summarise, studies examining imitation in individuals with autism make it apparent that there are differences in imitation abilities compared to matched controls. Early work that used non-specific prompts indicated that individuals with autism imitate less frequently (DeMyer et al., 1972), yet imitation is intact in tasks that measure rapid and nonconscious matching of others actions (Bird et al., 2007; Leighton et al., 2008; Sowden et al., 2016). Studies that explicitly instructed to imitate indicated that individuals with autism can successfully imitate the goal (i.e., meaningful) of an observed action (Hamilton et al., 2007; Jiménez, Lorda, & Méndez, 2014) yet they have difficulties imitating the form of the action (i.e., non-meaningful) to achieve the goal (Rogers et al., 1996; Hobson & Lee, 1999). Examining imitation of kinematic features of the movement isolates the contribution of lower-level processes and has provided novel contributions to further understanding towards specific imitation deficits in those with autism, such as movement speed (Wild et al., 2012) and path length (Stewart et al., 2013).

1.6 Processing Accounts of Imitation in Autism

Over the year's researchers have attempted to identify the underlying processes underpinning impaired imitation in autism which will be discussed below:

1.6.1 Mirror Neuron System; Self-Other Mapping Processing

One of the earliest presuppositions towards a greater understanding of the underlying mechanisms of impaired imitation in individuals with autism is reflected by a restricted capacity to form and manage representation of self and other (i.e., self-other mapping). Through the discovery of mirror neurons (Di Pelligrino et al., 1992) and a system that underpins self-other mapping (Iacoboni et al., 1999) it was subsequently suggested that early developmental failures within the mirror neuron system in autism were responsible for the reported discrepancies in self-other mapping, accompanying other aspects of social-cognition such as Theory of Mind (Williams, Whiten, Suddendorf, & Perrett, 2001). This suggestion is now commonly referred to as the 'mirror neuron system' hypothesis and is still one of the most prominent accounts for imitation difficulties in autism. This hypothesis has been supported by further reviews of the literature (Williams, Whiten, & Singh, 2004; Iacoboni & Dapretto, 2006), as well as behavioural and neuroimaging studies showing different activity within the mirror neuron system during action-observation and imitation in autism (Oberman, Hubbard, McCleery, Altschuler, Ramachandran, & Pineda, 2005; Théoret, Halligan, Kobayashi, Fregni, Tager-Flusberg, & Pascual-Leone, 2005; Williams, Waiter, Gilchrist, Perrett, Murray, & Whiten, 2006). For instance, Dapretto and colleagues (2006) examined the mirror neuron system in children with autism and typically developing children (matched for chorological age and full-scale IQ) when imitating five facial expressions (anger, fear, neutral, happiness, and sadness) during one of two sessions: (1) they were required to imitate the observed facial expression, or; (2) simply sit and observe the facial expression. Behavioural data indicated that both autistic and typically developing children successfully imitated different facial expressions, which was accompanied by similar

eye movement patterns (fixations on the demonstrator's eyes during action-observation and motor-execution). Neuroimaging data showed that during imitation of facial expressions, typically developing children exhibited similar neural activity to that previously illustrated in neurotypical adults (e.g., Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003). The data also showed that autistic children showed inactivity within a core component (IFG) of the mirror neuron system during imitation of the facial expressions which positively correlated with autism severity (Dapretto et al., 2006).

Williams and colleagues also used fMRI paradigm to examine the neural mechanisms during imitation in autism (Williams et al., 2006). Fifteen autistic adolescents and matched (age and IQ) control adolescents were scanned while either observing, executing or imitating index finger movements following three stimuli: (1) animation of index or middle finger being lifted; (2) photograph with black cross marking the index or middle finger; (3) plain background with black cross on left or right side of screen. Though imitation of finger sequences was similar between group, fMRI data showed robust differences between the autism and control adolescents in neural activity. The autism group did not show activation within the somatosensory cortex during non-imitative action. In addition, contrary to the control group, during imitation the autism group did not show activation of the right posterior middle temporal gyrus at the temporo-parietal junction (TPJ). They did, however, show such activation during action-observation. These findings (Dapretto et al., 2006; Williams et al., 2006) of altered activity within the mirror system result in problems with integrating visual analysis, motor action, proprioception and emotional processing during the self-other mapping in imitation. Finally, this suggestion of altered self-other mapping has also been reported in recent behavioural

work (Stewart et al., 2013). When autistic adolescents and matched (age) neurotypicals imitated motor actions on a graphics tablet performed by a human or non-human (only the end-point of the movement seen) model, autistic adolescents showed significantly less imitation accuracy in action duration and path length in both conditions. By using a model that displayed only the movement end-point (i.e., non-human model), and controlling for general factors associated with memory, spatial reasoning, motor control, attention, and social context, it was suggested that deficits in imitation were linked to impaired neural processes underpinning self-other mapping, and that these led to difficulties with representing the observed movement kinematics and mapping this to the motor system for imitation.

1.6.2 Social Top-Down Model

Through the increased evidence associated with intact spontaneous and elicited imitation of actions in autism (Hamilton et al., 2007; Bird et al., 2007; Leighton et al., 2008; Press et al., 2010; Wild et al., 2012; Sowden et al. 2016), combined with differences in the regions of previously reported abnormal brain activity (Dapretto et al., 2006; Grèzes, Wicker, Berthoz, & De Gelder, 2009) but typical responses when viewing goal-directed actions (Dinstein, Thomas, Humphreys, Minshew, Behrmann, & Heeger, 2010; Marsh & Hamilton, 2011) it has been suggested that the lower-level visuomotor processes in imitation are unimpaired in autism (Hamilton, 2008; 2009; 2013; 2015). Therefore, it has been advocated that problematic imitation abilities in individuals with autism may be due to failure of top-down control mechanisms (Southgate & Hamilton, 2008). These mechanisms involve the medial prefrontal cortex (mPFC) and TPJ (Hamilton, 2013, 2015), which are suggested to control the lower-level visuomotor processes by

selecting and controlling the imitative (or non-imitative) actions to represent. More specifically, the social top-down response modulation (STORM) model (Wang & Hamilton, 2012) suggests top-down control is based on the evaluation of a social context and situation, and that impairments of these top-down control mechanisms may result in atypicalities in imitation and mirror neuron system activity in autism. Initial research exploring social context and imitation in autism is consistent with this model and indicates that imitation in typically developing children and adults can be modulated by social cues such as eye contact (Wang, Newport, & Hamilton, 2011) and pro-social sentences (Cook & Bird, 2011). However, this is not the case for individuals with autism. For example, nineteen adults with autism and matched adults without autism were primed with either a pro-social (e.g., friend) or non-social (e.g., secluded) attitudes prior to mimicry of finger movements. Priming was achieved using a four or five word grammatically correct sentences (see Leighton et al., 2008). Consistent with their earlier work (Cook & Bird, 2011), Cook and Bird (2012) observed that neurotypical adults exhibited higher levels of automatic imitation following pro-social compared to non-social priming. In contrast, automatic imitation levels were not modulated following pro-social or non-social priming in autistic adults. These findings indicate that the human mirror system is operating during automatic imitation, yet is not regulated appropriately during social contexts (Cook & Bird, 2012; Wang & Hamilton, 2012).

1.6.3 Visual Attention Processes

It has recently been suggested that differences in visual attention may attribute towards impaired imitation of biological motion in autism (Gowen, 2012). In this case, it is not implied that there is general attention away from the imitation

task itself but more of a bias away from the kinematic features (e.g., velocity) of the observed action. A study by Vivanti et al. (2008) was one of the first to measure visual attention patterns in children with autism and matched (age and IQ) typically developing controls during imitation of meaningful and non-meaningful actions. Participants observed and imitated actions with objects such as striking a xylophone (i.e., meaningful) and gestures such as bending the arm at the elbow (i.e., non-meaningful), while eye movements were recorded. Behavioural data (sum of precision scores similar to Rogers et al., 1996; Hobson & Lee, 1999) revealed that children with autism were less accurate than controls when imitating meaningful actions with objects and non-meaningful actions. Eye movement data revealed that although autistic children had similar movement patterns as controls, they spent half as much time observing the models face (Vivanti et al., 2008). Wild et al. (2012) examined kinematic data and eye movements during the motor-execution phase of imitation. The results showed that the lack of modulation of movement speed exhibited by adults with autism was associated with less time tracking (i.e., smooth pursuit) the hand, and more time shifting gaze (i.e., saccade) and fixating on the action end-point. From these results, it was suggested that shifts in gaze, and thus attention away from the model was a compensatory mechanism, which consequently influenced the amount sensorimotor information processed from the hand trajectory resulting in low-fidelity imitation of movement kinematics (Wild et al., 2012). One way to influence visual attention is by proving explicit instructions. This has previously been shown to increase levels of contagious yawning (Senju, Kikuchi, Akechi, Hasegawa, Tojo, & Osanai, 2009), which is a response that facilitates joint attention during interpersonal contexts, and is underpinned by similar lower-level sensorimotor processes as those engaged during imitation (Senju, 2013). Though

children with autism were originally found to execute fewer yawns whilst observing a model than a control group (Senju, Maeda, Kikuchi, Hasegawa, Tojo, & Osanai, 2007), this behaviour was reversed following explicit instructions that directed overt visual attention to the eye region of the model (Senju et al., 2009). This is a point that will be discussed further in *Chapter Three* of this thesis.

1.6.4 Processing Biological Kinematics

It is well accepted that perception of biological motion plays an important role in imitation (Press, 2011) and it has been suggested that possible difficulties in biological motion perception may underlie the documented impairments in imitation in individuals with autism (Freitag et al., 2008). There is evidence that individuals with autism have difficulties perceiving biological motion (Nackaerts, Wagemans, Helsen, Swinnen, Wenderoth, & Alaerts, 2012). For example, Blake and colleagues (Blake, Turner, Smoski, Pozdol, & Stone, 2003) required children with autism and matched (chronological age) children without autism to complete two visual tasks. One involved grouping small line elements into a global figure, and the other involved perceiving human motion portrayed by point light displays. Results showed that although children with autism perform similarly to children without autism on the figure task, they performed with less accuracy when perceiving biological motion task. Moreover, in a more recent study, autistic and matched (age, gender and IQ) neurotypical participants were required to observe and recognise biological motion and emotions from point light displays. Results indicated that neurotypicals were significantly more accurate at recognising biological motion compared to the autistic participants. The reduction in accuracy of emotional recognition was associated with altered eye movements (Nackaerts et al., 2012).

Though studies have shown difficulties in perceiving biological motion in autism, recent data show that individuals with autism exhibit intact abilities to perceive biological motion (Moore, Hobson, & Lee, 1997; Wild et al., 2012; Cook, Blakemore, & Press, 2013; Cusack, Williams & Neri, 2015). Adults with autism and matched (age, gender, and IQ) neurotypical adults were required to determine the direction of movement of point light displays (walking person, translating rectangle or translating unfamiliar shape) embedded within noise dots that moved similarly. Results verified no differences in perceptual thresholds between autistic and neurotypical adults across all three conditions, with close to duplicate results (Saygin, Blakemore, & Press, 2013). Moreover, Cusack et al. (2015) examined action perception in adolescents with autism and controls (matched for age, IQ and Social Responsiveness Scale) using point light displays. During action-perception, participants were required to do the following: (1) biological motion detection (differentiate between biological and non-biological motion); (2) action discrimination (discrimination between robotic and natural motion); (3) limb fragments (discrimination of one form of action from another); (4) agent synchrony (integration of libs into full-body agents); (5) attention (discrimination of two agents that are temporally synchronous or not); (6) animate motion (attention to biological motion signals). Results indicated that across all six experiments autistic adolescents performed to the same level as neurotypicals signifying intact biological motion perception. To summarise, there is a sufficient body of evidence that suggests autistic individuals can process biological motion (Wild et al., 2012; Cook et al., 2013; Saygin et al., 2013; Cusack et al., 2015). Important to the present thesis is that many of the studies reporting difficulties in perceiving biological motion are linked to processing emotion (e.g., Nackaerts et al., 2012). To factor for this and ensure that

all participants with autism could successfully perceive differences in biological motion, an action-perception task was included and will be discussed further in *Chapter Three*.

1.7 Sensorimotor Control Processes in Autism

Though not classified as a core characteristic, individuals with autism often display sensorimotor impairments, which range from motor apraxia (Ming, Brimacombe & Wager, 2007) to differences in balance (Weimer, Schatz, Lincoln, Ballantyne & Trauner, 2001). Consequently, it is important to address the sensorimotor control processes in autism as this provides the potential to isolate areas that may influence any observed differences in imitation in autism. A previous review took a computational approach to sensorimotor control processes in autism (Gowen & Hamilton, 2013). In the review of the sensorimotor control process below, similar phases (1-4) have been sub-sectioned based on the imitation learning work of Buccino et al. (2004):

1. During action observation attention is directed towards the biological motion kinematics of the observed model such that sensorimotor information is attended to, and processed leading to the generation of a sensorimotor representation containing the goal (i.e., touch the ear) and the form (i.e., limb; limb velocity) to achieve the goal.
2. The representation forms the motor plan for execution by containing sensorimotor information about the motor components required to achieve the

goal, which are then mapped onto the motor system along with the expected sensory consequences (e.g., vision; proprioception).

3. During motor-execution the efferent sensory signals (from the motor plan), and afferent sensory (vision; proprioception) feedback are compared to the expected sensory consequences (efference copy; inverse model). Any discrepancy will be minimised by through online motor control processes and feedback from vision.
4. During the inter-trial delay between two trials (i.e., offline), the efferent, afferent, and sensorimotor feedback are continued to be processed and integrated such that the sensorimotor representation is refined. The updated sensorimotor representation can be used for the upcoming trial (i.e., trial *n*+1).

1.7.1 Action-Observation

Precise imitation of biological motion requires accurate sensory inputs, as this provides information about the task (i.e., kinematics). During observation of a novel human action (e.g., guitar chords played by a guitarist), visual information from eye movements is encoded into a representation which encompasses the goal(s) (e.g., touching the target on the table) and the form (e.g., velocity of the limb) to achieve the goal(s), and acts as an internal model (Wolpert, Miall, & Kawato, 1998). Current data from eye movement studies in autism point towards abnormalities in basic processes associated with saccadic (Schmitt, Cook, Sweeney, & Mosconi, 2014) and smooth pursuit (Takarae, Minshew, Luna, Krisky, & Sweeney, 2004) eye movements. Furthermore, previous studies have shown that individuals with autism showed variability in patterns towards social visual engagement (Rice, Moriuchi, Jones, & Klin, 2012), the actions of others (Barbaro & Dissanayake, 2013), as well as visual attention to non-social information (Sasson, Elison, Turner-Brown, Dichter,

& Bodfish, 2011; Elison, Sasson, Turner-Brown, Dichter, & Bodfish, 2012). For example, in comparison with matched typically developing children which orientated attention towards the eyes of the demonstrator during observation, children with autism exhibited altered patters of eye movements away from the demonstrator's eyes (Klin, Lin, Gorrindo, Ramsay, & Jones, 2009). Differences in eye movements have also been observed during imitation in autism, with dissimilarities in visual attention to observed stimuli (Vivanti et al, 2008, 2011; Wild et al., 2012; Vivanti & Dissanayake, 2014). This led to the suggestion that impaired imitation in autism may be a consequence of anomalous visual attention towards the model. This hypothesis has been previously discussed (1.6.3) and will be addressed further in *Chapter Three*. These studies demonstrate a clear difference in visual sensory inputs in autism, which may impact the calculation of expected sensory consequences and thus the ability to plan and adjust executed movements.

1.7.2 Motor-Planning

In order to successfully plan the observed action, an individual must process the desired goal (i.e., touching the ear) into a sequence of motor commands. One of the simplest and mostly used methods to examine planning is reaction times, which represents the time taken to formulate the motor plan. Compared to neurotypicals, autistic individuals frequently exhibit longer reaction times (Rinehart, Bellgrove, Tonge, Brereton, Howells-Rankin, & Bradshaw, 2006; Glazebrook, Elliott, & Szatma, 2008; Nazarali, Glazebrook & Elliott, 2009; Dowd, McGinley, Taffe, & Rinehart, 2012). For example, twelve autistic children that were matched (age and midrange IQ) with twelve children diagnosed with Asperger's syndrome, as well as eleven typically developing children, completed a serial-choice reaction task

(Rinehart, Bradshaw, Brereton, & Tonge, 2001). Results indicated that individuals with autism and Asperger's disorder had an intact ability to execute the movement, with similar movement times to typically developing children. However, both groups had significantly slower reaction times during motor preparation than typically developing children (Rinehart et al., 2001). An alternate approach to examining planning is by using reach-to-grasp task, which again reveals autistic individuals create motor-plans differently (Fabbri-Destro, Cattaneo, Boria, & Rizzolatti, 2009; Forti, Valli, Perego, Nobile, Crippa, & Molteni, 2011; Gonzalez, Glazebrook, Studenka, & Lyons, 2013). In an early study by Hughes (1996), thirty-six autistic children and twenty-eight matched (age and verbal IQ) typically developing children completed a rod placing task. Performance was quantified as the final hand posture (i.e., whether the participants finished in a comfortable (thumb up) or uncomfortable (thumb down) position. It was observed that compared to typically developing children, autistic children had significantly higher uncomfortable final postures, indicating autistic children did not consider the end-point of the movement. In addition, children with autism also demonstrate a diminished ability to complete standard tests (i.e., Tower of Hanoi/London tasks) of planning (Ozonoff, Pennington, & Rogers; 1991; Hughes, Russell, & Robbins, 1994), indicating that on the whole, individuals with autism have difficulty planning their movements (Fournier, Hass, Naik, Lodha, & Cauraugh, 2010).

1.7.3 Motor-Execution

During execution, particularly in the early stages of imitation, errors in the movement execution may arise due to planning difficulties and/or noise within the motor system (Gowen & Hamilton, 2013). To reduce these movement errors a

forward model is used which uses the outgoing signals (i.e., efference copy) and creates a prediction of the expected sensory input. This expected sensory input is compared to the actual sensory input (i.e., afference) and any resulting discrepancy between the expected and actual sensory consequences are minimised by online adjustments (Miall & Wolpert, 1996; Wolpert & Kawato, 1998; Wolpert & Flanagan, 2001). Atypicalities in motor-execution in autism have been repeatedly observed, and involve accurate execution of a movement (i.e., upper-limb pointing) but increased variability of movement kinematics (Gowen & Miall, 2005; Glazebrook, Gonzalez, Hansen, & Elliott, 2009; Papadopoulos, McGinley, Tonge, Bradshaw, Saunders, & Rinehart, 2012; Mosconi, Mohanty, Greene, Cook, Vaillancourt, Sweeney, 2015; Cook et al., 2013). For example, Mari and colleagues (2003) found that average (IQ 80-89) and high (IQ > 90) ability autistic children completed a reach to grasp task more rapidly than control participants. Also, low-ability (IQ < 80) autistic participants reached with longer movement duration, lower deceleration and peak velocity, and delayed maximum grip apertures for grasping (Mari, Castiello, Marks, Marraffa, & Prior, 2003). A later follow-up study investigated how adults with autism execute and control goal-directed movements (Glazebrook, Elliott, & Lyons, 2006). Nine adults with autism were matched (chronological age) with nine neurotypical adults and completed pointing movements. All participants placed their index finger on a ‘home’ position and then moved as quickly and as accurately as possible to targets that were manipulated by length (short; long) and target size (small; large). Kinematic analysis indicated that although movement accuracy was similar to matched controls, adults with autism exhibited greater temporal and spatial variability over the initial phase of the movement, along with lower peak velocities. The authors suggested that the varying

results were due to a problem in the timing of muscular forces. In sum, the evidence above provides a strong indication that increased motor noise and timing deficits may lead to increased variability in temporal and spatial aspects of execution during imitation in autism.

1.7.4 Sensorimotor Integration and Consolidation

Sensorimotor adaptation is essential for successfully imitating novel actions. Recently, the ability of individuals with autism to successfully form and refine sensorimotor representations has come under close scrutiny. This area has been examined at length by Mostofsky and colleagues through adaptations in motor-execution in response to a change within the environment (Mostofsky, Dubey, Jerath, Jansiewicz, Goldberg, & Denckla, 2006; Fuentes, Mostofsky, & Bastian, 2011). For instance, hand displacement was measured during a ball-catching task in eight boys with high-functioning autism and eights boys without autism (matched for age). During baseline, when a light ball was used followed by a heavier ball, greater initial hand displacement was evident which gradually reduced to a steady-state displacement. When returning to a light ball, less hand displacement was evident compared to the baseline light ball. Notably, these adaptation effects were observed regardless of disorder (Mostofsky, Bunoski, Morton, Goldberg, & Bastian, 2004). In a later study, during two tasks (ball catching; moving a novel tool) where the environment had been changed (prism goggles; perturbed forces), results showed that children with autism developed and transformed a representation akin to children without autism. This was verified through after effects (updating of the representation through processing motor reafference on a trial-by-trial basis), which resulted in adaptation of motor-execution (Gidley Larson, Bastian, Donchin,

Shadmehr, & Mostofsky 2008). In both studies, it was concluded that successful adaptation was underpinned by normal cerebellar function. fMRI findings support cerebellar contribution to motor adaption and motor sequence learning (Mier & Peteresen, 2002) and was previously suggested to be altered in individuals with autism (Courchesne, Townsend, & Saitoh, 1994).

Though the above findings demonstrate successful motor adaptation in those with autism, there is also increasing evidence that they have difficulty integrating sensorimotor information (Mostofsky & Ewen, 2011; Whyatt & Craig, 2013a, 2013b; Marko, Crocetti, Hulst, Donchin, Shadmehr, & Mostofsky, 2015). For example, children with autism and matched children with attention deficit hyperactivity disorder (ADHD), as well as typically developing children completed the Movement Assessment Battery for Children. Comparable with previous studies also employing this battery of tests (Whyatt & Craig, 2012), children with autism had difficulties in the ball-catching task. Notably, these difficulties were not only dissimilar to typically developing controls but also children with ADHD (Ament et al., 2015). These findings, as well as studies showing a bias towards reliance on visual over proprioceptive feedback when learning a novel movement (Haswell, Izawa, Dowell, Mostofsky, & Shadmehr, 2009; Izawa, Pekny, Marko, Haswell, Shadmehr, & Mostofsky, 2012), points towards imitation difficulties in autism being specific to perception-action coupling, and associated sensorimotor integration.

1.8 Summary of Research and Current Thesis

The aim of the above introductory sections was to provide an overview of imitation of biological motion kinematics in individuals with autism, and some of the motor

control processes that influence imitation. After doing so it is clear that children and adults with autism can successfully imitate meaningful (i.e., known) actions (Sowden et al., 2016) and goal-directed actions (Hamilton et al., 2007), but have difficulties imitating the form (i.e., producing a gentle or harsh sound) to achieve the goal (Rogers et al., 1996; Hobson & Lee, 1999). To date, studies examining imitation of kinematic measures that isolate lower-level processing is limited. Work thus far has indicated that individuals with autism have difficulties imitating movement speed (Wild et al., 2012) and amplitude (Stewart et al., 2013). Based on these findings several attempts have been made to account for impaired imitation in autism, such as differences in the lower-level visuomotor processes that map the visual information onto the motor system (Williams et al., 2001, 2004) or top-down control of these lower-level visuomotor processes associated with social interaction (Wang & Hamilton, 2012) and/or differences in visual attention (Vivanti et al., 2008). Furthermore, observed problems in sensorimotor processes associated with motor-planning (Glazebrook et al., 2008), motor-execution (Glazebrook et al., 2006) and sensorimotor consolidation (Ament et al., 2015) could also cause complications in imitation of biological motion kinematics in autism.

1.8.1 Imitation Task

At present, previous work has provided understanding of specific imitation deficits in autism by isolating the contribution of lower-level processes (Wild et al., 2012; Stewart et al., 2013). This was achieved by manipulating the speed or amplitude of the modelled movement. In terms of biological kinematics, the aforementioned context requires an imitator to scale an existing motor pattern (upper-limb movement) to meet new task demands (e.g., faster movement), but does

not isolate whether the deficit is attributable to imitating specific lower-level properties (e.g., velocity) of biological motion kinematics. In the current programme of work, a different approach will be employed where imitation of biological motion in adults with autism was examined by using a novel protocol that required participants to imitate movements that had distinctly different, but still biologically plausible, movement kinematics (Hayes, Roberts, Elliott, & Bennett, 2014; Hayes et al., 2016; Andrew, Bennett, Elliott, & Hayes, 2016). Using a stylus on a digital graphics tablet, adults with autism and matched neurotypical controls will observe and subsequently imitate a model that displays a single horizontal trajectory that originates from the left-hand side of the screen and ends at a right-hand side of the screen (**Figure 1.2a**). The experimental models display a movement that has exactly the same spatial and temporal outcomes as a control model, but with a velocity profile of either *typical*, *atypical*, or *constant* (*Chapter Two* only) kinematics (**Figure 1.2b**). The *atypical* model ensures an observer must configure the sensorimotor system to represent the novel movement kinematics, as opposed to the *typical* model that can be achieved by rescaling an existing representation of a typical upper-limb aiming movement (Hayes, Timmis, & Bennett, 2009). It is well accepted that biological motion is coded via lower-level processes that is influenced by top-down attentional (end-state goals) and social (human form; eye contact) factors (Kilner et al., 2007; Stanley, Gowen & Miall, 2007). Therefore, to control for these potential modulatory affects the model reflects movement in an 'unmodulated' social context (Cook & Bird, 2011). To control for factors associated with social interaction (Spengler, Bird, & Brass, 2010; Wang & Hamilton, 2012), the model of the to-be-imitated movement will be displayed as a non-human agent model (a single white-dot) which has limited social context (**Figure 1.2a**). To regulate for factors related to

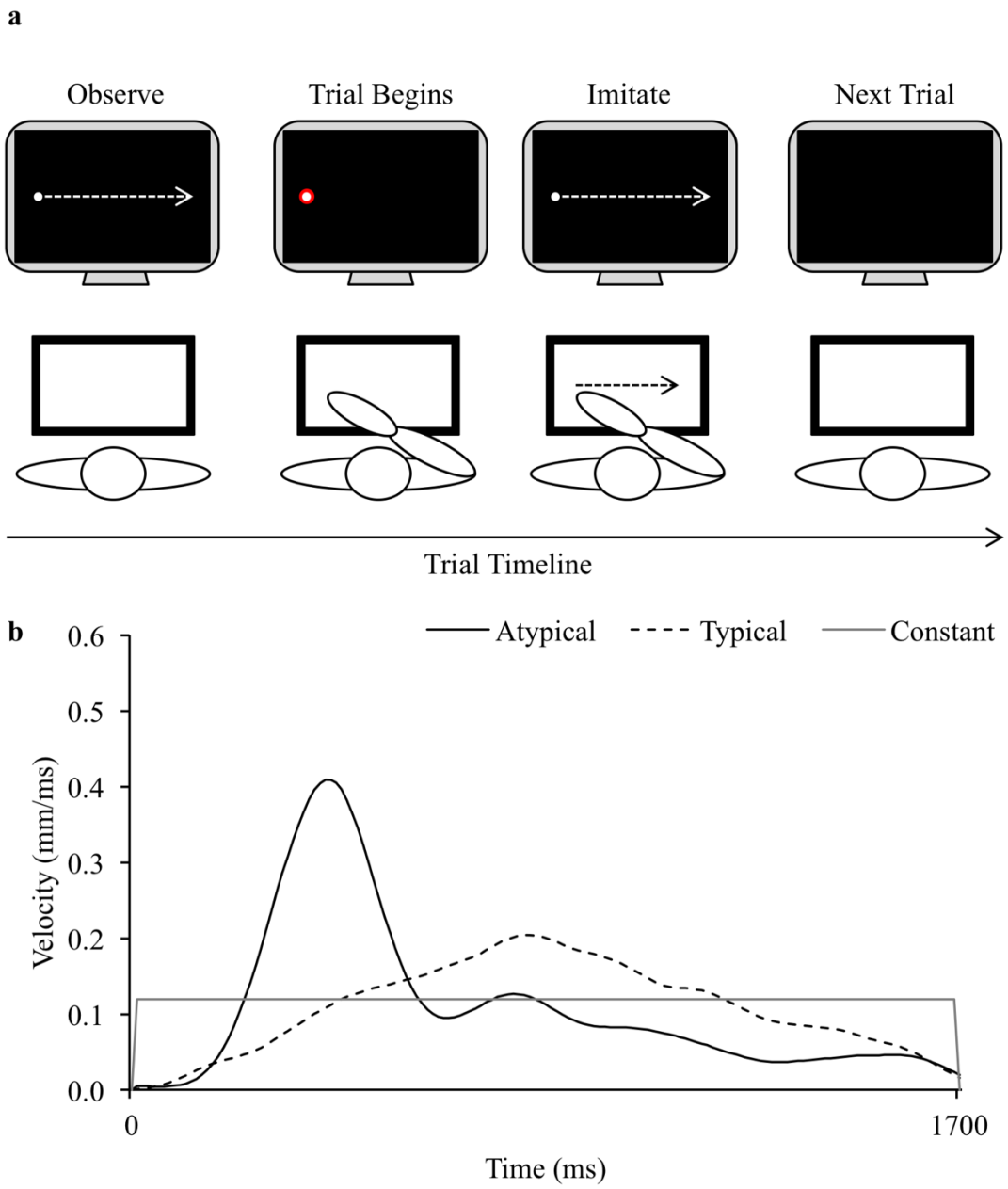

Figure 1.2 (a) A schematic representation of the laboratory/experimental set-up for the imitation task. The black outlined rectangle represents a graphics tablet. The white circle displayed on the CRT monitor represents the model. The single-segment movement is depicted by the arrow. **(b)** *Typical* (dashed-black trace), *atypical* (solid-black trace) and *constant* (solid-dark-grey trace) velocity models presented as a function of time.

target goal-directed features of the task (Wild et al., 2010), in *Chapter Two* target goals will only be displayed in half the trials to encourage attention towards the trajectory of the model. Furthermore, by displaying target goals in half of the trials will allow a direct comparison between target and no-target conditions, examining visual attention towards the goal directed features of the movement, and whether imitation of biological motion in autism is influenced by goals consistent with previous studies (Wild et al., 2012). Given the lack of top-down effects of target goals in *Chapter Two*, in *Chapters Three* and *Four*, the target goals will be removed in all trials.

1.9 Aims of Thesis

The overall aim of the present thesis is to examine imitation of biological motion kinematics in adults with autism. The main question is whether adults with autism can adapt imitation and represent biological motion kinematics following specific manipulations to the learning context (e.g., attentional instructions; practice structure). In the following subsections, specific hypotheses will be presented in relation to each individual chapter.

1.9.1 Chapter Two

The aim is to examine whether adults with autism have difficulty imitating *atypical* biological kinematics. Imitation, and imitation adaption (i.e., performance change from the early-phase to late-phase of imitation), of biological motion kinematics will be examined using a novel behavioural protocol that requires adults with autism and neurotypical controls to observe a model that displays distinctly

different but biologically plausible kinematics (Hayes et al., 2014; 2016). Based on the findings, two possible accounts for processes associated with imitation of biological motion in autism will be presented. Firstly, it is possible that visual attention away from the kinematic features of the model could lead to differences in sensorimotor information extracted (Vivanti et al., 2008; Wild et al., 2012; Gowen, 2012; Vivanti & Dissanayake, 2014). Secondly, it is possible that individuals with autism might have difficulty integrating sensorimotor information across trials that do not promote an opportunity for consolidation (Nebel et al., 2015; Sharer, Mostofsky, Pascual-Leone, & Oberman, 2015).

1.9.2 Chapter Three

In the second experimental chapter, the first of the two possible accounts for processes associated with imitation of biological motion will be investigated by examining overt visual attention when imitating biological motion kinematics. Using the same general protocol as *Chapter Two*, adults with and without autism will be provided with selective-attention instructions (Bach, Peatfield, & Tipper, 2007; Hayes et al., 2014) prior to imitation that to direct visual attention towards the kinematics of the to-be-imitated model(s). Furthermore, in order to examine whether imitation deficits in autism are related to processes associated with visual attention, eye movements will be recorded during the action-observation phase.

1.9.3 Chapter Four

In this chapter, the second of the two possible accounts for processes associated with imitation of biological motion will be investigated across three independent studies. In the first study, the same protocol as previous chapters will be

used, but now with the to-be-imitated model will be presented in a fixed (i.e., blocked) rather than random trial order. The aim is to determine if adults with autism can learn to adapt and represent biological motion following specific manipulations to trial order in which imitation is occurring. The aim of the second study will be to investigate the possible underlying processes of the above adaptation. To examine where these processes are occurring, participants will complete a similar protocol with a fixed trial order but now a secondary motor task (drawing circles on the tablet) will be completed in the inter-trial delay (i.e., in between motor-execution of trial *n* and action-observation on trial *n*+1). The aim of the third study will be to determine if functional imitation of biological motion kinematics in individuals in autism is associated with the opportunity to consolidate and integrate sensorimotor information. Here, participants will complete an identical protocol as *Chapter Two,* yet rather than being naïve to the protocol, they will be familiar (i.e., they will have completed the previous experiments) with the aim to replicate the kinematics data from this chapter using a random trial order.

1.9.4 Chapter Five

The aim of the final chapter is to provide a clear and concise summary of the findings of this entire programme of work, and to critically analyse these findings with reference to current literature in the area of imitation of in autism. Implications will then be drawn for both recent theoretical accounts of impaired imitation in autism, as well as sensorimotor control processes in autism. Lastly, implications for future translational research on imitation in autism will be discussed, with the intention of offering prospective social rehabilitation protocols in autism.

2 Low-Fidelity Imitation of Biological Kinematics in Autism is Modulated by Self-Generated Selective Attention.

2.1 Abstract

The aim of the present study was to further examine imitation of biological motion in individuals with and without autism. A novel protocol was employed that required participants to imitate movements that had distinctly different, but still biologically plausible, movement kinematics. To reduce the impact that higher-order processes have on imitation a non-human agent model was used to control social attention, and removed end-state target goals in half of the trials to minimise goal-directed attention. Findings showed that only neurotypical adults imitated *atypical* biological kinematics. Adults with autism did, however, become significantly more accurate at imitating movement time. This confirmed they engaged in the task, and that sensorimotor adaptation was self-regulated. The attentional bias to movement time suggests the attenuation in imitating kinematics might be a compensatory strategy due to deficits in lower-level visuomotor processes associated with self-other mapping, or selective attention modulated the processes that represent biological kinematics.

2.2 Introduction

Imitation is a powerful mechanism for learning new sensorimotor behaviours (e.g., throwing a Frisbee) as well as for developing socio-cognitive skills (e.g., rapport; Chartrand & Bargh, 1999) and affiliation (Lakin & Chartrand, 2003). One way humans acquire these behaviours is by copying a novel movement displayed by another person. This process is defined as true imitation because an observer is required to copy the properties of human movement (biological motion) after observing a model, rather than being able to merely reproduce the movement using an already learned movement pattern based on previous experience (Byrne & Russon, 1998). In the context of human movement, biological motion is the visual-sensory information contained in a movement that describes a particular action (Johansson, 1973; Kozlowski & Cutting, 1977). For example, a person can be judged to be walking based on how the arms and legs move in relation to each other. Therefore, during true imitation (henceforth imitation) attention is directed to the biological motion kinematics (joint configurations; limb velocity) of the observed person/model. Over repeated observations and physical attempts at imitating the model, a new sensorimotor pattern is represented and refined based on the available afferent and efferent sensorimotor feedback (Carroll & Bandura, 1982; Wolpert et al., 2011).

The mechanism underpinning imitation combines higher-order cognitive/attention and lower-level visuomotor processes (Bandura, 1977; Byrne & Russon, 1998; Heyes, 2001) embedded within a system linking perception with action (Prinz, 1997). Although not fully understood, individuals with autism exhibit different neuropsychological processes and behaviour during imitation compared to

typically developed individuals (Williams et al., 2004; Hamilton, 2013; Edwards, 2014; Vivanti & Hamilton, 2014). That is, people with autism often imitate the end-state goal (to reach a target) of an action (Hamilton et al., 2007; Bird et al., 2007; Wild et al., 2012), but show difficulties imitating the form (i.e., a gentle or harsh hand action) in which the movement goal is achieved (Rogers & Pennington, 1991; Smith & Bryson, 1994; Rogers et al., 1996; Rogers, 1999; Perra et al., 2008; Salowitz et al., 2013).

Extending upon original work that used descriptive measures (Rogers et al., 1996; Bernier et al., 2007; Vivanti et al., 2008), kinematic analysis has been used to determine what, if any, aspects of movement form (e.g., velocity; timing of peak velocity) are imitated (Wild et al., 2012; Stewart et al., 2013). Specifically, participants in the study of Wild et al. (2012) observed a human model performing an upper-limb pointing movement that differed in speed, while context was manipulated so the model aimed to targets (dots on a table), or to end space (dots removed). The notion is that, when targets are removed from the environment, the imitator focuses their attention on imitating the model's movement (kinematics; velocity) as opposed to merely reaching the target (dot) goal. The imitation of the model's movement is thought to occur via direct lower-level visuomotor mapping (Heyes, 2001; Southgate & Hamilton, 2008) and is suggested to be compromised in autism (Williams et al., 2004; Stewart et al., 2013; Edwards 2014). When targets are present, the goal is to aim at a target (an action goal), which occurs via goal-directed processes, and are less affected in autism (Hamilton et al., 2007). The results from Wild et al. (2012) showed only control participants imitated the different speeds when targets were removed. The lack of scaling of movement speed exhibited in participants with autism was accompanied by less time spent smoothly pursuing the

hand (with the eyes) and thus more shifts of gaze between the targets. It was suggested the shift in gaze, and thus attention away from the hand, may have modulated the amount of action-based biological motion information extracted from the model (Vivanti et al., 2008; Vivanti & Dissanayake, 2014), which thereby influenced the imitation of movement speed.

Notwithstanding an attentional contribution, reduced imitation of kinematics in individuals with autism has been linked to lower-level visuomotor processes (Stewart et al., 2013). For instance, imitation in a neurotypical control group was similar after observing a human or non-human model, thus indicating that top-down processes associated with social modulation (Spengler et al., 2010; Cook & Bird, 2012; Wang & Hamilton, 2012) did not exert an influence on behaviour. However, the autism group exhibited greater path length error and action duration in both observation conditions, which was attributed to impaired lower-level visuomotor processes that compromised self-other mapping in the mirror system (Nishitani, Avikainen, & Hari, 2004; Williams et al., 2004; 2006; Bernier et al. 2007). These lower-level processes link action-observation to action-execution, and sub-serve imitation by mapping observed biological motion onto the motor system (Iacoboni et al., 1999; 2001; Buccino et al., 2004; Di Dio et al., 2013).

Although previous work has provided novel contributions to understanding specific imitation deficits in autism by isolating the contribution of lower-level processes (Wild et al., 2012; Stewart et al., 2013), the examination of biological motion kinematics was undertaken by manipulating only the speed or amplitude of the modelled movement and by evaluating performance based on data from the whole imitation session. In terms of biological kinematics, the aforementioned context requires an imitator to scale an existing motor pattern (upper-limb

movement) to meet new task demands (e.g., faster movement), but does not isolate whether the deficit is attributable to imitating specific lower-level properties (e.g., velocity) of biological motion kinematics. To this end, to further examine imitation of biological motion in individuals with and without autism a novel protocol was employed that required participants to imitate movements that had distinctly different, but still biologically plausible, movement kinematics (Hayes et al., 2014). The experimental models displayed movement that had exactly the same spatial and temporal outcomes as a control model, but with a velocity profile of either *typical* or *atypical* kinematics. The *atypical* model ensured that an observer had to configure the sensorimotor system to represent the novel movement kinematics, as opposed to the *typical* model that could be achieved by rescaling an existing representation of a typical upper-limb aiming movement (Vivanti et al. 2008; Hayes et al., 2009). To control for top-down influences a protocol was used that minimised social attention (Cook & Bird, 2012; Wang & Hamilton, 2012) by presenting a non-human agent model (white-dot) with limited social context. To control for visual attention towards end-state target goal-directed features of the task environment (Vivanti et al., 2008; Wild et al., 2012), target goals were only displayed in half of the imitation trials in order to encourage attention towards the trajectory of the model. Consistent with previous work examining imitation of biological motion kinematics (Wild et al., 2012; Stewart et al., 2013) it can be expected that the findings of *Chapter Two* will demonstrate that compared to the neurotypical control group, the autism group will demonstrate similar imitation fidelity of the *typical* biological kinematics, yet will demonstrate low-fidelity imitation of *atypical* biological kinematics.

Furthermore, because imitation is an active process whereby a novel representation is developed and refined over repeated observations, it might be the

case that important information about imitation adaptation is masked by collapsing the analysis over all trials. An alternative approach that can reveal more about adaptation is to evaluate performance in the early and late stages of imitation, which is a typical in observation learning studies (Byrne & Russon, 1998; Bird & Heyes, 2005; Hayes, Ashford, & Bennett, 2008). Previous work has demonstrated successful motor adaptation in those with autism. For example, when required to catch a ball that varied in weight (i.e., light or heavy), children with autism showed similar rates of adaptation in hand displacement across practice as their typically developing counterparts (Mostofsky et al., 2004). This successful adaptation exhibited by children with autism was attributed towards a normal or compensatory cerebellum (Mostofsky et al., 2006; Gidley Larson et al., 2008; Fuentes et al., 2011). Therefore, to examine adaption performance, the early-phase and late-phase of imitation will be examined. Based on this abovementioned work (see also Haswell et al., 2009; Izawa et al., 2012) it can also be hypothesised that individuals with autism will show an adaptation effect over trials during imitation.

2.3 Method

2.3.1 Participants

Fifteen typical control participants (14 male; 1 female) and 15 participants with autism (14 male; 1 female) volunteered for the study. The volunteers with autism were recruited from an autistic society in North West of England, the University of Manchester, UK, and Liverpool John Moores University, UK. The volunteers were provided with a participant information sheet and selected if they consented to be part of the study. The control participants were recruited from

Table 2.1 Participant characteristics of the autism and control groups.

	Autism (n= 15)		**Control (n = 15)**		
	Mean (SD)	**Range**	**Mean (SD)**	**Range**	***P* Value**
Chronological Age	26 (8) years	18 - 44	26 (9) years	18 - 45	0.835
IQ:					
Full Scale	106 (10)	89 - 119	109 (7)	98 - 119	0.333
Verbal	104 (11)	88 - 127	108 (8)	95 - 122	0.218
Performance	105 (10)	90 - 128	106 (11)	90 - 124	0.771
ADOS:					
Total	10 (2)	8 - 16			
Communication	4 (1)	2 - 16			
Social Interaction	6 (2)	5 - 10			
Gender	14 M: 1F		14 M: 1F		

Liverpool John Moores University, UK. All participants were right-hand dominant (evaluated using the Edinburgh Handedness Inventory) (Oldfield, 1971), had normal or corrected-to-normal vision and were screened via self-report for the following exclusion criteria: dyspraxia, dyslexia, epilepsy and other neurological or psychiatric conditions. The participants with autism had a diagnosis of autism, Asperger's syndrome or autism spectrum disorder by an independent clinician. Diagnosis was confirmed by a researcher trained (with research-reliability status) in the administration of module 4 of the Autism Diagnostic Observation Schedule-2 (ADOS-2) (Lord et al., 2000). All participants with autism met the threshold for autism spectrum disorder on the ADOS-2 total classification score, and on the communication and reciprocal social interaction subscales. Groups were equated for age, and matched for full-scale IQ, and the verbal and performance subscales using the Wechsler Abbreviated Scale of Intelligence-2 (WASI-II) (Wechsler, 1999) which was confirmed by an independent samples t-test. Sample characteristics are presented in **Table 2.1**. The experiment was designed in accordance with the 1964 deceleration of Helsinki and approved by the local research ethics committee.

2.3.2 Apparatus

Participants sat facing a 21-inch CRT monitor (Iiyama Vision Master 505) operating with a resolution of 1280 x 1024 pixels and a refresh rate of 85 Hz, located on a table at a viewing distance of approximately 555 mm. The monitor was connected to a desktop PC (Dell Optiplex GX280), which received input from a graphics tablet and hand-held stylus (Wacom Intuos Pro XL) (**Figure 2.1a**). Experimental stimuli were generated on the desktop PC using the COGENT toolbox

(developed by John Romaya at the Laboratory of Neurobiology at the Wellcome Department of Imaging Neuroscience) implemented in MATLAB (Mathworks Inc.).

2.3.3 Procedure

Participants were provided with general instructions to "*watch and then copy the movement displayed by a white dot on the computer monitor*". The models (i.e., a non-human agent) displayed a single horizontal trajectory that originated from a home-target (diameter = 12.50 mm) or home-position (i.e., no target) on the left-hand side of the screen and terminated at an end-target (diameter = 12.50 mm) or end-position on the right-hand side of the screen (**Figure 2.1a**). The movement amplitude was 200 mm and total duration was 1700 ms. To examine imitation of biological motion, three models were created that displayed either *typical, atypical* or *constant* velocity profiles. The *typical* model was created by a human volunteer who practiced the task of typical goal-directed aiming movements using a hand-held stylus on a graphics tablet until a white-dot (diameter = 6.25 mm), which represented the stylus cursor, moved from the home-target to end-target in exactly 1700 ms. The model displayed a typical (Flash & Hogan, 1985; Elliott et al., 2010) bell-shaped velocity profile (displacement time-series is displayed as the dashed-black trace in **Figure 2.1b**) that had a magnitude of peak velocity equal to 0.200 mm/ms that occurred at 44 % of the movement duration. The *atypical* biological motion (solid-black trace in **Figure 2.1b**) was created by the same volunteer, but instead an atypical movement was practiced over the same amplitude and duration. The *atypical* biological motion had a magnitude of peak velocity equal to 0.410 mm/ms that occurred at 18 % of the movement duration. The method of using a human volunteer to generate both models was critical because it ensured the kinematics

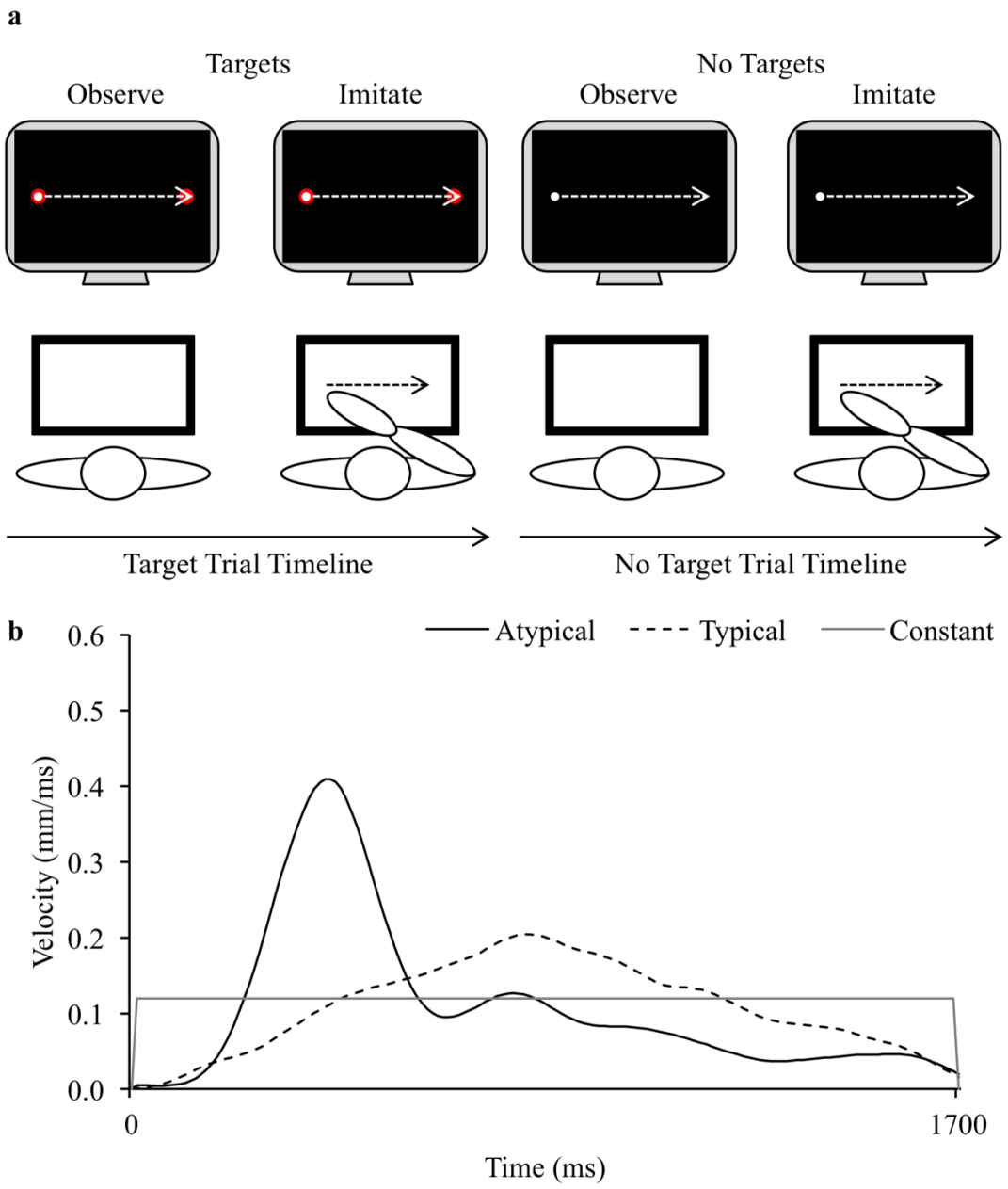

Figure 2.1. **(a)** A schematic representation of the laboratory/experimental set-up for the imitation task. The black outlined rectangle represents a graphics tablet. The white circle displayed on the CRT monitor represents the model. The single-segment movement is depicted by the arrow in the target (red target) and no-target conditions. **(b)** *Typical* (dashed-black trace), *atypical* (solid-black trace) and *constant* (solid-dark-grey trace) velocity models presented as a function of time.

were biological in origin and could be reproduced by the participants. The model displaying *constant* velocity was created according to the amplitude (200 mm) and time (1700 ms) constraints associated with the task. The model displayed the exact movement time and moved at constant velocity in the horizontal axis (0.118 mm/ms), with no deviations in the perpendicular axis (**Figure 2.1b**).

Volunteers performed 14 blocks of 6 trials (84 trials). A block contained the *typical*, *atypical* and *constant* velocity models, each performed in the target and no-target conditions. Trial order within a block, as well as block order, was randomised across volunteers. Prior to the experimental phases, all volunteers completed a familiarisation period that replicated the conditions of the imitation task. Volunteers performed four trials, 2 trials representing the target condition, and 2 trials representing the no-target condition. Each trial commenced with the model cursor positioned in the home-position after which it moved to the end-position with a *constant* velocity. The use of this model ensured construct validity by preventing volunteers experiencing biological motion before the imitation trials. Participants were not informed about the time duration of the movement, the different types of stimulus, or the end-state target manipulation. Therefore, after observing a model, participants were only provided with a general instruction to copy the model (not a specific instruction to copy a certain aspect of the model; e.g., the kinematics) by moving the stylus on the tablet so that the cursor moved to the end-target (i.e., target condition), or end-position (i.e., no-target condition), as per the movement of the model. All volunteers confirmed they understood the model, the instruction to imitate the model, and the sensorimotor association between the stylus on the graphics tablet and the corresponding movement of the cursor on the monitor.

2.3.4 Data Reduction

To quantify imitation of timing error and variability, movement duration was extracted from each trial, after which an error score was calculated (*timing error*) that reflected the signed (+ or -) difference between a participant's movement duration and model (e.g., 1900 ms – 1700 ms = 200 ms), and a variability score (*timing variability*) that represented the within-participant standard deviation of movement time within an attention condition. The start of the participant's movement was defined as the moment the centre of the cursor moved beyond the perimeter of the home-target (i.e., target condition) or home-position (i.e., no-target condition), whereas movement end equated to the moment the participant clicked the lower-button on the stylus. Intra-participant means were calculated from the first and last six trials associated with each model and target conditions.

To quantify imitation of movement kinematics the focus of the analysis was on x-axis data only (Hayes et al., 2016; Andrew et al., 2016). Within the x-axis position data, the start and end of the movement (as defined above) was identified. For each imitation trial, the resulting position data were filtered using a low pass 4th order autoregressive filter with an 8 Hz cut-off. The filtered data were then differentiated using a central difference algorithm to obtain velocity. A MATLAB routine extracted *peak velocity* and *time-to-peak-velocity* from each trial. Intra-participant means were calculated from the first and last six trials associated with each model and target conditions. These kinematic dependent variables were chosen as they provide discrete measures that accurately reflect whether participants imitate the magnitude and timing characteristics of the observed biological motion kinematics (Hayes et al., 2014).

2.3.5 Data Analysis

Descriptive analyses of data from the dependent variables using box-plots illustrated that no individual point(s) sat outside the upper- and lower-quartiles. Normality was quantified using Shapiro-Wilk tests that indicated data were normally distributed and did not violate the assumption of parametric analysis (all *ps* > 0.050). Data from all dependent variables were submitted to separate 2 group (autism; control) x 3 model (*atypical*; *typical*; *constant*) x 2 phase (early-phase; late-phase) repeated measures ANOVA. Significant main and/or interactions effects involving more than two means were analysed using Tukey HSD post-hoc procedure. Alpha was set at $p < 0.050$, and partial eta squared (η_p^2) expressed the size of the effect. To further express modulation across comparisons of interest (e.g., early-phase to late-phase) in the kinematic variables a percent change score was calculated using group mean data separately from the two phases in the following equation: ((late-phase – early-phase)/ early-phase)*100. Additional correlation analysis on relevant significant comparisons indicated by ANOVA were then completed to assess whether the dependent measure correlated with autism severity (i.e., ADOS total score).

2.4 Results

2.4.1 Timing Data

2.4.1.1 Timing Error

A main effect [$F(2, 56) = 51.267$, $p = 0.001$, $\eta_p^2 = 0.647$] of model indicated participants timing was significantly more accurate when imitating *atypical* (M =

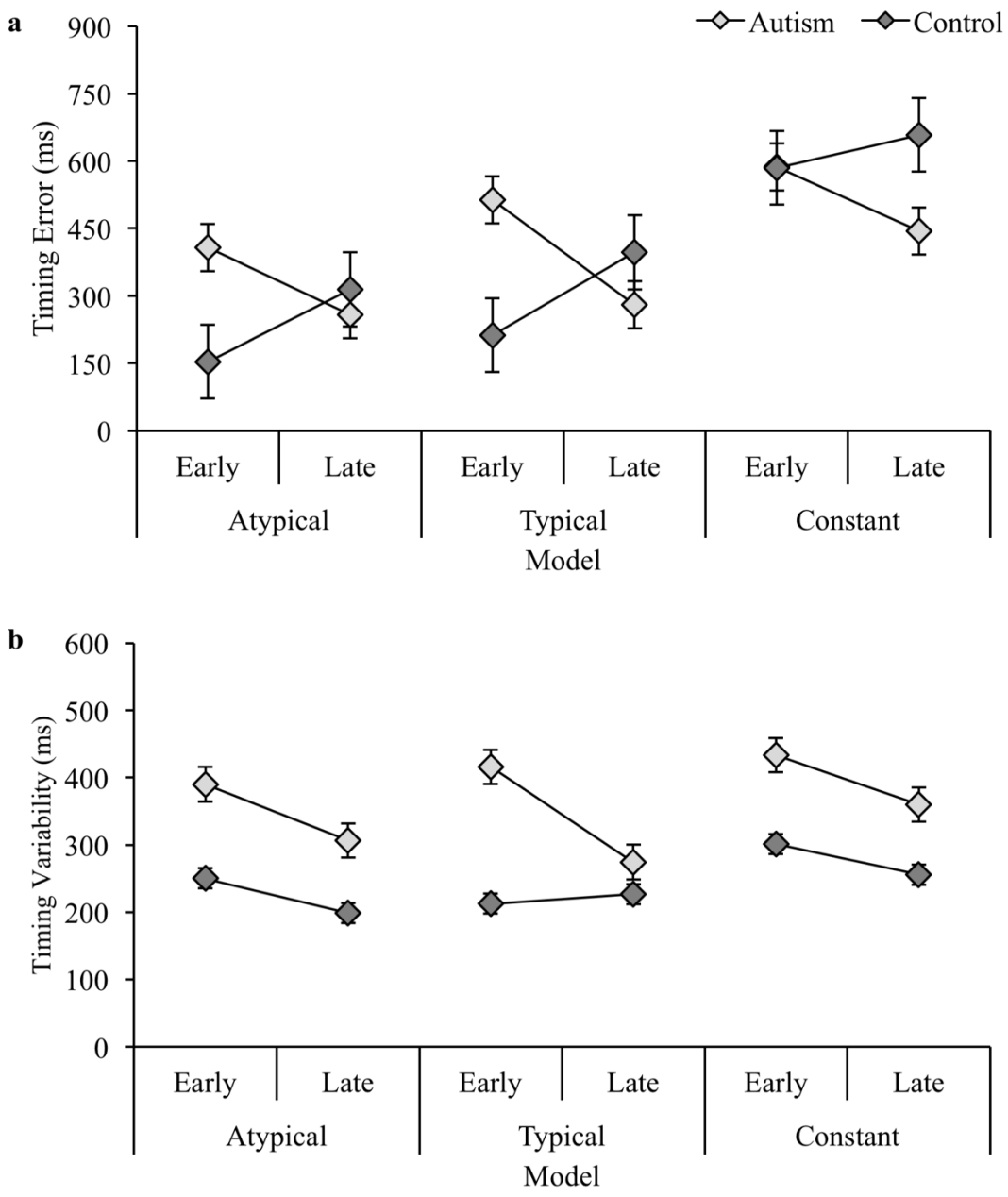

Figure 2.2 (a) Timing error and **(b)** timing variability for the imitation task (error bars represent standard error of the mean) presented as a function of group, model and phase.

283 ms; SD = 286 ms) compared to *typical* (M = 350 ms; SD = 282 ms) and *constant* (M = 568 ms; SD = 337 ms) velocity models, and when imitating *typical* compared to *constant* velocity models (**Figure 2.2a**). A group x phase interaction [F(1, 28) = 9.480, $p = 0.005$, $\eta_p^2 = 0.253$] indicated that timing error significantly decreased by 175 ms (35 % change) from the early-phase to the late-phase for the autism group. Out of the fifteen participants in the autism group, eight decreased motor timing error by 421 ms (65 % change), the remaining seven participants in the autism group slightly increased motor timing error by 106 ms (32 % change). Timing error significantly increased by 139 ms (44 % change) from the early-phase to the late-phase for the control group (**Figure 2.2a**). Correlation analysis revealed no relationship between motor timing error in the early-phase and ADOS total score (Pearson's r(15) = 0.118, $p = 0.338$) or late-phase and ADOS total score (Pearson's r(15) = -0.022, $p = 0.470$). There was no significant main effect of target [F(1, 28) = 3.476, $p = 0.073$, $\eta_p^2 = 0.110$] which indicated that both group had similar motor timing error when visual targets were present (M = 429 ms; SD = 287 ms) or removed (M = 372 ms; SD = 317 ms).

2.4.1.2 Timing Variability

A main effect [F(2, 56) = 4.679, $p = 0.013$, $\eta_p^2 = 0.143$] of model indicated participants timing was significantly less variable when imitating *atypical* (M = 286 ms; SD = 182 ms) and *typical* (M = 282 ms; SD = 176 ms) compared to *constant* (M = 337 ms; SD = 196 ms) velocity models (**Figure 2.2b**). Although a group main effect [F(1, 28) = 11.610, $p = 0.002$, $\eta_p^2 = 0.293$] indicated timing variability was significantly lower for the control (M = 241 ms; SD = 120 ms) than autism (M = 363 ms; SD = 217 ms) group (**Figure 2.2b**), a group x phase interaction [F(1, 28) =

4.770, $p = 0.037$, $\eta_p^2 = 0.146$] indicated that timing variability significantly decreased by 99 ms (24 % change) from the early-phase to the late-phase for the autism group. Out of the fifteen participants in the autism group, twelve decreased motor timing variability by 145 ms (65 % change), the remaining three participants in the autism group slightly increased motor timing variability by 106 ms (21 % change). There was a non-significant decrease of 27 ms (11 % change) in timing variability from the early-phase to the late-phase for the control group (**Figure 2.2b).** Correlation analysis revealed no relationship between motor timing variability in the early-phase and ADOS total score (Pearson's r(15) = 0.261, $p = 0.174$) or late-phase and ADOS total score (Pearson's r(15) = -0.060, $p = 0.415$). There was no significant main effect of target [F(1, 28) = 2.293, $p = 0.141$, $\eta_p^2 = 0.076$] which indicated that both group had similar motor timing variability when visual targets were present (M = 287 ms; SD = 167 ms) or removed (M = 317 ms; SD = 203 ms).

2.4.2 Kinematic Data

2.4.2.1 Peak Velocity

A main effect of model [F(2, 56) = 74.405, $p = 0.001$, $\eta_p^2 = 0.727$] for peak velocity indicated magnitude was higher when imitating *atypical* (M = 0.238 mm/ms; SD = 0.037 mm/ms), compared to *typical* (M = 0.192 mm/ms; SD = 0.045 mm/ms) and *constant* (M = 162 mm/ms; SD = 0.030 mm/ms) velocity models (**Figure 2.3a**). A group x phase interaction [F(1, 28) = 5.999, $p = 0.033$, $\eta_p^2 = 0.152$] indicated that magnitude of peak velocity increased by 0.024 mm/ms (12 % change) from the early-phase to the late-phase for the autism group. Out of the fifteen participants in the autism group, eleven increased peak velocity by 0.047 mm/ms (23

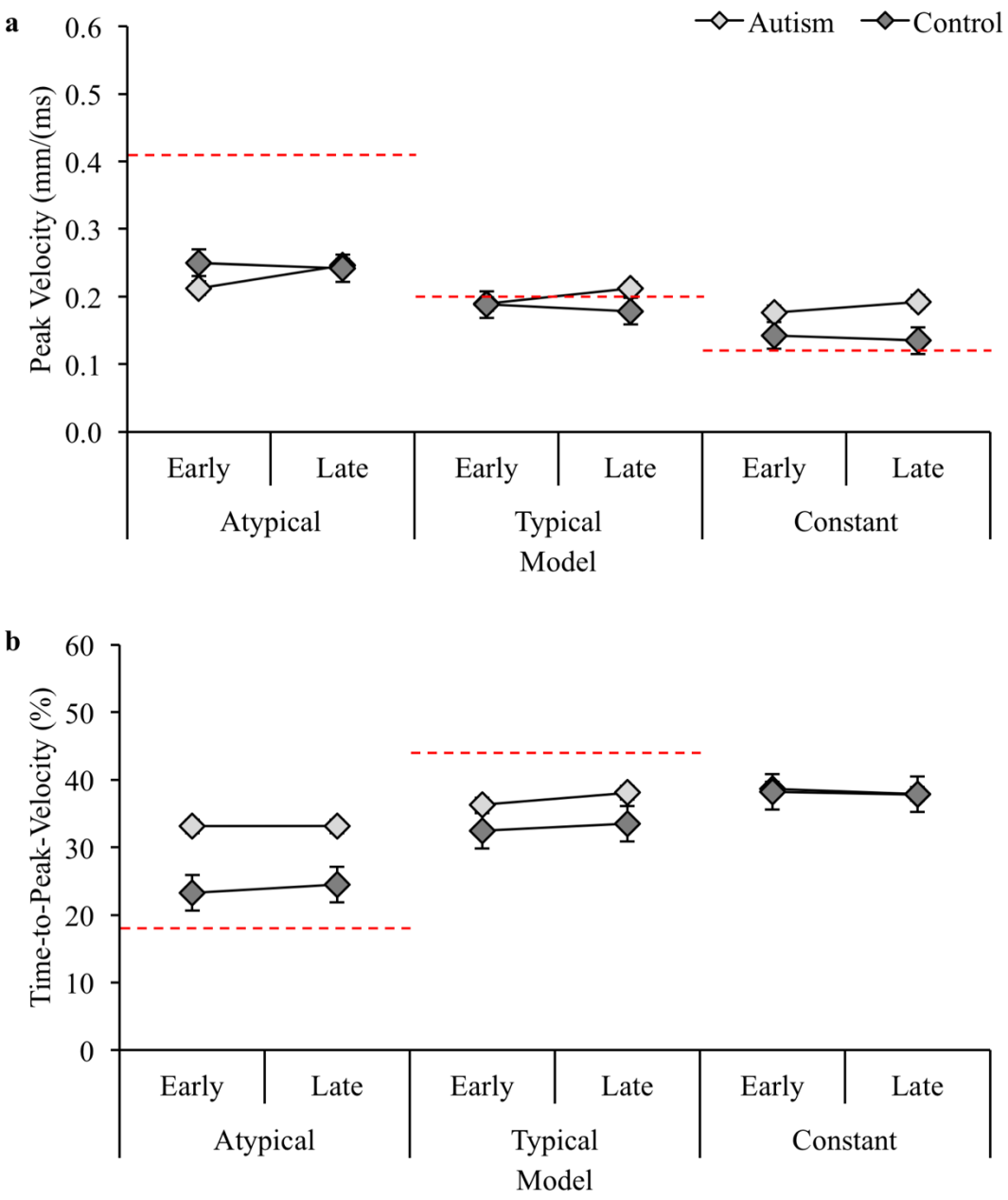

Figure 2.3 (a) Peak velocity and **(b)** time-to-peak-velocity for the imitation task (error bars represent standard error of the mean) presented as a function of group, model and phase. The dashed-red lines in **a** represent the magnitude of peak velocity for the *typical* (i.e., 0.410 mm/ms), *atypical* (i.e., 0.200 mm/ms) and *constant* (i.e., 0.118 mm/ms) models. In **b**, they represent the time-to-peak-velocity for the *typical* (i.e., 44 %) and *atypical* (i.e., 18 %) models.

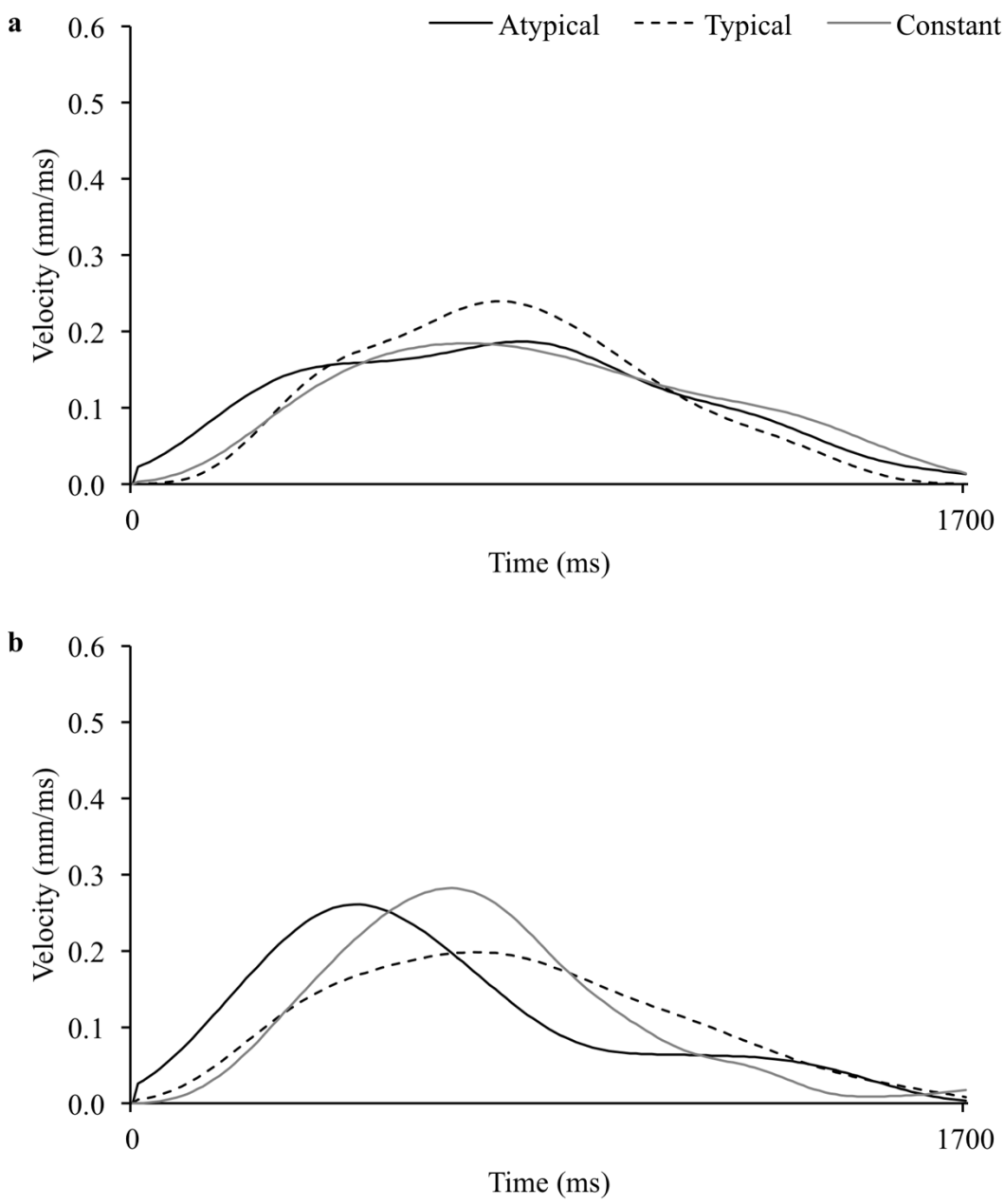

Figure 2.4 Velocity traces displaying exemplar kinematic data for the **(a)** autism and **(b)** control groups during imitation of the *typical* (dashed-black trace), *atypical* (solid-black trace) and *constant* (dashed-dark-grey trace) velocity models presented as a function of time.

% change), the remaining four participants in the autism group slightly decreased peak velocity by 0.012 mm/ms (7 % change). There was a non-significant decrease of 0.009 mm/ms (4 % change) in magnitude of peak velocity from the early-phase to the late-phase for the control group (**Figure 2.3a**). Correlation analysis revealed no relationship between peak velocity in the early-phase and ADOS total score (Pearson's r(15) = 0.028, p = 0.461) or late-phase and ADOS total score (Pearson's r(15) = -0.055, p = 0.423). There was no significant main effect of target [F(1, 28) = 2.513, p = 0.124, η_p^2 = 0.082] which indicated that both group had similar magnitude of peak velocity when visual targets were present (M = 0.194 mm/ms; SD = 0.065 mm/ms) or removed (M = 0.200 mm/ms; SD = 0.074 mm/ms).

2.4.2.2 Time-to-Peak-Velocity

A main effect of model [F(2, 56) = 41.536, p = 0.001, η_p^2 = 0.597], where peak velocity occurred significantly earlier when imitating *atypical* (M = 28 %; SD = 9 %) compared to *typical* (M = 35 %; SD = 10 %) and *constant* (M = 38 %; SD = 12 %) velocity models (**Figure 2.3b**). This was superseded by a group x model interaction [F(1, 28) = 8.569, p = 0.001, η_p^2 = 0.234], which indicated peak velocity occurred significantly earlier when imitating *atypical* velocity model (**Figure 2.3b**) for the control (M = 24 %; SD = 8 %) than autism group (M = 33 %; SD = 10 %). The early occurrence of peak velocity in the control group was more reflective of *atypical* biological motion (18 %; dashed-red line), than *typical* biological motion (44 %; dashed-red line). There was no significant difference (p > 0.050) between the groups when imitating *typical* (autism: M = 37 %; SD = 9 %, control: M = 33 %; SD = 10 %) and *constant* (autism: M = 38 %; SD = 11 %, control: M = 38 %; SD = 13 %) velocity models. Correlation analysis revealed no relationship between

Table 2.2 Mean (standard deviation) timing and kinematic data presented as a function of group, model and phase.

		Autism		**Control**	
Timing Data		**Early**	**Late**	**Early**	**Late**
Timing Error (ms)	**Atypical**	407	258	153	314
	Typical	513	280	212	397
	Constant	587	444	585	658
Timing Variability (ms)	**Atypical**	390	307	250	199
	Typical	416	274	212	227
	Constant	433	360	301	256
Kinematic Data		**Early**	**Late**	**Early**	**Late**
Peak Velocity (mm/ms)	**Atypical**	0.212 (0.045)	0.246 (0.045)	0.250 (0.049)	0.242 (0.043)
	Typical	0.189 (0.044)	0.212 (0.039)	0.188 (0.036)	0.178 (0.033)
	Constant	0.177 (0.039)	0.192 (0.038)	0.143 (0.025)	0.135 (0.019)
Time-to-Peak-Velocity (%)	**Atypical**	33 (11)	33 (9)	23 (8)	24 (7)
	Typical	36 (10)	38 (9)	32 (10)	33 (9)
	Constant	39 (11)	38 (11)	38 (13)	38 (12)

time-to-peak-velocity when imitating the *atypical* velocity model and ADOS total score (Pearson's r(15) = 0.401, p = 0.069), *typical* velocity model and ADOS total score (Pearson's r(15) = 0.375, p = 0.084) or *constant* velocity model and ADOS total score (Pearson's r(15) = 0.214, p = 0.222). There was no significant main effect of target [F(1, 28) = 1.358, p = 0.254, η_p^2 = 0.046] which indicated that both groups had similar timing of peak velocity when visual targets were present (M = 33 %; SD = 10 %) or removed (M = 34 %; SD = 10 %).

These above effects can be seen in the exemplar velocity traces illustrated in **Figure 2.4**. When imitating the *atypical* velocity model, peak velocity occurred significantly earlier in the movement for the control group (**Figure 2.4b**) than the autism group (**Figure 2.4a**). When imitating the *typical* velocity model, peak velocity occurred toward the midpoint of the movement for both groups (dashed-black trace; **Figure 2.4**). When imitating the *constant* velocity model, peak velocity occurred toward the midpoint of the movement for both groups (solid-dark-grey trace; **Figure 2.4**). For a full breakdown of each dependent variable see **Table 2.2**.

2.5 Discussion

Imitation, and imitation adaption (i.e., performance change from the early- to late-phase of imitation), of biological motion kinematics was examined using a novel behavioural protocol that required adults with and without autism to observe a model that displayed distinctly different but biologically plausible kinematics. Importantly, the *atypical* biological motion would not have been represented in the sensorimotor repertoire of observers, and thus could not be imitated by rescaling a typical upper-limb aiming movement. After observing an *atypical* model, participants in the

control group exhibited movements with a time-to-peak-velocity that occurred at 24 % of the movement trajectory. This early occurrence of peak velocity was similar to that displayed by the *atypical* model (time-to-peak-velocity = 18 %), and significantly different to the time-to-peak-velocity exhibited after observing *typical* (M = 34 %) and *constant* (M = 39 %) velocity control models. The presence of temporal correspondence between control participants' movements and the *atypical* model indicates high-fidelity imitation of biological motion kinematics based on lower-level sensorimotor processes (Brass et al., 2001; Gangitano, Mottaghy, & Pascual-Leone, 2001; Heyes 2001; Hayes et al., 2014; 2016).

Equivalent high-fidelity imitation of biological motion kinematics was not found for adults with autism. Although the magnitude of peak velocity was similar to control adults, there was a lack of temporal correspondence to the *atypical* model. The kinematic data showed time-to-peak-velocity occurred at 33 % of the movement trajectory, which was significantly different from the control group, but statistically similar to the time-to-peak-velocity exhibited when imitating the *typical* (M = 38 %) and *constant* velocity (M = 39 %) control models. In this respect, this data is consistent with other work that demonstrated differences between those with and without autism in imitating the form (e.g., a gentle or harsh hand action) of a movement (Smith & Bryson, 1994; Rogers et al., 1996; Hobson & Lee, 1999) or movement speed (Wild et al., 2012; Stewart et al., 2013). Importantly, however, the present findings extend understanding by showing differences in imitation are directly related to attenuation in representing the temporal occurrence of peak velocity associated with the observed biological motion kinematics.

Before interpreting this effect, it is important to highlight that the examination of biological kinematics was isolated using a protocol that controlled

higher order factors known to constrain imitation. First, an *atypical* model was displayed to ensure imitation was associated with representing novel biological kinematics, as opposed to presenting a movement that could be imitated using a pre-existing motor pattern recalled via higher-order semantic (Rumiati et al., 2005) or action-goal (Bekkering et al., 2000; Southgate & Hamilton, 2008) processes. Second, because imitation is modulated by social top-down factors (Chartrand & Bargh, 1999; Spengler et al., 2010; Cook & Bird, 2012; Wang & Hamilton, 2012), a non-human agent model was used that reduced the influence of emotional (Grèzes et al., 2009) and/or Theory of Mind (Baron-Cohen et al., 1999) constraints that are inherent in realistic human models. Third, the influence of end-state target goal attainment (Bekkering et al., 2000) was controlled for by displaying a movement trajectory that had no visual targets in half of the trials. In combination, the use of these control measures minimises the likelihood that the deficit in imitating biological motion kinematics in adults with autism is attributable to higher-order processes associated with reaching a target, or social imitation.

One explanation for the attenuation in imitating biological motion kinematics could be associated with lower-level processes that integrate visuomotor information (Théoret et al., 2005; Oberman et al., 2005; Dapretto et al., 2006; Williams et al., 2006; Stewart et al., 2013). For example, visuomotor integration of biological motion occurs through specialised visual areas (posterior superior temporal sulcus) (Grossman et al., 2000; Grossman, Battelli, & Pascual-Leone, 2005) and lower-level sensorimotor processes linked to the mirror system (Iacoboni, 2005; Southgate & Hamilton, 2008). These processes are part of a functional network that represents an observed movement by mapping the biological motion characteristics directly onto the motor system (Iacoboni et al., 1999; Rizzolatti & Craighero, 2004). However,

while lower-level processing deficits associated with visuomotor integration during self-other mapping (Williams et al., 2004; 2006; Stewart et al., 2013) could attenuate imitation of *atypical* biological kinematics, it is notable that adults with autism show intact mapping of biological motion during automatic imitation (Bird et al., 2007), which is a behavioural protocol that isolates processing to the lower-level mirror system. Moreover, results from neuropsychological work is mixed on whether such a fundamental impairment is present in autism (Hamilton, 2013).

The data revealed an adaptation effect whereby adults with autism became significantly more accurate at representing movement time (eight out of fifteen), reducing movement time variability (twelve out of fifteen), and increasing the magnitude of peak velocity (eleven out of fifteen) over trials during imitation, compared to the control group that showed no significant change. This adaptation must have been self-regulated, as opposed to augmented, because external feedback regarding movement time performance was not provided. This change in behaviour can be ascribed to active and functional true imitation, with sensorimotor adaptation most likely a result of attending to, and comparing against, the observed stimulus using feedforward and feedback processes (Carroll & Bandura, 1982; Byrne & Russon 1998; Kilner et al., 2007). Moreover, within the group of high-functioning autism participants recruited in the current study, it would seem this adaptation is a general process that is not related to autism severity as determined by correlations with ADOS total score. In addition to modulating the magnitude of peak velocity, the decrease in movement timing error also reduced the influence of end-state-target-goals such that timing and kinematics changed similarly for target and no-target conditions. Moreover, there was also no evidence found that the adult control group prioritised the attainment of an end-state-target-goal, over the imitation of *atypical*

biological kinematics, when present during observation. Although goal-directed imitation effects have been reported in complex movement sequences (Wild et al., 2010; 2012) or a full body point-light model (Hayes, Hodges, Huys, & Williams, 2007), it seems the target was less constraining when individuals observed a point-light non-human agent model performing a single segment movement.

The fact that adults with autism became significantly more accurate at imitating movement time, and exhibited a magnitude of peak velocity that was similar to the control group, suggests visual attention was orientated to the information displayed by the non-human agent model. This effect is consistent with data showing visual attention to action features of a model (Vivanti et al., 2008), and non-human stimuli (Swettenham et al., 1998), is typical in autism, whereas attention to facial features differs from controls (Boucher & Lewis 1992; Bird, Catmur, Silani, Frith, & Frith, 2006; Vivanti et al. 2008). Moreover, because no other attention-distracting stimuli were present in the display, it is unlikely that reduced imitation of *atypical* biological kinematics was associated with visual attention being drawn away from the non-human agent model (Wild et al., 2012). A more parsimonious explanation is that the selective attention bias to movement time during imitation was controlled via alternative (and efficient) higher-order processes (Hamilton et al., 2007; Southgate & Hamilton, 2008; Wild et al., 2012). A possibility is the movement time goal was imitated using processes associated with action comprehension, which are functional in autism (Dinstein et al., 2010), and as such goal attainment was secured using an efficient pre-existing motor pattern. This interpretation is consistent with the kinematic data, which showed individuals with autism executed movements that exhibited typical (peak velocity occurred towards the mid-point of the trajectory; Elliott et al., 2010) motor control trajectories when

imitating both the *atypical* and *typical* models.

In addition to a goal-directed and action comprehension interpretation, the selective-attention bias to movement time may have modulated input to the lower-level mirror system. Input modulation is suggested to impact the activation, or development, of sensorimotor representations via the intentionally mediated orientation of visual attention (Heyes & Bird, 2007; Longo, Kosobud, & Bertenthal, 2008; Liepelt & Brass 2010; Heyes, 2011). Therefore, because it was not specified within the task instructions what aspect of the model to imitate, the self-selected focus on movement time may have regulated the lower-level processes such that this temporal variable was placed higher on the embedded hierarchy of imitation goals (Wöhlschlager et al., 2003; Hamilton & Grafton 2007; Hayes et al., 2014) than *atypical* kinematics. Although it is unclear if such input modulation is operational in autism (Vivanti & Hamilton, 2014), it has previously been shown that imitation of *atypical* biological kinematics and movement time in neurotypical volunteers can be differentially modulated using pre-specified verbal instructions (Hayes et al., 2014). For example, the imitation of *atypical* biological kinematics can be modulated if volunteers are instructed to focus attention on imitating the movement time goal. Likewise, imitation accuracy can be enhanced if selective-attention is directed to the kinematics. Therefore, it cannot be said for certain if the focus on motor timing in individuals with autism is causally related to deficits in lower-level self-other mapping processes and/or motor ability, or whether the attentional effect is a compensatory strategy. One way to determine if the attenuation in imitating *atypical* biological kinematics is associated with top-down attentional modulation is to present a similar non-human agent model and employ a selective-attention protocol that uses explicit instructions to guide observers to attend and imitate the *atypical*

biological kinematics (Stewart et al., 2013), as opposed to the observers self-selecting which action-based information to imitate.

When considering the findings in respect to the broader context of imitation in autism, it is important to highlight the study was designed to examine true imitation. True imitation is a fundamental developmental process as it underpins the acquisition of novel social, and important sensorimotor skills that facilitate everyday life such as, tying shoe laces or riding a bicycle, or playing ice hockey. Although the data showed an attenuation in the imitation of biological motion kinematics, it was shown that movement time error and variability was significantly improved. The implication is that sensorimotor adaption and representation (Gidley Larson et al., 2008) of movement time is intact in high-functioning adults with autism. These are first data to show this adaptation in a true imitation context and indicates adults with autism do imitate, but they seem to do so with a selective-attention bias to movement time over kinematics which was controlled via alternative higher-order processes (Southgate & Hamilton, 2008; Wild et al., 2012). Therefore, the challenge is to examine the possibility that adults with autism can learn to imitate and represent biological motion kinematics following specific manipulations to the learning context (e.g., practice type, instructions, feedback). If the results are positive, then social and environmental procedures can be implemented by clinicians and practitioners to facilitate acquisition of social and sensorimotor behaviours in autism.

2.6 Summary

To conclude, the aim of this chapter was to examine whether adults with autism have difficulty imitating *atypical* biological motion. Data presented in *Chapter Two*

demonstrate for the first time experimentally, that adults with autism have difficulties imitating the velocity characteristics associated with *atypical* biological motion kinematics. A further aim of the chapter was to examine adaption performance. Analysis of the early and late phases of imitation specified that compared to control participants, adults with autism became significantly more accurate at imitating movement time across trials. The positive change in behaviour confirmed they actively engaged in the task, and that sensorimotor adaptation during imitation is self-regulated in autism. The bias to movement time suggests the attenuation in imitating biological motion kinematics in autism is perhaps a compensatory strategy due to deficits in lower-level visuomotor processes associated with self-other mapping and/or motor ability, or that selective-attention input to the processes that represent *atypical* biological motion kinematics. The latter suggestion will be further examined in following chapter.

3 Low-Fidelity Imitation of Biological Kinematics in Autism is Not Associated with Focus of Visual Attention

3.1 Abstract

A deficit in imitating biological kinematics is a feature of autism spectrum disorders, and could be underpinned by altered visual attention. Here, selective-attention and eye movements in autism and controls were examined when imitating *atypical* and *typical* biological kinematics. To manipulate selective-attention, general-attention instructions not specifying what aspects of the model to imitate were provided in the control phase. In the experimental phase, selective-attention instructions directed visual attention towards biological kinematics. In the general-attention condition, both groups performed similarly at imitating *typical* biological kinematics (autism: M = 36 %, control: M = 36 %), but the control group (M = 28 %) was more accurate than the autism group (M = 32 %) at imitating the *atypical* biological kinematics. Data showed the autism group had similar timing (autism: M = 39 %, control: M = 41 %) and peak (autism: M = 11.22 deg/s, control: M = 11.47 deg/s) smooth pursuit eye velocity, as well as a similar number (autism: M = 4.84 saccades, control: M = 4.34 saccades), and amplitude (autism: M = 1.82 deg, control: M = 1.49 deg), of saccades, during action-observation of both models as the control group. With selective-attention instructions, imitation of *atypical* biological kinematics remained unchanged, with the control group (M = 25 %) more accurate than the autism group (M = 31 %). Only the control group became more accurate when imitating the *typical* biological kinematics (11 % change). Eye movements were again similar between the groups, and modulated to become closer to the model after receiving selective-attention instructions. Low-fidelity imitation of *atypical* biological kinematics is unlikely to be underpinned by difficulties tracking the model with the eye, and thereby the focus of visual attention.

3.2 Introduction

Imitation is a complex multimodal mechanism (Byrne & Russon, 1998; Subiaul, 2010) associated with copying familiar and unfamiliar action forms, or goals. It is important in social settings for facilitating interpersonal behaviour, and acquisition of language and movements. When observing unfamiliar actions that are not represented in a movement repertoire, the resulting motor imitation (Subiaul, 2010) has the primary objective to copy biological motion properties, rather than imitating an end-goal (e.g., touching the ear), and has minimal input from processes associated with social cognition (empathy). During motor imitation attention is directed to biological motion, and across repeated exposures combined with physical attempts at imitating the model, a new sensorimotor pattern is represented (Wolpert et al., 2011).

Although it is unclear whether imitation deficits are universally present in autism (Vivanti & Hamilton, 2014), people with this condition typically have problems imitating the form (e.g., gentle movement) of a movement (Rogers & Pennington, 1991; Smith & Bryson, 1994). Kinematic analysis (Wild et al., 2012; Stewart et al., 2013) has isolated low-fidelity motor imitation to specific characteristics (*Chapter Two*) of observed biological kinematics (e.g., velocity). Moreover, having controlled the modulatory effects of social-affective processes using a non-human agent model (Stewart et al., 2013), difficulties imitating biological kinematics have been linked to compromised self-other mapping associated with lower-level visuomotor processes (Williams et al., 2006; Dapretto et al., 2006) operating in an action-observation matching system (Iacoboni et al., 2001).

Motor imitation is an active and volitional process that engages higher-order social, cognitive, and attentional processes that differentially modulate lower-level

sensorimotor processing (Bandura, 1977; Heyes, 2011; Hamilton, 2013). For example, instructing neurotypical observers to direct overt visual attention to movement kinematics, as opposed to temporal parameters (e.g., movement time), results in enhanced imitation of biological motion stimuli (Hayes et al., 2014). Such an attentional influence is referred to as 'input' modulation because instructions modulate lower-level sensorimotor processing of the observed stimulus (Heyes & Bird, 2007; Heyes, 2011). In people with autism, input modulation has been found during contagious yawning (Senju et al., 2009), which is a response that facilitates joint attention during interpersonal contexts, and is underpinned by similar lower-level sensorimotor processes as those engaged during imitation (Senju, 2013). Indeed, although children with autism were originally found to execute fewer yawns whilst observing a model than a control group (Senju et al., 2007), this behaviour was reversed following explicit instructions that directed overt visual attention to the eye region of the model (Senju et al., 2009).

Differences in attentional focus during imitation in people with autism can also be found in their eye movement behaviour (Vivanti et al., 2008). For example, as well as showing low-fidelity imitation of biological kinematics compared to a neurotypical control group, adults with autism spend less time tracking the hand of a model and more time shifting gaze between an action end-point target and end-space (Wild et al., 2012). People with autism also typically orientate overt visual attention away from the eyes of a model (Hobson & Hobson, 2007; Vivanti & Dissanayake, 2014), which attenuates the processing of social information that describes communicative signals and relevance from the model (Klin et al., 2009). The implication is that rather than poor imitation of biological motion in autism being underpinned by a basic dysfunction in lower-level sensorimotor processes associated

with self-other mapping, there could be a modulatory effect of attention (Vivanti & Dissanayake, 2014), especially in the absence of explicit instructions. Indeed, it is notable that previous work on imitation of biological motion in autistic adults (Wild et al., 2012) and children (Vivanti et al., 2008) involved general instructions to "*copy what you saw*", and thus did not provide adequate explicit direction in terms of where to focus attention and what aspect of the movement to copy (*Chapter Two*; Stewart et al., 2013).

To this end, a two-phase study was conducted that examined the influence of attentional instructions (general or selective) on eye movements during action-observation and the subsequent motor imitation of biological kinematics. With the provision of general-attention instructions, it is expected that the findings of *Chapter Two* will be replicated that compared to a neurotypical control group, an autism group will demonstrate low-fidelity imitation of biological kinematics (*Chapter Two*). Then, if low-fidelity imitation in autism is associated with processes underpinning selective-attention, it is expected that the autism group to demonstrate high-fidelity imitation when instructed to explicitly pay attention, and intend to imitate biological kinematics. By quantifying eye movements, it can be determined if any differences in movement kinematics during imitation is associated with how participants pursued the model during action-observation. Finally, in order to examine whether imitation of biological kinematics in autism is based at a perceptual level (Freitag et al., 2008; Saygin et al., 2010). Participants will complete a judgment task whereby they will observe two models and judge whether they have similar or dissimilar movement trajectories. It is expected that consistent with previous work (Wild et al., 2012; Cook et al., 2013) adults with autism will show intact biological motion perception and is thus will not attributed with data in the imitated task.

3.3 Method

3.3.1 Participants

Twenty typical control participants (19 male; 1 female) and 20 participants with autism (19 male; 1 female) volunteered for the study. The volunteers with autism were recruited from an autistic society in North West of England, and Liverpool John Moores University, UK. The volunteers were provided with a participant information sheet and selected if they consented to be part of the study. The control participants were recruited from Liverpool John Moores University, UK. None of the volunteers participated in *Chapters Two* and thus were naïve to the experiment. Sample characteristics are presented in **Table 3.1**.

3.3.2 Apparatus

The apparatus used was identical to that in *Chapter Two*. Movement of the left eye was recorded at 250 Hz using an EyeLink eye tracker (SR Research) with remote optics, which were located just below the lower edge of the monitor. The host PC and EyeLink were synchronized using a TTL signal.

3.3.3 Procedure

3.3.3.1 Imitation Task

The imitation task consisted of a general-attention phase, followed by a selective-attention phase. In the general-attention phase, participants were provided with instructions to "*watch and copy the dot as it moves across the monitor*". In the selective-attention phase, participants observed and imitated exactly the same

Table 3.1 Participant characteristics of the autism and control groups.

	Autism (n= 20)		**Control (n = 20)**		
	Mean (SD)	**Range**	**Mean (SD)**	**Range**	***P* Value**
Chronological Age	22 (3) years	18 - 28	21 (2) years	18 - 26	0.226
IQ:					
Full Scale	100 (10)	82 - 116	105 (9)	92 - 123	0.122
Verbal	102 (12)	87 - 127	106 (10)	89 - 126	0.225
Performance	98 (12)	75 - 116	101 (9)	82 - 115	0.385
ADOS:					
Total	9 (2)	7 - 14			
Communication	3 (1)	2 - 5			
Social Interaction	6 (2)	3 - 9			
Gender	19 M: 1 F		19 M: 1 F		

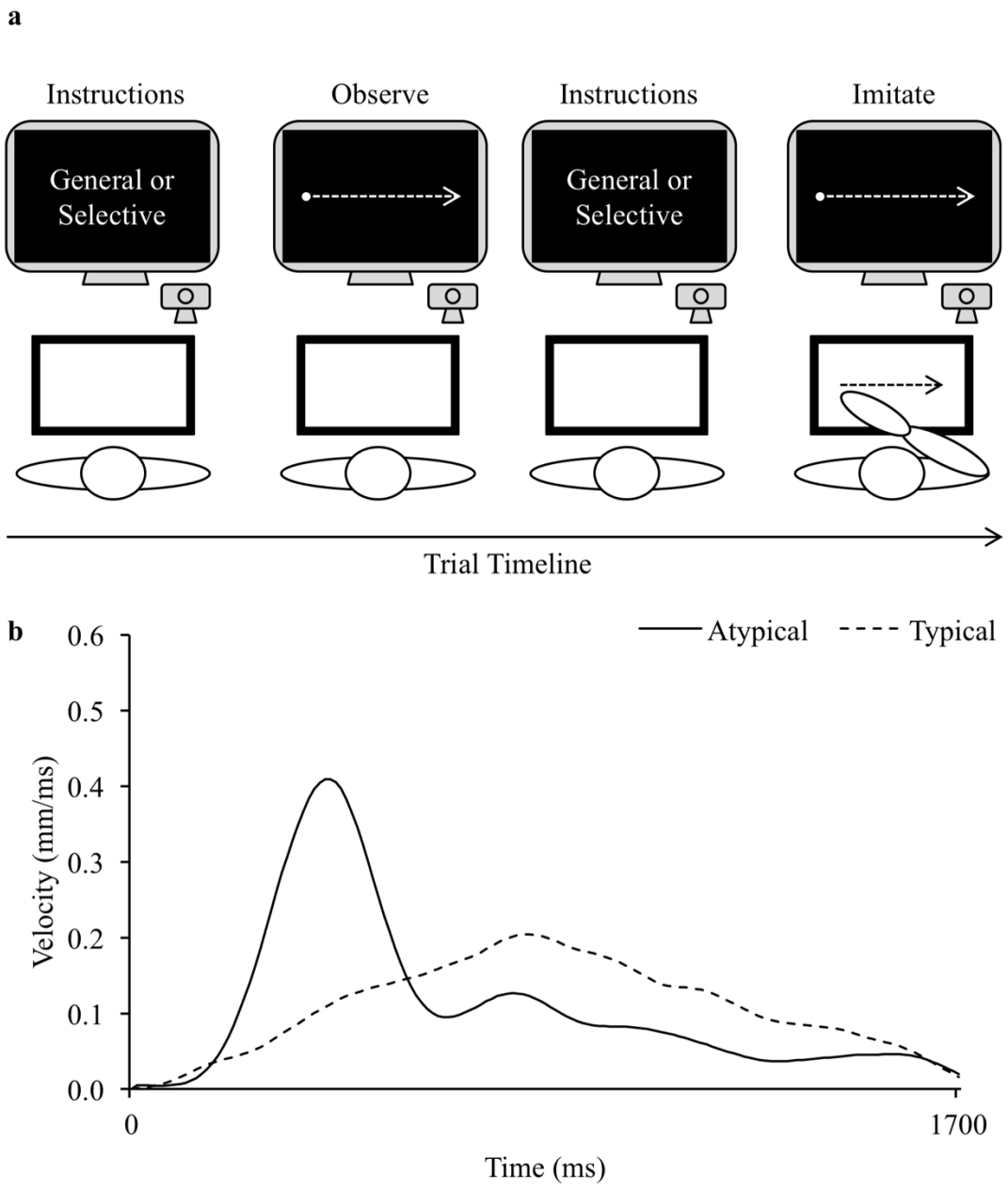

Figure 3.1 (a) A schematic representation of the laboratory/experimental set-up for the imitation task. The black outlined rectangle represents a graphics tablet. The white circle displayed on the CRT monitor represents the model. The single-segment movement is depicted by the arrow. **(b)** *Typical* (dashed-black trace) and *atypical* (solid-black trace) velocity models presented as a function of time.

stimuli, which ensured task complexity of imitating the two models was controlled. Although the presentation of the same stimuli may introduce practice effects given the increased exposure and imitation of the models, this procedure was deemed preferable in order to minimize potential carryover and use of the specific instructions from the first-phase to second-phase of the study. The specific nature of instructions provided in the selective-attention phase explicitly instructed participants to "*watch and pay attention to the dot's trajectory, with the intention to then copy the trajectory*". The latter instruction is characterised by: attention, intention and trajectory (Hayes et al., 2014). The models and procedure were identical to that *Chapter Two*, except the *constant* velocity model was removed.

Volunteers performed 5 blocks of 6 trials in each phase (60 trials). A block contained 3 *typical* or 3 *atypical* velocity models. Trial order within a block, as well as block order, was randomised across volunteers. Recording of the eye was performed for all observation trials and was obtained from seventeen volunteers with autism and eighteen control participants. Eye tracking was attempted on all participants but data were lost (3 autism; 2 control) due to recording difficulties.

3.3.3.2 Judgement Task

Volunteers completed a biological motion judgment task to determine ability at perceiving differences in biological kinematics. This was a same/different movement-pairs protocol that displayed two pre-recorded models, presented as a white dot (diameter = 6.25 mm) as per the imitation task. Models displayed either *typical*, *atypical* or *constant* velocity kinematics. The *typical* and *atypical* velocity models were identical to the motor imitation task. The *constant* velocity model was identical to that used during familiarisation. The movement-pairs protocol

commenced by displaying a model on trial *n*. Following observation of model *n*, participants observed model *n*+1 and were instructed to judge whether the model (e.g., *typical*) on trial *n* was the same (e.g., *typical*) or different (e.g., *atypical*) to the model presented on trial *n*+1. Participants pressed the 'S' key (for same) and 'D' (for different) on a QWERTY keyboard (Dell KB212) (**Figure 3.10a**). Following the first pair of models, this procedure continued for a total of 45 same/different movement-pairs. The 45 pairs were structured into 5 blocks, which contained 9 different combinations that were randomised to control for order effects. There was a 33 % chance that the models presented in the movement-pairs were the same.

3.3.3.3 Post-Experimental Debrief

Participants completed a debriefing session that was audio recorded. The session determined whether participants had engaged in the experiment and understood the task instructions. The questions posed were as follows: Did you notice anything about the movements you observed? How did you try to copy the dot? Did you understand what we meant by "*watch and pay attention to the dot's trajectory, with the intention to then copy the trajectory*"? Do you feel you changed how you imitated after receiving these task instructions?

3.3.4 Data Reduction

3.3.4.1 Imitation Task

To quantify imitation of timing accuracy, variability, and movement kinematics was identical to *Chapter Two*. Intra-participant means were calculated from 15 trials associated with each model and instruction condition.

3.3.4.2 Eye Movements

Analysis of eye behaviour was based on the x-axis data recorded from the left-eye during observation of the model stimuli. Saccades were identified using the proprietary algorithm in the EyeLink software, and then removed and replaced from the corresponding eye velocity using a linear interpolation routine (Bennett & Barnes, 2003). Desaccaded smooth eye velocity was low-pass filtered using a moving average zero-phase filter (40 ms window). To quantify how well the eyes pursued the observed model during each trial, we extracted the *peak* and *time-to-peak* smooth eye velocity, and the *number of saccades* and *saccade amplitude.* Intra-participant means were calculated from 15 trials associated with each model and instruction condition. These discrete measures of smooth and saccadic eye movements provide a means to quantify pursuit of the observed model (Orban de Xivry, Bennett, Lefevre, & Barnes, 2006), whereby it can be expected that overt visual attention coincides with the moving stimulus (Lovejoy, Fowler, & Krauzlis, 2009), albeit sometimes with a slight lead (Van Donkelaar & Drew, 2002). Covertly attending to other areas or locations would be possible, although effortful and unlikely given that there were no other relevant cues within the stimulus presentation.

3.3.5 Data Analysis

Data from all dependent variables were submitted to separate 2 group (autism; control) x 2 model (*atypical; typical*) x 2 instruction (general-attention; selective-attention) repeated measures ANOVA. To further express modulation across comparisons of interest (e.g., general-attention to selective-attention) in the kinematic variables a percent change score was calculated using group mean data

separately from the two attention conditions in the following equation: ((selective-attention – general-attention)/ general-attention)*100.

To quantify the accuracy of judging different motion kinematics, the total number of correct responses made by the autism and control groups was examined using an independent *t*-test. Alpha was set at $p < 0.050$.

3.4 Results

3.4.1 Imitation Task

3.4.1.1 Timing Data

3.4.1.1.1 Timing Error

A main effect [$F(1, 38) = 25.837$, $p = 0.001$, $\eta_p^2 = 0.405$] of model indicated participants timing was significantly more accurate when imitating *atypical* (M = 178 ms; SD = 296 ms) compared to *typical* (M = 295 ms; SD = 305 ms) velocity models (**Figure 3.2a**). A model x instruction interaction [$F(1, 38) = 25.837$, $p = 0.034$, $\eta_p^2 = 0.405$] indicated timing accuracy decreased by 77 ms (30 % change) from the general-attention to selective-attention condition when imitating *typical* velocity models ($p < 0.050$). There was a non-significant increase of 14 ms (8 % change) in timing accuracy from general-attention to selective-attention condition when imitating a*typical* velocity models ($p > 0.050$). There was no significant interaction for group x model [$F(1, 38) = 0.027$, $p = 0.871$, $\eta_p^2 = 0.001$], group x instruction [$F(1, 38) = 0.690$, $p = 0.411$, $\eta_p^2 = 0.018$], or group x model x instruction [$F(1, 38) = 1.676$, $p = 0.203$, $\eta_p^2 = 0.042$].

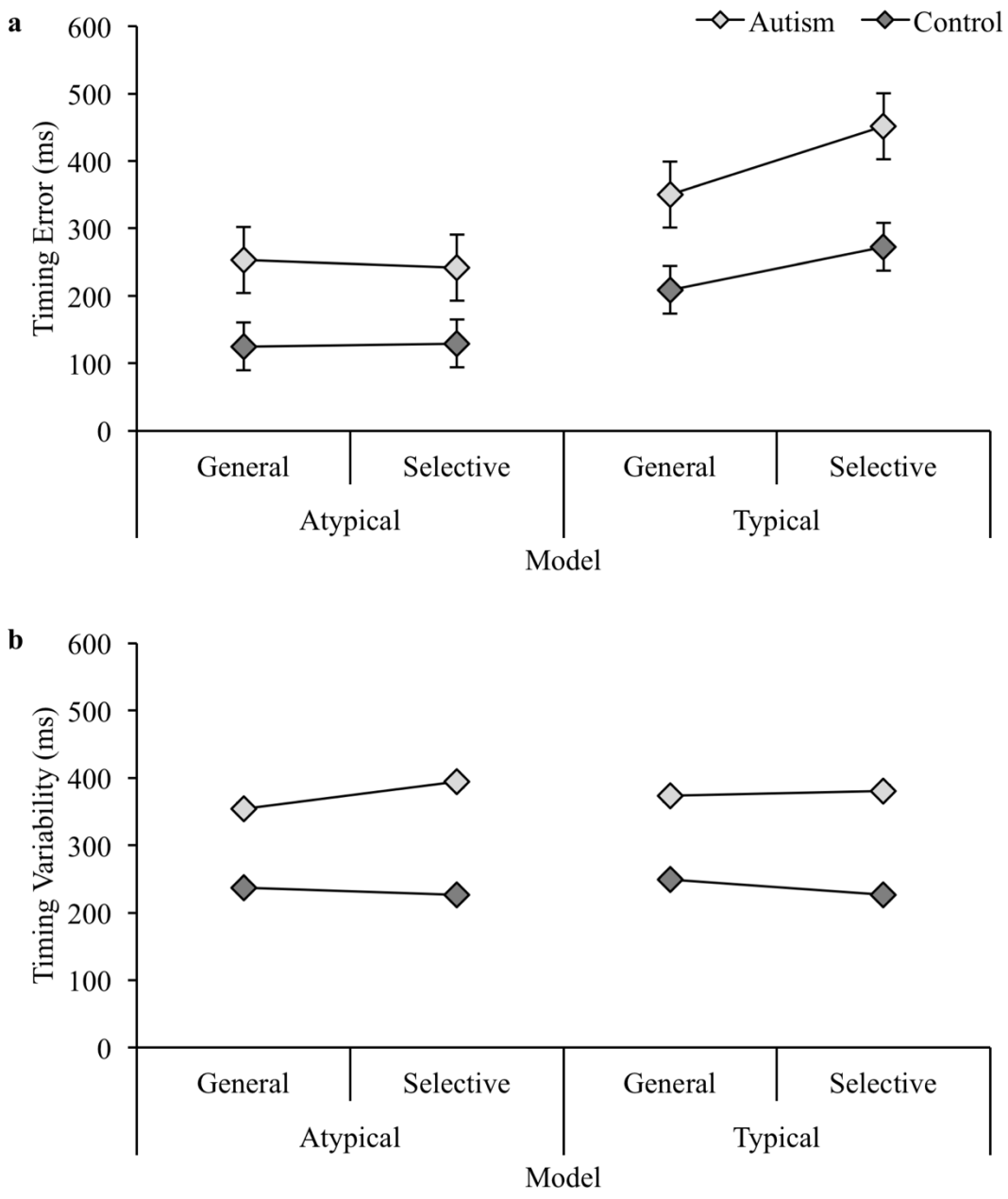

Figure 3.2 (a) Timing error and **(b)** timing variability for the imitation task (error bars represent standard error of the mean) presented as a function of group, model and instruction.

3.4.1.1.2 Timing Variability

A group main effect [F(1, 38) = 7.907, p = 0.001, η_p^2 = 0.349] indicated timing variability was significantly lower for the control (M = 234 ms; SD = 70 ms) than autism (M = 367 ms; SD = 144 ms) group (**Figure 3.2b**). There was no significant interaction for group x model [F(1, 38) = 0.063, p = 0.803, η_p^2 = 0.002], group x instruction [F(1, 38) = 1.045, p = 0.313, η_p^2 = 0.027], or group x model x instruction [F(1, 38) = 0.001, p = 0.977, η_p^2 = 0.001].

3.4.1.2 Kinematic Data

3.4.1.2.1 Peak Velocity

A group main effect [F(1, 38) = 7.907, p = 0.001, η_p^2 = 0.172] indicated peak velocity was higher for the control (M = 0.220 mm/ms; SD = 0.040) than autism (M = 0.192 mm/ms; SD = 0.046) group (**Figure 3.3a**). A main effect of model [F(1, 38) = 85.177, p < 0.001, η_p^2 = 0.691] for peak velocity indicated magnitude was higher when imitating *atypical* (M = 0.235 mm/ms; SD = 0.049), compared to *typical* velocity models (M = 0.178 mm/ms; SD = 0.037) (**Figure 3.3a**). A model x group interaction neared significance [F(1, 38) = 4.4076, p = 0.051, η_p^2 = 0.097] and indicated peak velocity was higher when imitating the *atypical* velocity models (**Figure 3.3a**) for the control (0.255 mm/ms; SD = 0.046) than autism group (0.215 mm/ms; SD = 0.051). There was no significant difference (p > 0.050) between the groups when imitating *typical* velocity models (autism: M = 0.170 mm/ms; SD = 0.040, control: M = 0.186 mm/ms; SD = 0.034). Correlation analysis revealed no relationship between peak velocity when imitating the *atypical* velocity models and ADOS total score (Pearson's r(20) = 0.024, p = 0.920) or *typical* velocity models

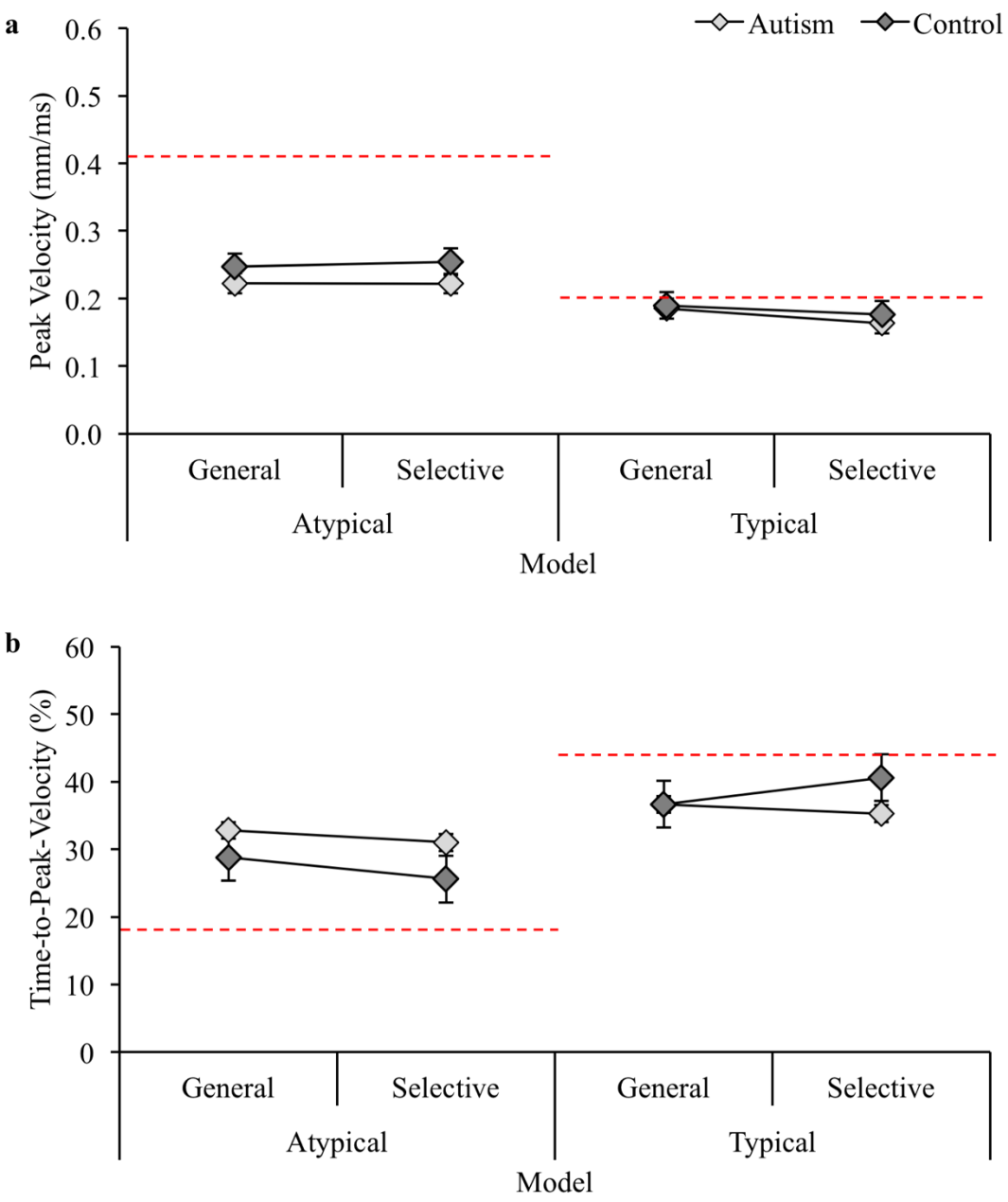

Figure 3.3 (a) Peak velocity and **(b)** time-to-peak-velocity for the imitation task (error bars represent standard error of the mean) presented as a function of group, model and instruction. The dashed-red lines in **a** represent the magnitude of peak velocity for the *typical* (i.e., 0.410 mm/ms) and *atypical* (i.e., 0.200 mm/ms) models. In **b**, they represent the time-to-peak-velocity for the *typical* (i.e., 44 %) and *atypical* (i.e., 18 %) models.

and ADOS total score (Pearson's r(20) = 0.360, $p > 0.119$). A model x instruction interaction [F(1, 38) = 9.908, $p = 0.001$, $\eta_p^2 = 0.207$] indicated magnitude of peak velocity decreased by 0.016 mm/ms (autism = 12 % change, control = 6 % change) from the general-attention to selective-attention condition when imitating *typical* velocity models ($p < 0.050$). There was a non-significant increase of 0.001 mm/ms (autism = 2 % change, control = 1 % change) in peak velocity from general-attention to selective-attention condition when imitating *atypical* velocity models ($p > 0.050$). There was no significant interaction for group x instruction [F(1, 38) = 1.044, $p > 0.05$, $\eta_p^2 = 0.027$] or group x model x instruction [F(1, 38) = 0.061, $p > 0.050$, $\eta_p^2 = 0.002$].

3.4.1.2.2 Time-to-Peak-Velocity

There was no main effect of group [F(1, 38) = 0.405, $p > 0.528$, $\eta_p^2 = 0.011$]. There was a main effect of model [F(1, 38) = 53.496, $p < 0.001$, $\eta_p^2 = 0.585$], where peak velocity occurred significantly earlier when imitating *atypical* (M = 29 %; SD = 9 %), compared to the *typical* velocity models (M = 37 %; SD = 11 %) (**Figure 3.3b**). This was superseded by a model x group interaction [F(1, 38) = 12.492, $p = 0.001$, $\eta_p^2 = 0.247$], which indicated peak velocity occurred significantly earlier when imitating *atypical* velocity models (**Figure 3.3b**) for the control (M = 27 %; SD = 8 %) than autism group (M = 31 %; SD = 10 %). The early occurrence of peak velocity in the control group was more reflective of *atypical* velocity models (18 %; dashed-red line), than *typical* velocity models (44 %; dashed-red line). There was no significant difference ($p > 0.050$) between the groups when imitating *typical* velocity models (autism: M = 35 %; SD = 10 %, control: M = 38 %; SD = 11 %). Correlation analysis revealed no relationship between time-to-peak-velocity when imitating the

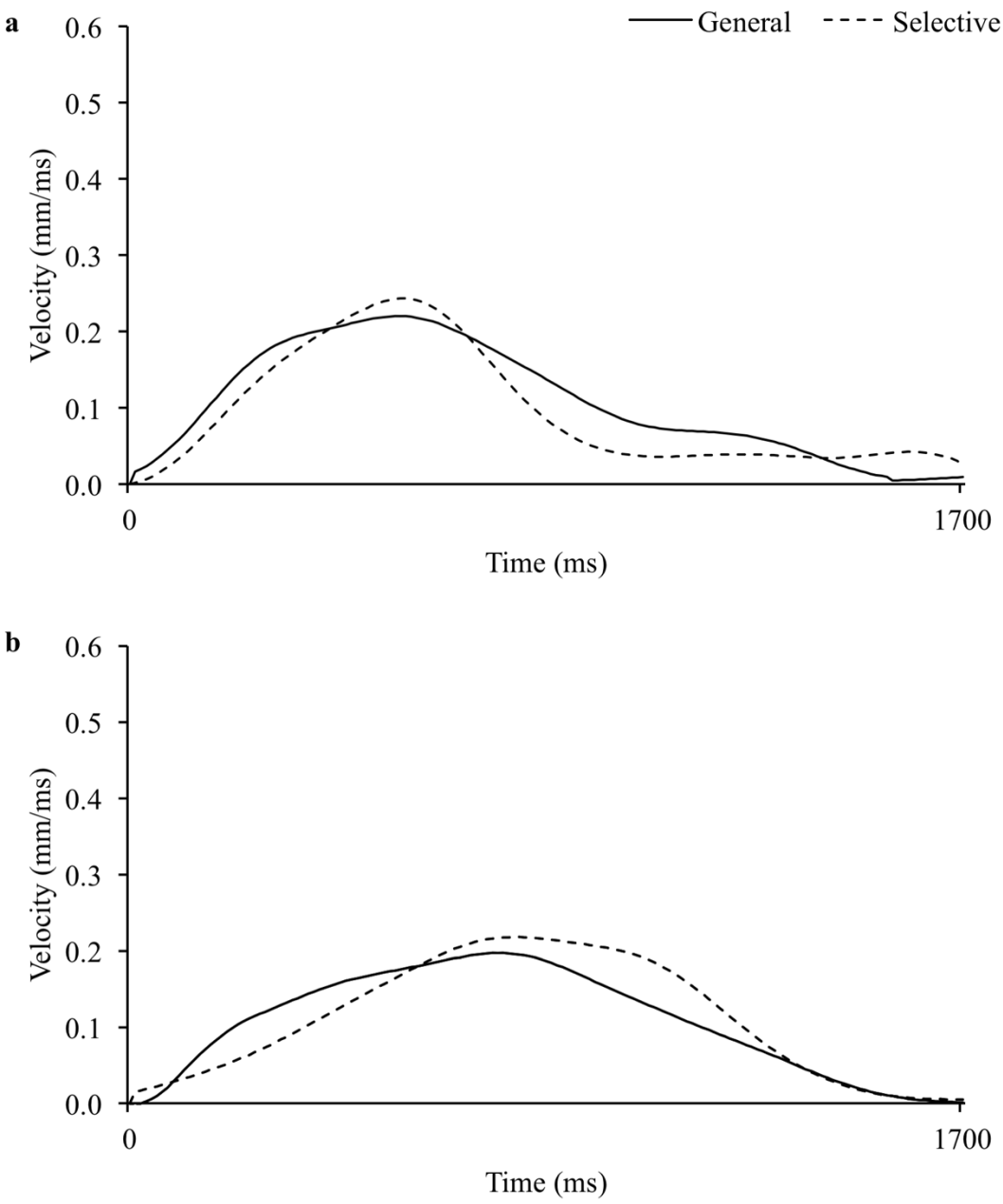

Figure 3.4 Velocity traces displaying exemplar kinematic data for the autism group during imitation of the **(a)** *typical* and **(b)** *atypical* velocity models in the general-attention (solid-black trace) and selective-attention (dashed-black trace) instructions presented as a function of time.

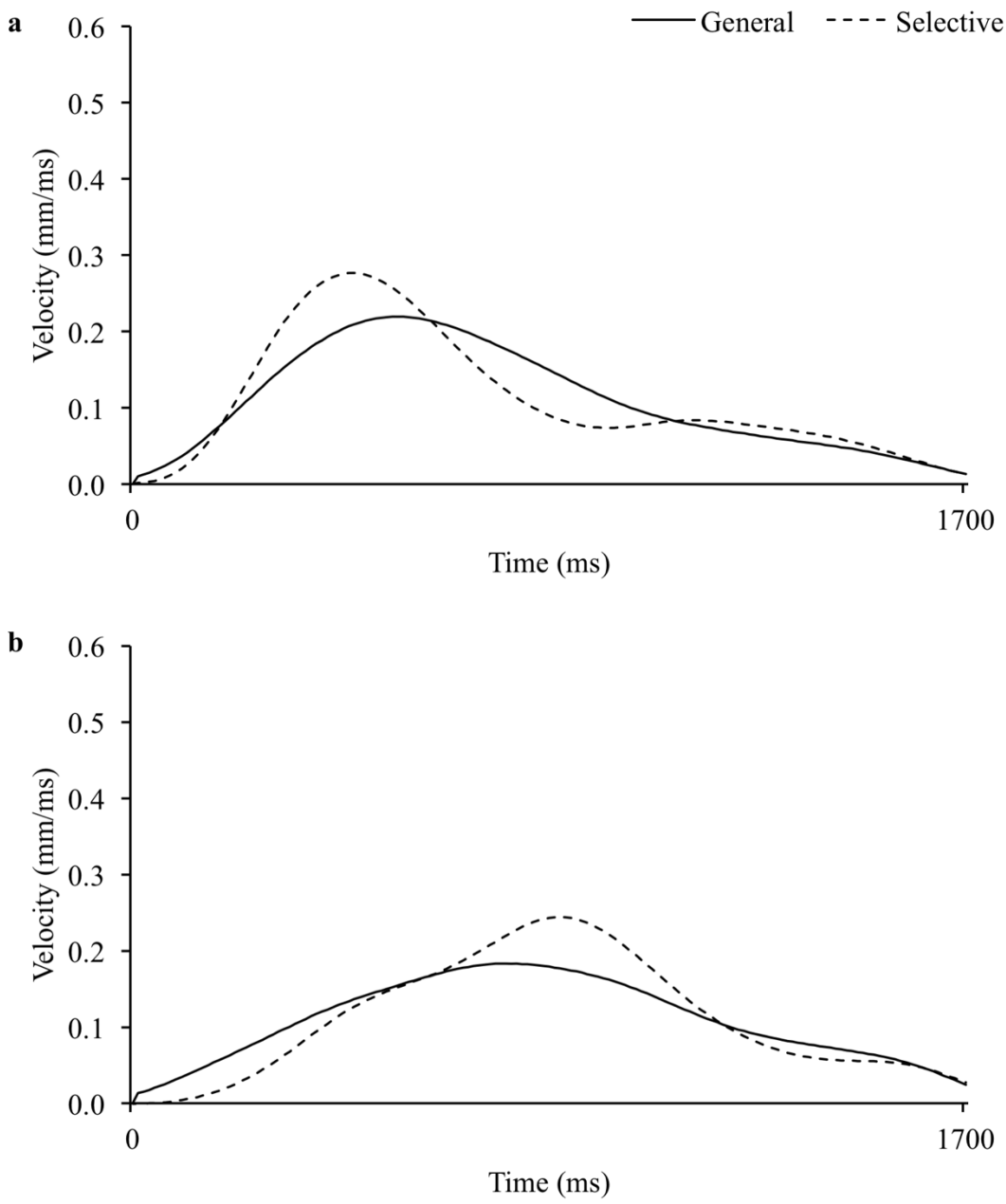

Figure 3.5 Velocity traces displaying exemplar kinematic data for the control group during imitation of the **(a)** *typical* and **(b)** *atypical* velocity models in the general-attention (solid-black trace) and selective-attention (dashed-black trace) instructions presented as a function of time.

Table 3.2 Mean (standard deviation) timing and kinematic data for the imitation task presented as a function of group, model and instruction.

		Autism		Control	
Timing Data		**General**	**Selective**	**General**	**Selective**
Timing Error (ms)	**Atypical**	271	296	71	74
	Typical	342	468	172	201
Timing Variability (ms)	**Atypical**	355	372	232	223
	Typical	370	369	254	225
Kinematic Data		**General**	**Selective**	**General**	**Selective**
Peak Velocity (mm/ms)	**Atypical**	0.217 (0.051)	0.212 (0.052)	0.254 (0.047)	0.257 (0.045)
	Typical	0.180 (0.040)	0.159 (0.040)	0.191 (0.037)	0.180 (0.031)
Time-to-Peak-Velocity (%)	**Atypical**	32 (9)	31 (10)	28 (8)	25 (8)
	Typical	36 (10)	34 (11)	36 (10)	40 (11)

atypical velocity models and ADOS total score (Pearson's r(20) = 0.307, $p = 0.187$) or *typical* velocity models and ADOS total score (Pearson's r(20) = 0.191, $p = 0.421$). A group x model x instruction interaction [$F(1, 38) = 8.976$, $p = 0.002$, $\eta_p^2 = 0.191$] indicated selective-attention had a specific modulatory effect on the timing of peak velocity. As illustrated in **Figure 3.3b**, imitation of *atypical* velocity models in both groups (autism: $p = 0.075$, control: $p = 0.108$) did not differ between general-attention and selective-attention instructions (autism = 5 % change, control = 8 % change). However, peak velocity in the control group occurred significantly ($p = 0.001$) later following selective-attention compared to general-attention instructions when imitating the *typical* velocity model. The 11 % change indicated the control group imitated with a time-to-peak velocity that was closer to the *typical* model (44 %; dashed-red line on **Figure 3.1b**). There was no significant ($p = 0.148$) change (6 %) for the autism group. Correlation analysis revealed no relationship between time-to-peak-velocity for general-attention condition and ADOS total score (*atypical*, Pearson's r(20) = 0.296, $p = 0.206$; *typical*, Pearson's r(20) = 0.211, $p = 0.373$) or selective-attention and ADOS total score (*atypical*, Pearson's r (20) = 0.318, $p = 0.172$; *typical*, Pearson's r (20) = 0.111, $p = 0.640$).

These above effects can be seen in the exemplar velocity traces illustrated in **Figures 3.4** and **3.5**. When imitating the *atypical* velocity models, peak velocity occurred significantly earlier in the movement for the control group (**Figure 3.5a**) than the autism group (**Figure 3.4a**). When imitating the *typical* velocity models, peak velocity occurred toward the midpoint of the movement for both groups during the general-attention condition (solid-black trace), yet peak velocity occurred significantly later in the movement for the control group (**Figure 3.5b**) than the

autism group (**Figure 3.4b**) during the selective-attention condition (dashed-black trace). For a full breakdown of each dependent variable see **Table 3.2**.

3.4.2 Eye Movements

3.4.2.1 Smooth Pursuit Data

3.4.2.1.1 Peak Velocity

The exemplar traces show similar magnitude, and timing, of smooth eye velocity for both groups when observing *atypical* (autism: **Figure 3.6a**, control: **Figure 3.7a**) and *typical* (autism: **Figure 3.6b**, control: **Figure 3.7b**) velocity models. After an expected delay in onset of eye motion relative to the stimulus (Barnes & Asselman, 1991). Smooth eye velocity was well matched to stimulus velocity and resulted in no significant main effects for group [$F(1, 33) = 0.055$, $p > 0.050$, $\eta_p^2 = 0.002$] or model [$F(1, 33) = 0.155$, $p = 0.697$, $\eta_p^2 = 0.005$]. An instruction main effect [$F(1, 33) = 5.018$, $p = 0.033$, $\eta_p^2 = 0.143$] indicated participants significantly increased peak velocity by 7 % (0.795 deg/s) from the general-attention to the selective-attention conditions. There were no interactions involving group x model [$F(1, 33) = 2.628$, $p = 0.115$, $\eta_p^2 = 0.081$], group x instruction [$F(1, 33) = 4.086$, $p = 0.052$, $\eta_p^2 = 0.120$], or group x model x instruction [$F(1, 33) = 3.740$, $p = 0.063$, $\eta_p^2 = 0.111$] (**Figure 3.8a**).

3.4.2.1.2 Time-to-Peak-Velocity

There was no main effect of group [$F(1, 33) = 0.115$, $p = 0.697$, $\eta_p^2 = 0.005$]. There was a main effect of model [$F(1, 33) = 21.464$, $p < 0.001$, $\eta_p^2 = 0.417$], where

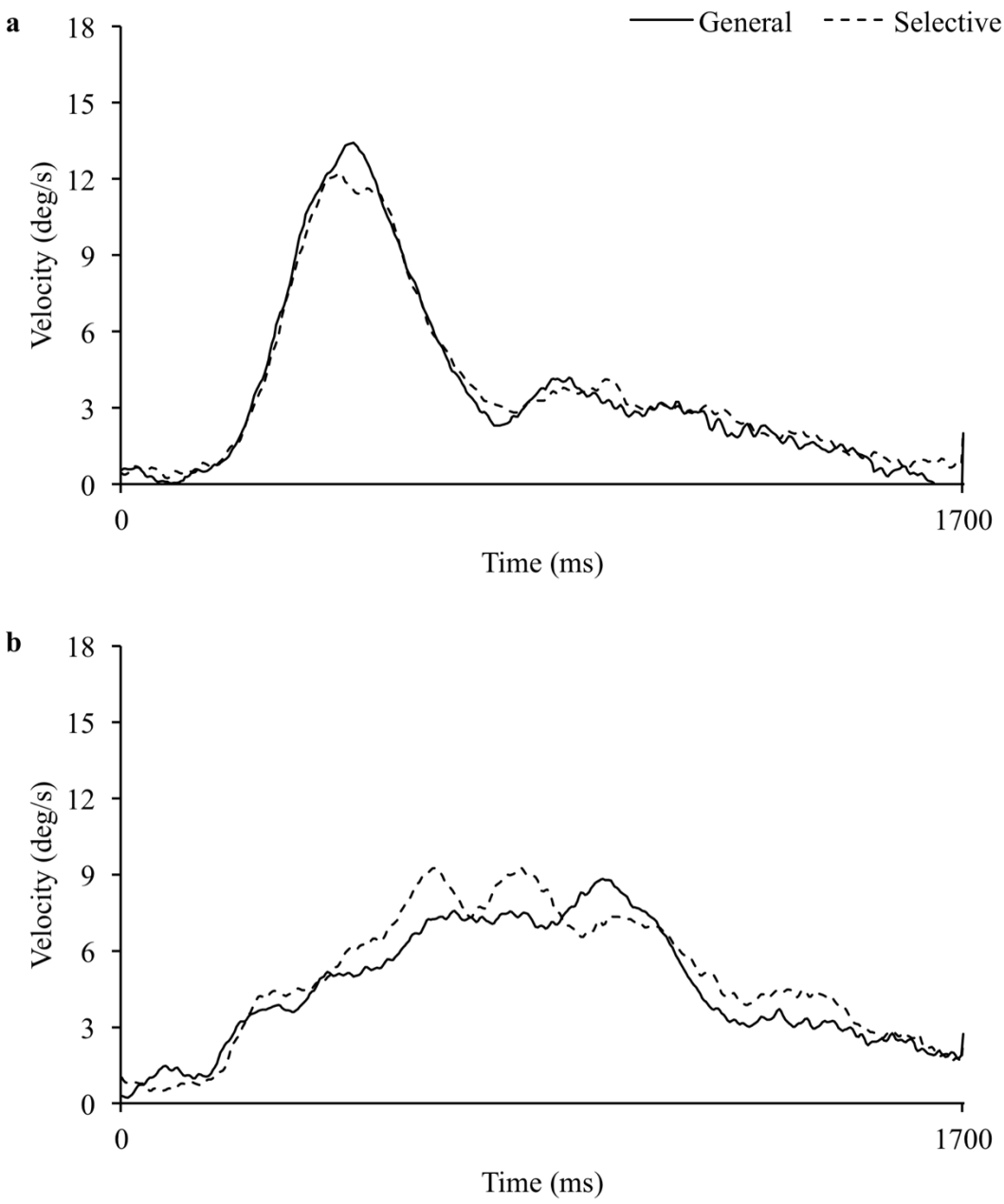

Figure 3.6 Smooth velocity traces displaying exemplar eye movement data for the autism group during observation of the **(a)** *typical* and **(b)** *atypical* velocity models in the general-attention (solid-black trace) and selective-attention (dashed-black trace) instructions presented as a function of time.

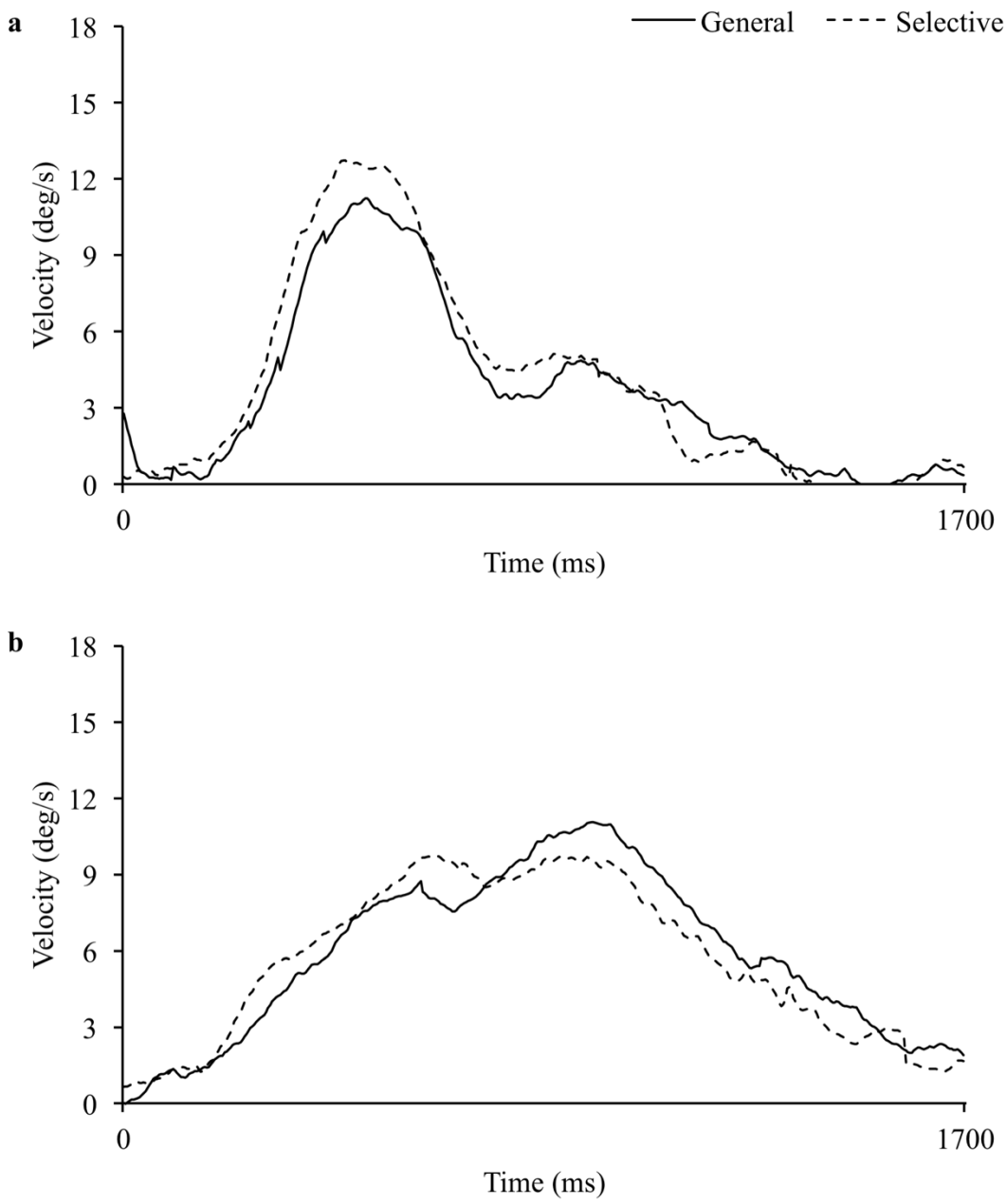

Figure 3.7 Smooth velocity traces displaying exemplar eye movement data for the control group during observation of the **(a)** *typical* and **(b)** *atypical* velocity models in the general-attention (solid-black trace) and selective-attention (dashed-black trace) instructions presented as a function of time.

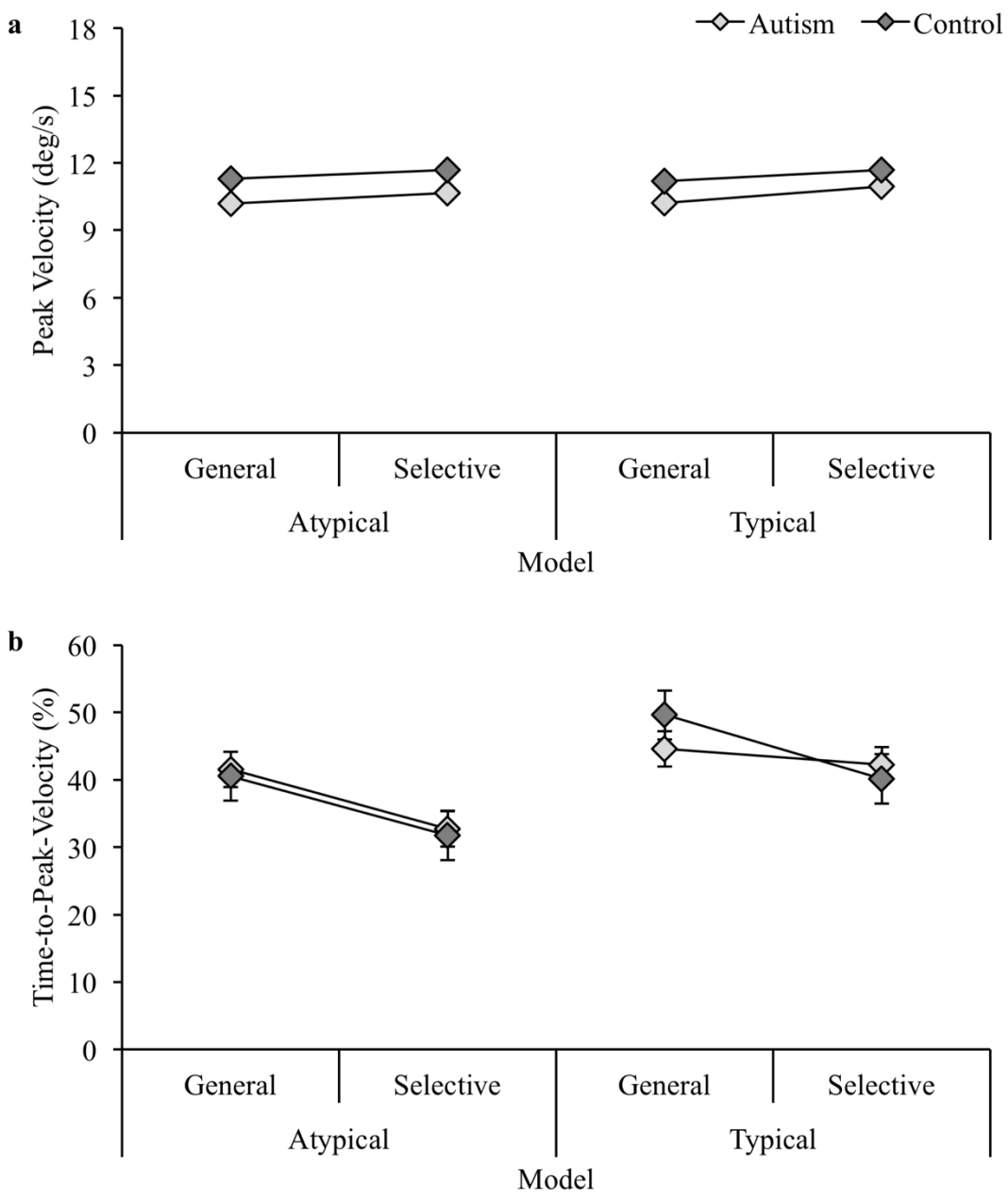

Figure 3.8 (a) Peak velocity and **(b)** time-to-peak-velocity for the eye movements (error bars represent standard error of the mean) presented as a function of group, model and instruction.

peak velocity occurred significantly earlier when observing *atypical* (M = 36 %; SD = 12 %), compared to the *typical* (M = 43 %; SD = 12 %) velocity models ($p <$ 0.001). There was an instruction main effect [F(1, 33) = 13.917, $p = 0.001$, $\eta_p^2 =$ 0.317] that indicated peak eye velocity occurred significantly later by 18 % (8 units) in the general-attention compared to selective-attention conditions (*ps* = 0.001) (**Figure 3.8b**). There were no interactions involving group x model [F(1, 33) = 1.454, $p = 0.237$, $\eta_p^2 = 0.046$], group x instruction [F(1, 33) = 0.929, $p > 0.343$, $\eta_p^2 =$ 0.030], or group x model x instruction [F(1, 33) = 2.752, $p = 0.108$, $\eta_p^2 = 0.084$].

3.4.2.2 Saccade Data

3.4.2.2.1 Number of Saccades

A main effect [F(1, 33) = 4.538, $p = 0.040$, $\eta_p^2 = 0.128$] of model indicated participants exhibited a greater number of saccades when observing *typical* (M = 4.761 saccades; SD = 2.119 saccades) compared to *atypical* (M = 4.415 saccades; SD = 1.845 saccades) velocity models (**Figure 3.9a**).

3.4.2.2.2 Saccade Amplitude

A main effect of model for saccade amplitude [F(1, 33) = 5.190, $p = 0.030$, $\eta_p^2 = 0.143$], which indicated participants generally exhibited larger saccade amplitudes when observing *atypical* (M = 1.755 deg; SD = 0.785 deg) compared to *typical* (M = 1.552 deg; SD = 0.705 deg) velocity models (**Figure 3.9b**). An instruction main effect [F(1, 33) = 6.022, $p = 0.020$, $\eta_p^2 = 0.163$] indicated an 18 % (0.277 deg) larger saccade amplitude in the selective-attention compared to general-

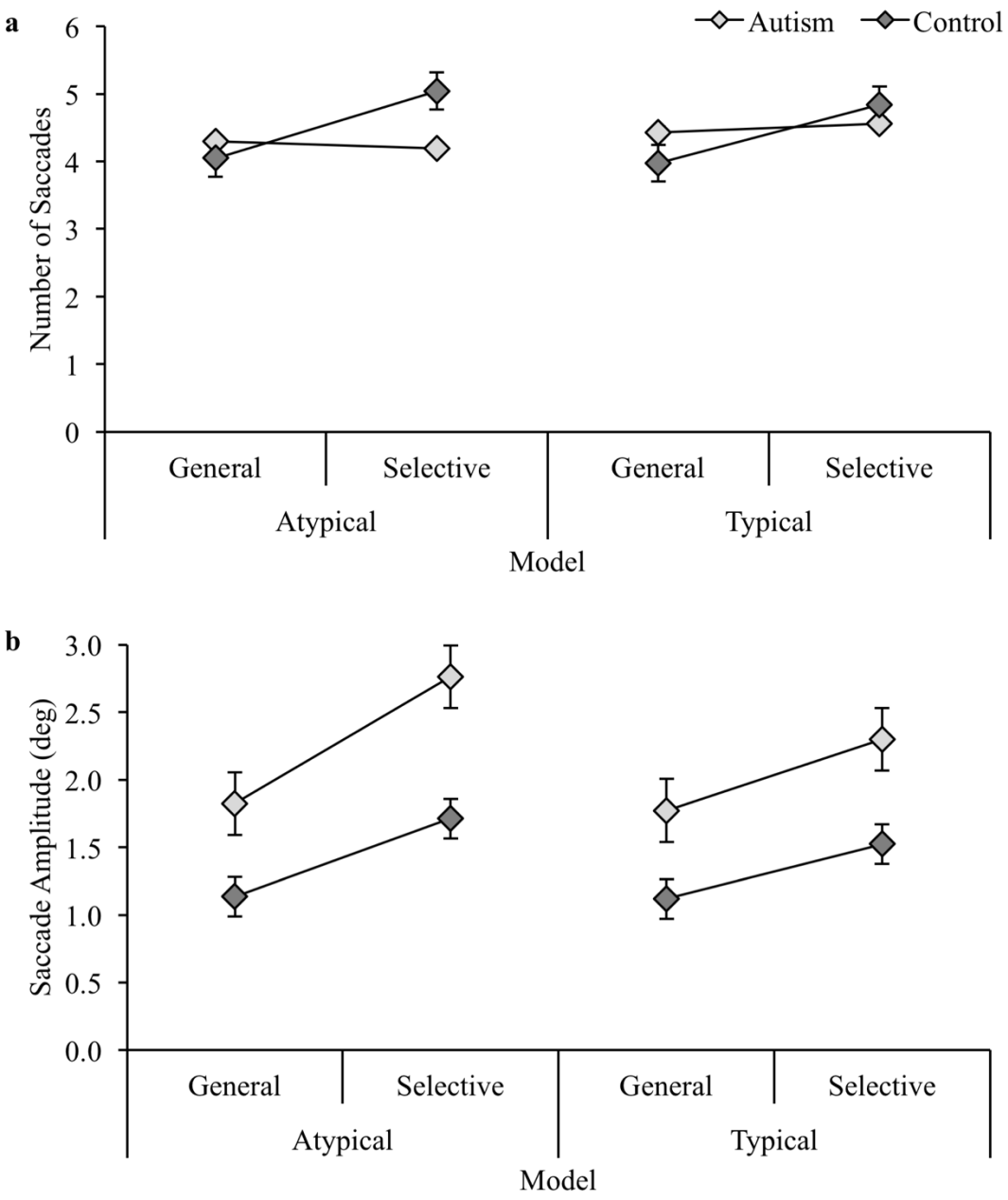

Figure 3.9 (a) Number of saccades and **(b)** saccade amplitude for the eye movements (error bars represent standard error of the mean) presented as a function of group, model and instruction.

Table 3.3 Mean (standard deviation) smooth and saccade data for the eye movements presented as a function of group, model and instruction.

		Autism		Control	
Smooth Data		**General**	**Selective**	**General**	**Selective**
Peak Velocity (deg/s)	**Atypical**	10.36 (2.26)	12.96 (5.08)	11.08 (2.13)	11.48 (2.14)
	Typical	10.59 (3.12)	10.97 (2.07)	11.76 (4.43)	11.55 (2.11)
Time-to-Peak-Velocity (%)	**Atypical**	40 (12)	33 (13)	38 (9)	32 (8)
	Typical	46 (14)	36 (12)	50 (11)	42 (11)
Saccade Data		**General**	**Selective**	**General**	**Selective**
Number of Saccades	**Atypical**	4.31 (1.69)	4.68 (1.74)	4.07 (1.54)	4.61 (1.46)
	Typical	5.02 (2.42)	5.33 (2.40)	4.10 (1.42)	4.59 (1.54)
Saccade Amplitude (deg)	**Atypical**	1.65 (1.12)	2.23 (1.33)	1.51 (0.69)	1.63 (0.62)
	Typical	1.55 (1.02)	1.84 (0.89)	1.35 (0.55)	1.47 (0.52)

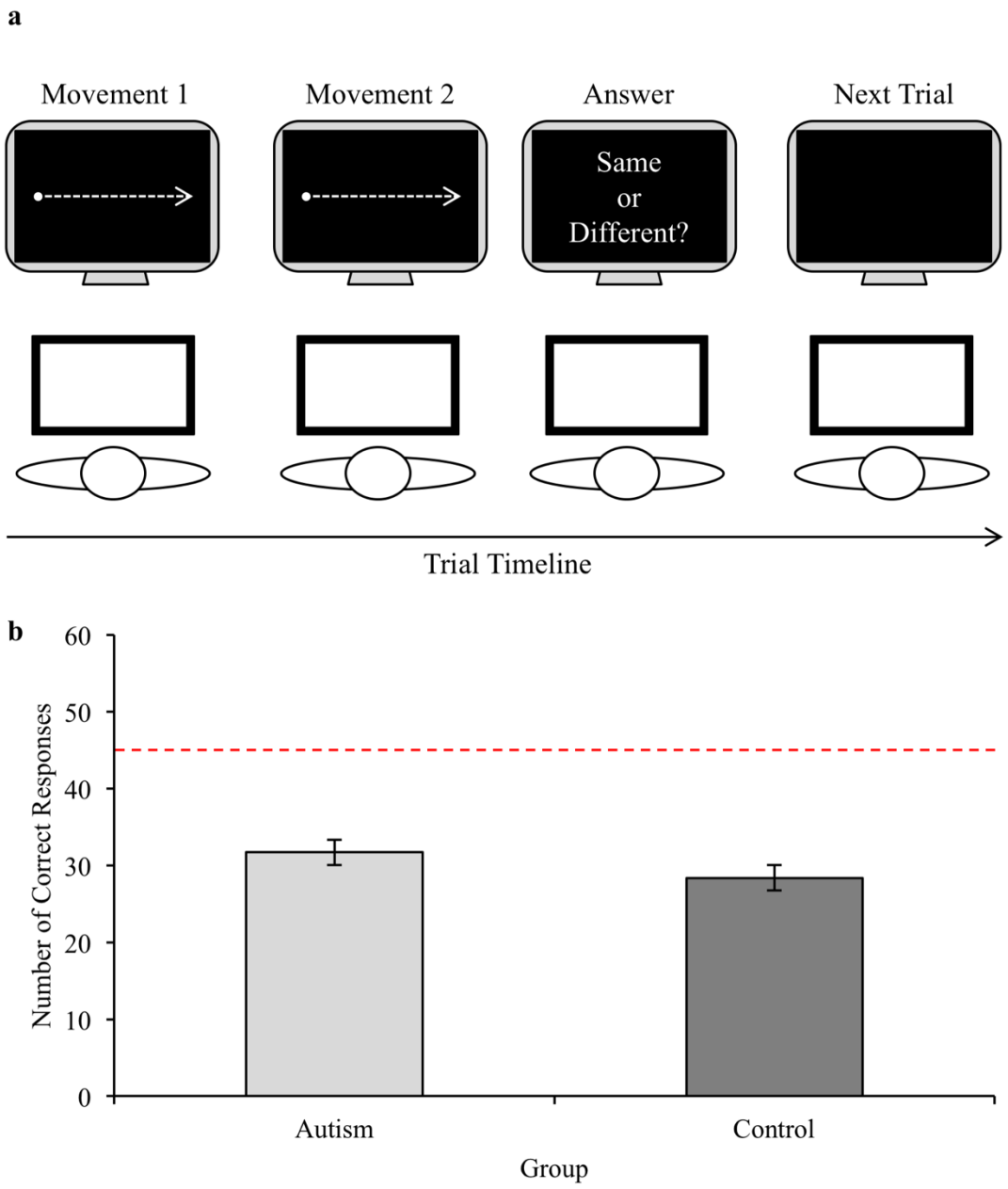

Figure 3.10 (a) A schematic representation of the laboratory/experimental set-up for the judgment task. The white circle displayed on the CRT monitor represents the model. The single-segment movement is depicted by the arrow. **(b)** Number of correct responses for the judgement task (error bars represent standard error of the mean) presented as a function of group. The dashed-red line represents the maximum possible number of correct responses.

attention condition (**Figure 3.9b**). For a full breakdown of each dependent variable see **Table 3.3**.

3.4.3 Judgement Task

From a possible total of 45 correct responses, the autism group made 30 (SD = 9) correct responses and the control group made 27 (SD = 7) correct responses. Both groups were equally successful [t(28) = 1.051, p = 0.300] at judging whether model n was the same or different to model n+1 (autism: M = 66 %; SD = 19 %, control: M = 60 %; SD = 16 %) (**Figure 3.10b**). Using single-sample t-tests with a critical value set at 50 % (i.e., 22.5 correct response), the results showed recognition performance for both groups was significantly greater than chance (autism: t(19) = 3.585, p = 0.002, control: t(19) = 3.002, p = 0.007).

3.5 Discussion

The influence of instructions on selective-attention, inferred from eye movements during action-observation, and imitation behaviour in autism was examined using a behavioural protocol that presented a non-human agent model displaying distinctly different (i.e., *atypical* and *typical*), but biologically plausible kinematics (Hayes et al., 2014). When provided with general-attention instructions, the control group imitated an *atypical* movement such that time-to-peak-velocity occurred at 27 % of the movement trajectory. The early occurrence of peak velocity was similar to the *atypical* model (18 %), but significantly different to the timing of peak velocity imitated (38 %) after observing the *typical* (44 %) model (see **Figure 3.3b**). This finding indicates the control group demonstrated high-fidelity imitation by

representing an *atypical* biological movement that was not part of an existing sensorimotor repertoire (Hayes et al., 2009). This supports previous work showing biological motion (Brass et al., 2001; Press, Gillmeister, & Heyes, 2006), and *atypical* biological kinematics (Hayes et al., 2014; 2016), is processed and represented during imitation. Although the autism group imitated magnitude of peak velocity to a similar level of accuracy as the control group (**Figure 3.3a**), time-to-peak-velocity occurred significantly later in the movement trajectory (*atypical* = 31 %; *typical* = 35 %) irrespective of model observed. These kinematic data indicate the autism group exhibited low-fidelity imitation of *atypical* biological kinematics, which is consistent with *Chapter Two* that used the same imitation protocol in a group of high-functioning adults with autism.

Extending this work in the current study, the examination of eye movements during action-observation indicated both groups exhibited similar peak and time-to-peak smooth pursuit eye velocity (**Figure 3.8**), combined with fewer saccades of greater amplitude, when observing *atypical* compared to *typical* biological motion (**Figure 3.9**). The change in eye movements whilst observing different biological motion kinematics is consistent with visual attention being maintained on the observed model(s). This pattern of pursuit eye movements was similar in both groups, thus providing comparable retinal and extra-retinal input for the configuration of the upper limb motor response required in imitation.

In the second-phase of the study the control group imitated with a time-to-peak-velocity that occurred early in the movement after observing the *atypical* model, and significantly different to the timing of peak velocity imitated after observing the *typical* model. There was no significant change in the accuracy of imitating *atypical* biological kinematics in the autism group following selective-

attention instructions. As before, time-to-peak velocity occurred significantly later in the movement trajectory. The eye movement data replicated the effects from the first phase of the study, but importantly in a context where selective-attention was controlled by instructing groups to explicitly pay attention to, and intend to imitate the trajectory displayed by the model(s). However, even though the eye data indicated the autism group allocated overt visual attention to motion trajectory information, imitation was still attenuated. Furthermore, debriefing data (Questions 1 - 4) provided context as the autism group reported they understood the instruction to pay more attention, and intend, to imitate the trajectory following selective-attention instructions. For example, Participant #5 responded to Question 3 "*Yes I think that you meant to watch the dot more closely, to notice the dot a bit more. I think that I noticed the dot more, like the way that it (the dot) moved and where the dot sped up and slowed down, and I tried to copy it (the dot) the same way*". Moreover, Participant #11 reported their imitation strategy changed after receiving selective-attention instructions (Question 4) "*Yes I think so, I think I was better than the time before, I think I was faster, I think I sped up and slowed down like the fast then slow video that I watched*". Finally, the group reported they could differentiate the two models as Participant #4 responded to Question 1 "*yes, one of the movements was fast and jagged then slowed right down, and the other one (the dot) was kind of like a similar speed all the way through*". This ability to differentiate biological kinematics was confirmed experimentally with the judgement data indicating the autism group accurately, and at a level significantly greater than chance (66 %), perceived differences between *atypical*, *typical*, and *constant* velocity kinematics (**Figure 3.10b**). It is therefore unlikely the impairment in imitating biological kinematics is based at a perceptual level given the autism group perceived

differences in biological motion; for further evidence of intact perception of biological motion in autism see (Freitag et al., 2008; Saygin et al., 2010). More importantly, the impairment in imitating *atypical* biological motion is not based on differences in eye movements, visual attention to motion trajectory, or the explicit intention to imitate the kinematic trajectory, as these processes were similar to the control group.

The method of using a human volunteer to generate both models was critical because it ensured the kinematics were biological in origin and could be reproduced by the participants. It is also important to acknowledge that by using this non-human agent in the study controlled the influence of social attention (e.g., facial features; eyes; human form) during imitation (Vivanti & Hamilton, 2014). Although this control was important for isolating any modulatory effects to the manipulation of instructions on selective-attention in imitation, it does limit the generality of the results to other forms of imitation that occur in human social settings. Indeed, unlike these effects, imitation of body orientation (Hobson & Hobson, 2007) and goal-directed actions (Vivanti & Dissanayake, 2014) was enhanced in autistic children that allocated more visual attention (e.g., increase number or fixations) to the face of a human model, compared to those that paid less attention to this region. These effects suggest that altered visual attention to important social factors attributable to eye gaze may modulate, or inhibit, processing of relevant biological information from a model (Vivanti et al., 2011). Although the use of a non-human agent limited the influence of social modulation during imitation, the fact that adults with autism orientated visual attention to the non-human model, and imitation was still attenuated, provides evidence that other sensorimotor processes contribute to a reduction in imitation efficacy in autism.

Sensorimotor processing of biological motion also occurs in the inter-trial delay of true imitation (Bandura, 1977; Byrne & Russon, 1998; Heyes, 2013). For example, during the imitation of a novel upper-limb motor skill, sensorimotor regions (inferior parietal, pre-motor cortex, inferior frontal gyrus) are active during: (1) action-observation; (2) motor-preparation; (3) and motor-execution (Buccino et al., 2004). Accordingly, combined sensorimotor activity across several imitation phases plays an integral part in the generation of a sensorimotor representation, and the efficacy of an imitated movement. Here, it is important to note that the *atypical* and *typical* models were presented in a randomised order resulting in a stimulus on trial *n*+1 being unpredictable. This influences planning and execution by limiting the opportunity for consolidation of a representation because sensorimotor information from trial *n* (e.g., *atypical* model) will most likely be different to trial *n*+1 (e.g., *typical* model). In this context, rather than a representation being refined by updating error using expected (e.g., what was imitated on trial *n* (i.e. *atypical*), and information from action-observation on trial *n*+1 (i.e., *atypical*)) and actual (reafferent) sensorimotor consequences from trial *n* (e.g., *atypical*) over similar trial types (Elliott, Helsen, & Chua, 2001; Wolpert et al., 2011), the random order would lead to the representation being repeatedly constructed and reconstructed such that the sensorimotor system receives sensorimotor interference (Shea & Morgan, 1979). It is therefore possible that low-fidelity imitation of *atypical* biological kinematics in autism is associated with difficulties in integrating sensorimotor information across trials that do not promote an opportunity for consolidation, or planning and execution difficulties that arise due to random nature of the presentation order. Although no data is presented to support these specific suggestions, they are consistent with previously reported findings that individuals with autism have

neurological difficulties with sensorimotor integration (Marko, Crocetti, Hulst, Donchin, Shadmehr, & Mostofsky, 2015; Ament, Meja, Buhlman, Erklin, Caffo, Mostofsky, & Wodka, 2015; Nebel et al., 2015) and execution (Hughes, 1996; Rinehart et al., 2006; Nazarali et al., 2009; Dowd et al., 2012).

3.6 Summary

In conclusion, it has been shown that adults with autism have specific difficulties imitating the velocity characteristics associated with *atypical* biological kinematics. However, the autism group had similar smooth pursuit and saccadic eye movements during action-observation as the control group. Moreover, the autism group modified pursuit eye movements in a similar manner as the control group following selective-attention instructions. It is therefore unlikely that impaired imitation of *atypical* biological motion kinematics is related to poor tracking of the model trajectory, and thereby the focus of overt attention. Importantly, although eye movement behaviour changed following selective-attention instructions in both groups, imitation behaviour only changed in the control group. This suggests that altered imitation could be associated with differences in 'input' modulation, where lower-level sensorimotor processes do not effectively encode biological motion, or integrate sensorimotor information across trials during true imitation. The latter suggestion will be examined further in the following chapter.

4 High-Fidelity Imitation of Biological Kinematics in Autism is Associated with Sensorimotor Integration

4.1 Abstract

Adults with autism often show impairments in imitating biological motion which has been associated with differences in integrating and consolidating sensorimotor information. Here imitation of *atypical* biological kinematics was examined by manipulating practice that facilitates and attenuates integration of sensorimotor information. To reduce the influence of top-down factors on imitation a non-human agent model was used to control social attention, and end-state target goals were removed in to minimise goal-directed attention. In a Fixed Experiment adults with autism imitated *atypical* biological kinematics to the same extent as matched neurotypicals when the to-be-imitated models were presented in a known structure, which facilitates greater integration and consolidation of sensorimotor information. In an Interference Experiment where participants were required to complete a secondary motor task during the inter-trial delay, adults with autism exhibited difficulties imitating the *atypical* biological kinematics. The implication is that adults with autism were influenced by disruption to greater sensorimotor integration occurring offline during the inter-trial delay. Similar to *Chapter Two* when the presentation structure was randomised adults with autism exhibited differences in imitation of *atypical* biological kinematics. For the first time experimentally it has been demonstrated that adults with autism can imitate biological motion, and that previously reported impairments are associated with processes that integrate and consolidate sensorimotor information.

4.2 Introduction

To acquire gestures and actions that are not present within their motor repertoire the observer must imitate (i.e., copy) the novel action exemplified by another. This process is 'true' imitation since the observer is required to imitate the observed action following observation rather than reproducing the action before observation (Byrne & Russon, 1998). Along with acquiring novel sensorimotor behaviours (Hayes et al., 2007) imitation underpins the development of social-cognition (Rogers et al., 2010) such as feelings of interpersonal closeness (i.e., the desire to be like others) and rapport (Chartrand & Bargh, 1999; Lakin & Chartrand, 2003). A neurodevelopmental disorder which is primary categorised by atypicalities in social-cognition, verbal and non-verbal commutation, as well as a restricted repertoire of interest and activities is autism (American Psychiatric Association, 2013). Given the intricate link between imitation and social-cognition, imitation abilities in those with autism have been examined at length (Rogers & Pennington, 1991; Rogers & Williams, 2006; Hamilton, 2013; Edwards, 2014; Vivanti & Hamilton, 2014) and have consistently showed that individuals with autism have difficulties imitating the actions of others (Rogers & Pennington, 1991; Smith & Bryson, 1994; Rogers et al., 1996; Hobson & Lee, 1999; Stewart et al., 2013) which were reviewed elsewhere (Hamilton, 2013; Edwards, 2014; Vivanti & Hamilton, 2014) as well as the introductory chapter of this thesis (*Chapter One*). For instance, in a study by Wild et al. (2012) adults with and without autism were required to observe and imitate a human model perform a series of hand actions that were differentiated by amplitude (short; long) and duration (fast; slow). Behavioural data showed that there were no

differences when imitating the amplitude of the hand, but only adults without autism successfully imitated the hand speeds.

An essential aspect to the previous chapters and other work demonstrating differences in representing biological motion in autism (Wild et al., 2012; Stewart et al., 2013) is that the to-be imitated stimulus was presented in a random structure, thus causing the stimulus on trial *n*+1 to be ambiguous. This particular presentation structure influences planning and execution, both of which have previously been reported to be difficulties in autism (Hughes, 1996; Rinehart et al., 2001; Mari et al., 2003; Glazebrook et al., 2006, 2008). Moreover, it may limit the opportunity to integrate and consolidate the sensorimotor representation and as a result, it could be posited that low-fidelity imitation of *atypical* biological motion in autism is attributed to complications in integrating sensorimotor information on a trial-by-trial basis where there is opportunity for consolidation, or the previously reported problems in planning and execution occur due to the unknown presentation structure.

4.3 Fixed Experiment

The aim of the initial experiment in this chapter was to further examine imitation of *atypical* biological kinematics in adults with autism, where the to-be-imitated models will be presented in a fixed order, which provides opportunity for the sensorimotor representation to be refined by updating error using expected (e.g., imitation from *atypical* model on trial *n* and information from observation of *atypical* model on trial *n*+1) and actual sensory consequences from trial *n* over similar trial types (Elliott et al., 2001; Wolpert et al., 2011). If the structure of how the to-be-imitated models were presented influences imitation by providing an increased opportunity for

sensorimotor integration and consolidation across repeated trials of the same model, then it can be predicted that adults with autism would imitate the *atypical* biological kinematics more similarly compared to adults without autism. If, however, presentation structure does not influence imitation, then it should follow that consistent with *Chapters Two* and *Three* where the presentation structure is randomised, individuals with autism will exhibit differences in imitation of *atypical* velocity model. In this situation, the control group will exhibit movements with a time-to-peak-velocity that will occur earlier in the movement, similar to that displayed by the *atypical* model and significantly different to the time-to-peak-velocity exhibited after observing velocity control models. Contrary the autism group would show a time-to-peak-velocity which was significantly different from the control group, but statistically similar to the time-to-peak-velocity exhibited when imitating the *typical* velocity model.

4.4 Method

4.4.1 Participants

Fifteen typical control participants (12 male; 3 female) and 15 participants with autism (12 male; 3 female) volunteered for the study. The volunteers with autism were recruited from an autistic society in North West of England, and Liverpool John Moores University, UK. The volunteers were provided with a participant information sheet and selected if they consented to be part of the study. The control participants were recruited from Liverpool John Moores University, UK. None of the volunteers participated in *Chapters Two* and *Three* and thus were naïve to the experiment. Sample characteristics are presented in **Table 4.1**.

Table 4.1 Participant characteristics of the autism and control groups in the Fixed experiment.

	Autism (n= 15)		**Control (n = 15)**		
	Mean (SD)	**Range**	**Mean (SD)**	**Range**	***P* Value**
Chronological Age	22 (2) years	18 - 26	20 (2) years	18 - 26	0.217
IQ:					
Full Scale	101 (11)	82 - 118	103 (9)	84 - 123	0.574
Verbal	101 (12)	87 - 127	105 (9)	89 - 126	0.366
Performance	101 (13)	79 - 119	101 (11)	82 - 117	0.988
ADOS:					
Total	8 (1)				
Communication	3 (1)				
Social Interaction	5 (1)				
Gender	12 M: 3 F		12 M: 3 F		

4.4.2 Apparatus and Procedure

The apparatus used and the procedure was identical to that in *Chapters Two* and *Three.* Volunteers performed 2 blocks of 40 trials in each phase (80 trials). A block contained only either *typical* or *atypical* biological motion. In order to provide increased sensorimotor integration and consolidation over similar trial types, trial order within a block, as well as block order, was fixed across volunteers.

4.4.3 Data Reduction

Quantifying imitation of timing and kinematic data was identical to *Chapters Two* and *Three*. Intra-participant means were calculated from 40 trials associated with each model.

4.4.4 Data Analysis

Data from all dependent variables were submitted to separate 2 group (autism; control) x 2 model (*atypical; typical*) repeated measures ANOVA.

4.5 Results

4.5.1 Timing Data

4.5.1.1 Timing Error

A main effect [$F(1, 28) = 9.645$, $p = 0.004$, $\eta_p^2 = 0.256$] of model indicated participants timing was significantly more accurate when imitating *atypical* (M = -83 ms; SD = 284 ms) compared to *typical* (M = 121 ms; SD = 309 ms) velocity models (**Figure 4.1a**). Importantly, there was no significant main effect of group [F(1, 28) =

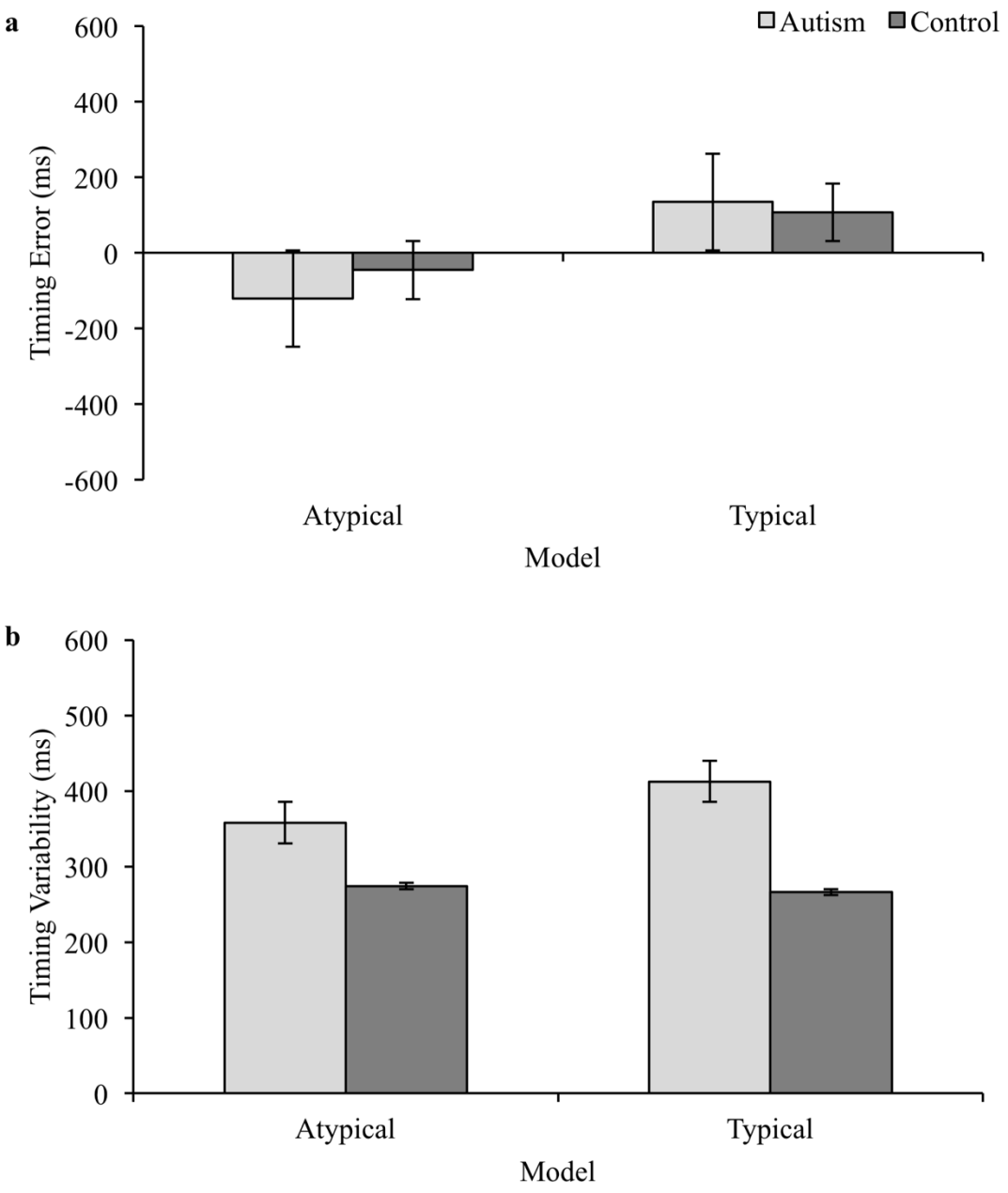

Figure 4.1 (a) Timing error and **(b)** timing variability for the imitation task (error bars represent standard error of the mean) in the Fixed experiment presented as a function of group and model.

0.065, p = 0.800, η_p^2 = 0.002], overall the autism group (M = 7 ms; SD = 310 ms) performed to the same extent as the control group (M = 31 ms; SD = 339 ms).

4.5.1.2 Timing Variability

For timing variability a group main effect [F(1, 28) = 6.835, p = 0.014, η_p^2 = 0.196] indicated timing variability was significantly lower for the control (M = 270 ms; SD = 99 ms) than autism (M = 385 ms; SD = 133 ms) group (**Figure 4.1b**).

4.5.2 Kinematic Data

4.5.2.1 Peak Velocity

A main effect of model [F(1, 28) = 70.616, p < 0.001, η_p^2 = 0.716] for peak velocity indicated magnitude was higher when imitating *atypical* (M = 0.274 mm/ms; SD = 0.046 mm/ms), compared to *typical* velocity models (M = 0.186 mm/ms; SD = 0.033 mm/ms) (**Figure 4.2a**).

4.5.2.2 Time-to-Peak-Velocity

There was a main effect of model [F(1, 28) = 65.117, p < 0.001, η_p^2 = 0.699], where peak velocity occurred significantly earlier when imitating *atypical* (M = 26 %; SD = 8 %), compared to the *typical* biological motion (M = 38 %; SD = 11 %) (**Figure 4.2b**). Importantly there was no group x model interaction [F(1, 28) = 1.839, p = 0.186, η_p^2 = 0.062].

These above effects can be seen in the exemplar velocity traces illustrated in **Figure 4.3**. When imitating the *atypical* velocity model, peak velocity occurred earlier in the movement for both groups (solid-black trace; **Figure 4.3**). When

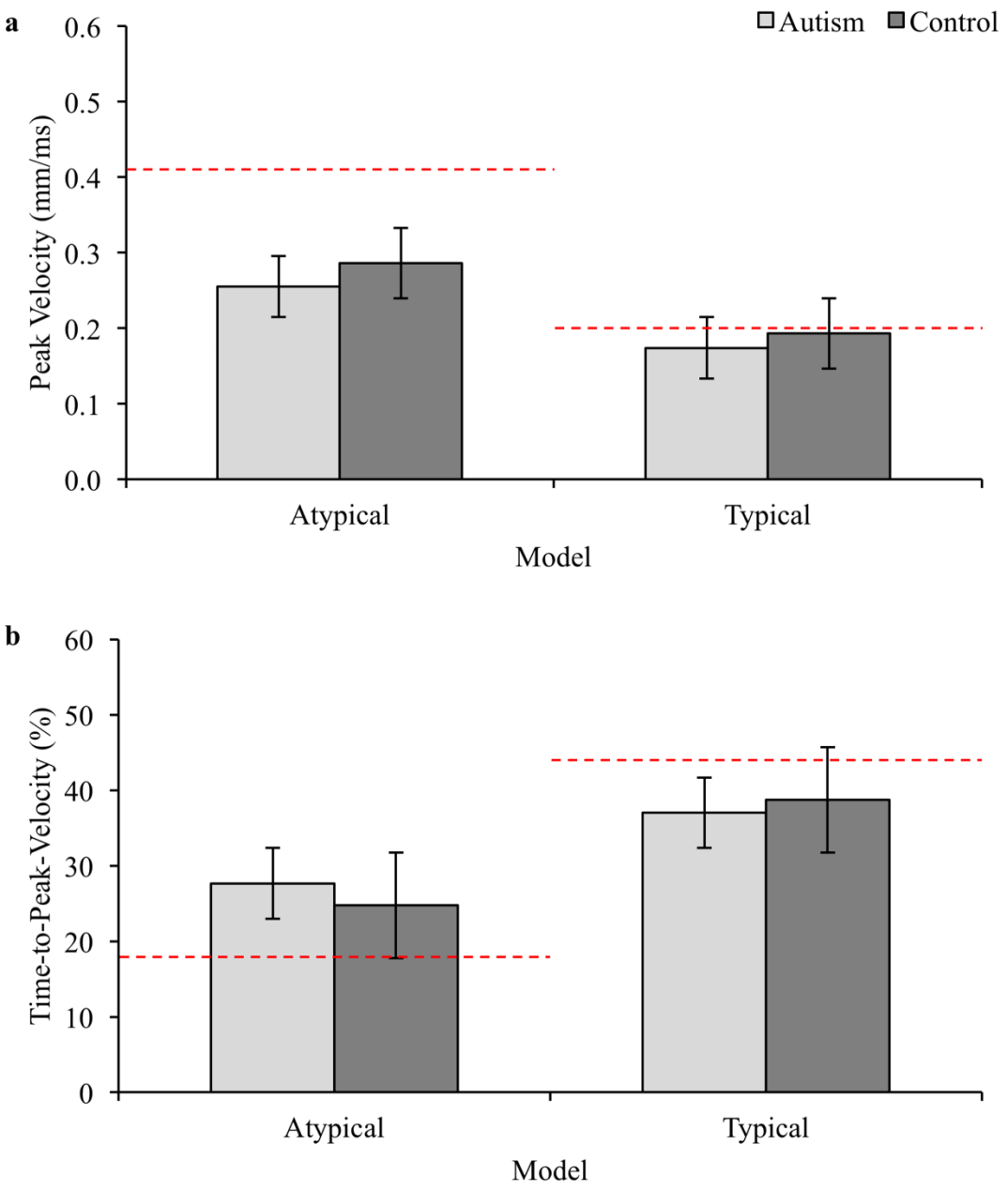

Figure 4.2 (a) Peak velocity and **(b)** time-to-peak-velocity for the imitation task (error bars represent standard error of the mean) in the Fixed experiment presented as a function of group and model. The dashed-red lines in **a** represent the magnitude of peak velocity for the *typical* (i.e., 0.410 mm/ms) and *atypical* (i.e., 0.200 mm/ms) models. In **b**, they represent the time-to-peak-velocity for the *typical* (i.e., 44 %) and *atypical* (i.e., 18 %) models.

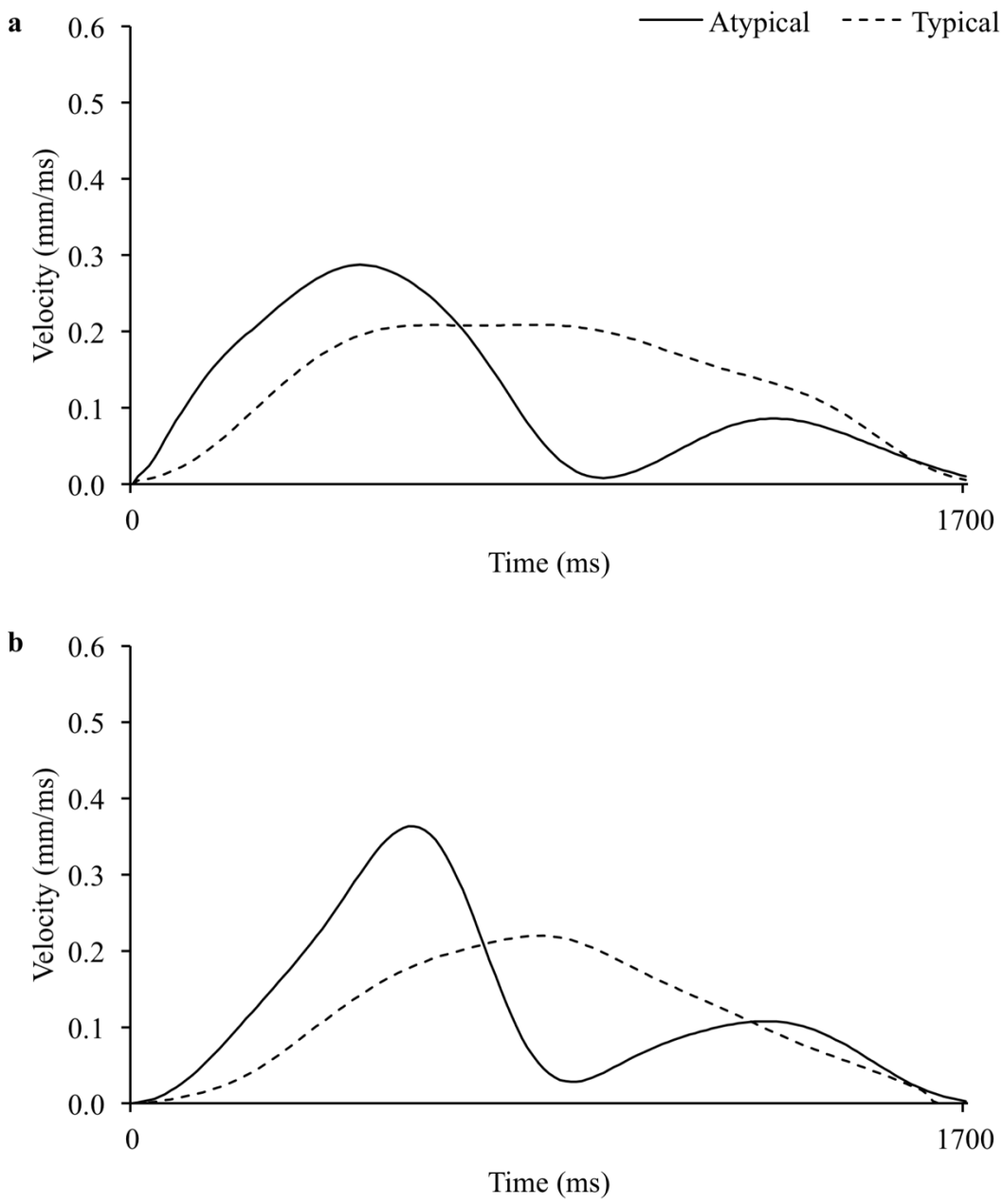

Figure 4.3 Velocity traces displaying exemplar kinematic data for the **(a)** autism and **(b)** control groups during imitation of the *typical* (dashed-black trace) and *atypical* (solid-black trace) velocity models in the Fixed experiment presented as a function of time.

Table 4.2 Mean (standard deviation) timing and kinematic data for the imitation task in the Fixed experiment presented as a function of group and model.

		Autism	Control
Timing Data			
Timing Error (ms)	**Atypical**	-127	-60
	Typical	95	68
Timing Variability (ms)	**Atypical**	358	274
	Typical	413	266
Kinematic Data			
Peak Velocity (mm/ms)	**Atypical**	0.259 (0.056)	0.289 (0.058)
	Typical	0.176 (0.033)	0.196 (0.034)
Time-to-Peak-Velocity (%)	**Atypical**	27 (9)	25 (7)
	Typical	37 (12)	39 (9)

imitating the *typical* velocity model, peak velocity occurred toward the midpoint of the movement for both groups (dashed-black trace; **Figure 4.3**). For a full breakdown of each dependent variable see **Table 4.2**.

4.6 Discussion

Imitation of biological kinematics was examined using a behavioural protocol that required adults with and without autism to observe and subsequently imitate models that displayed distinctly different but biologically plausible kinematics (Hayes et al., 2014). After observing an *atypical* velocity model, participants in the control group exhibited movements with a peak velocity that occurred at 25 % of the movement trajectory. This early occurrence of peak velocity was similar to that displayed by the *atypical* model (time-to-peak-velocity = 18 %), and significantly different to the time-to-peak-velocity exhibited after observing *typical* (M = 39 %) velocity model. Importantly, participants in the autism group also exhibited movements with a peak velocity that occurred at 27 % of the movement trajectory, and thus similar to that displayed by the *atypical* model, as well as being statistically different to that exhibited after observing the *typical* (M = 37 %) model (**Figure 4.2b**). This showed a relationship between the motor output (i.e., imitation) and the observed *atypical* biological motion signifies reasonably high-fidelity imitation of biological motion (Brass et al., 2001; Gangitano et al., 2001). Notably, these findings demonstrate that adults with autism can successfully represent velocity and are inconsistent with work indicating complications in imitating the form (Rogers et al., 1996; Hobson & Lee, 1999) or action speed (Wild et al., 2012; Stewart et al., 2013) in autism. The main difference between the current experiment and previous studies

illustrating problems in imitation of biological kinematics in autism (Wild et al., 2012; Stewart et al., 2013) is the presentation structure of the to-be-imitated model(s). Therefore, these findings are consistent with the proposal that low-fidelity imitation of *atypical* biological kinematics in autism can be attributed to complications in integrating sensorimotor information on a trial-by-trial basis where there is opportunity for consolidation, or the previously reported problems in planning and execution occur due to the unknown presentation structure.

4.7 Interference Experiment

The first experiment demonstrated that adults with autism can successfully imitate *atypical* biological kinematics when provided with the opportunity to integrate and consolidate sensorimotor information using expected and actual sensory signals over similar trial types (Elliott et al., 2001; Wolpert et al., 2011). These adjustments for error occur online (Kilner et al, 2007; Burke et al., 2010) during motor-execution as well as offline (Wolpert et al., 1995, 2011) during the inter-trial delay. However, it is currently unclear where in the imitation process this increased sensorimotor integration is occurring? One way to examine this is by providing a secondary motor task during the inter-trial delay which interferes with offline consolidation of the sensorimotor representation. This interference effect during the consolidation period has previously been reported from an observational learning study (Brown, Wilson, & Gribble, 2009) that induced motor interference using repeated transmagnetic stimulation (rTMS). In this study, participants observed naïve learners perform reaching movements using a robotic arm. The robot was programmed to perturb the upper-limb dynamics by applying force fields to the learner's arm in a clockwise or

counter-clockwise direction. To examine whether integration occurs during the consolidation period repeated transmagnetic stimulation was applied to the primary motor cortex with the expectation that learning would be attenuated. Consistent with previous work (Mattar & Gribble, 2005), reaching performance was facilitated by observational learning, however learning was significantly reduced in participants that received rTMS to the primary motor cortex during the consolidation period. This finding demonstrates sensorimotor information is consolidated in primary motor cortex, which is also known to be active during imitation learning (Nishitani et al., 2004).

With the above mentioned in mind, the aim of the second experiment is to further examine imitation of *atypical* biological motion in adults with autism, and whether the suggested increase in sensorimotor integration and consolidation facilitated by presenting a fixed structure occurs offline, during the inter-trial delay (i.e., in-between motor-execution on trial *n* and action-observation on trial *n*+1). Adults with and without autism observed and imitated biological kinematic models in a known structure that were undistinguishable from the Fixed Experiment however during the inter-trial delay they were required to create circular motions using the stylus on the digital graphics tablet. If the increase in sensorimotor integration and consolidation resulting in increased imitation fidelity of *atypical* biological kinematics occurs offline during the inter-trial delay, then it can be expected that the secondary motor task will cause an interference effect, whereby adults with autism will demonstrate differences in imitation of *atypical* biological kinematics, similar to those observed in *Chapters Two* (**Figure 2.3b**) and *Three* (**Figure 3.3b**) where the trial order was randomised. If the increase in sensorimotor integration and consolidation does not occur offline and rather may occur during

action-observation or motor-execution on trial *n*, then it can be expected that adults with autism will imitate the *atypical* velocity model to the same extent as control participants, exhibiting kinematics similar to those in the Fixed experiment (**Figure 4.2b**).

4.8 Method

4.8.1 Participants

Fifteen typical control participants (13 male; 2 female) and 15 participants with autism (13 male; 3 female) volunteered for the study. Nine participants with autism (8 male; 1 female), and nine typical control participants (8 male; 1 female), were recruited from the Fixed experiment (in order to reduce any retention effects associated with imitation learning, participants completed the Interference experiment three months following the Fixed experiment). Six new autism (4 male; 2 female) and six new typical control (4 male; 2 female) participants were recruited. The volunteers were provided with a participant information sheet and selected if they consented to be part of the study. The control participants were recruited from Liverpool John Moores University, UK. Sample characteristics are presented in **Table 4.3**.

4.8.2 Apparatus, Procedure and Data Reduction

The apparatus, procedure and data reduction were identical to that in the Fixed Experiment except all volunteers completed a secondary motor task offline during the inter-trial delay. Following motor-execution on trial *n* and before action-observation on trial *n*+1, a circular 'track' (i.e., small circle of diameter 15.78 cm

Table 4.3 Participant characteristics of the autism and control groups in the Interference experiment.

	Autism (n= 15)		**Control (n = 15)**		
	Mean (SD)	**Range**	**Mean (SD)**	**Range**	***P* Value**
Chronological Age	21 (2) years	18 - 25	20 (2) years	18 - 24	0.136
IQ:					
Full Scale	103 (14)	82 - 126	105 (11)	92 - 124	0.486
Verbal	102 (15)	84 - 130	109 (11)	95 - 130	0.298
Performance	102 (14)	79 - 121	102 (8)	89 - 115	0.910
ADOS:					
Total	8 (1)				
Communication	3 (1)				
Social Interaction	5 (1)				
Gender	13 M: 2 F		12 M: 2 F		

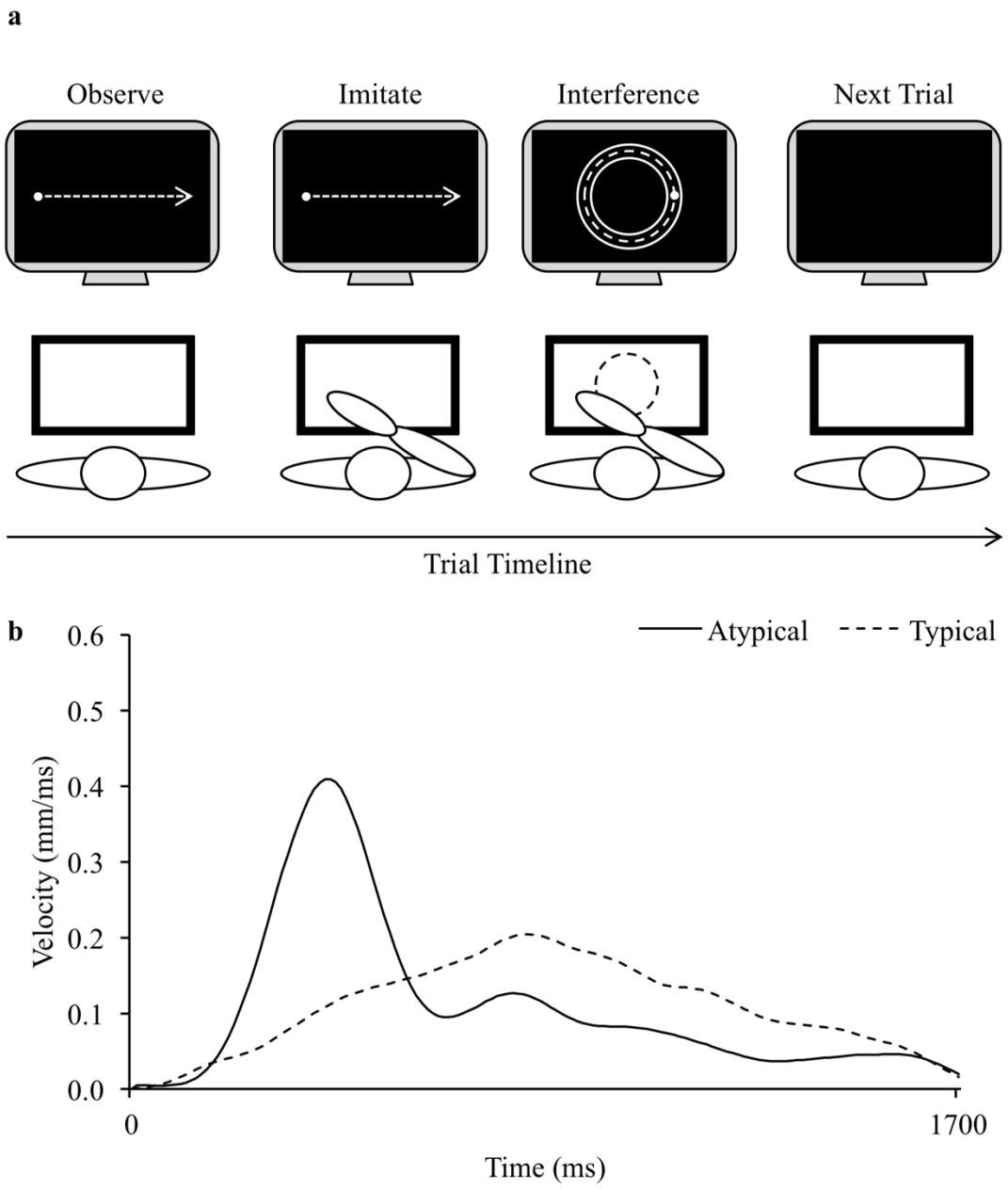

Figure 4.4 (a) A schematic representation of the laboratory/experimental set-up for the imitation task in the Interference experiment. The black outlined rectangle represents a graphics tablet. The white circle displayed on the CRT monitor represents the model. The single-segment movement is depicted by the arrow. The secondary task is depicted by the dashed-line. **(b)** *Typical* (dashed-black trace) and *atypical* (solid-black trace) velocity models presented as a function of time.

inside a large circle of diameter = 18.93 cm) appeared on the monitor along with a white cursor (diameter = 6.25 mm) that represented the position of the stylus (**Figure 4.4a**). Participants were instructed to move the stylus on the tablet so that the cursor moved from a start/finish position located on the right-hand side of the circle. Having clicked the lower-button on the stylus, participants moved the white cursor around the track in a clockwise direction, as many times as possible in 4000 ms. Participants were instructed to avoid moving outside of the circuit (they were not penalised if they did).

4.8.3 Data Analysis

Data analysis were identical to that in the Fixed experiment except to account for accrued prior experience in the participants that completed the Fixed experiment, a co-variate (i.e., experience) was included in the repeated measures ANOVA. This analyses revealed no significant influence of experience (*ps* > 0.050).

4.9 Results

4.9.1 Timing Data

4.9.1.1 Timing Error

A main effect [F(1, 28) = 6.966, $p = 0.014$, $\eta_p^2 = 0.199$] of model indicated participants timing was significantly more accurate when imitating *atypical* (M = -35 ms; SD = 250 ms) compared to *typical* (M = 153 ms; SD = 272 ms) velocity model (**Figure 4.5a**). Importantly, there was no significant main effect of group [F(1, 28) =

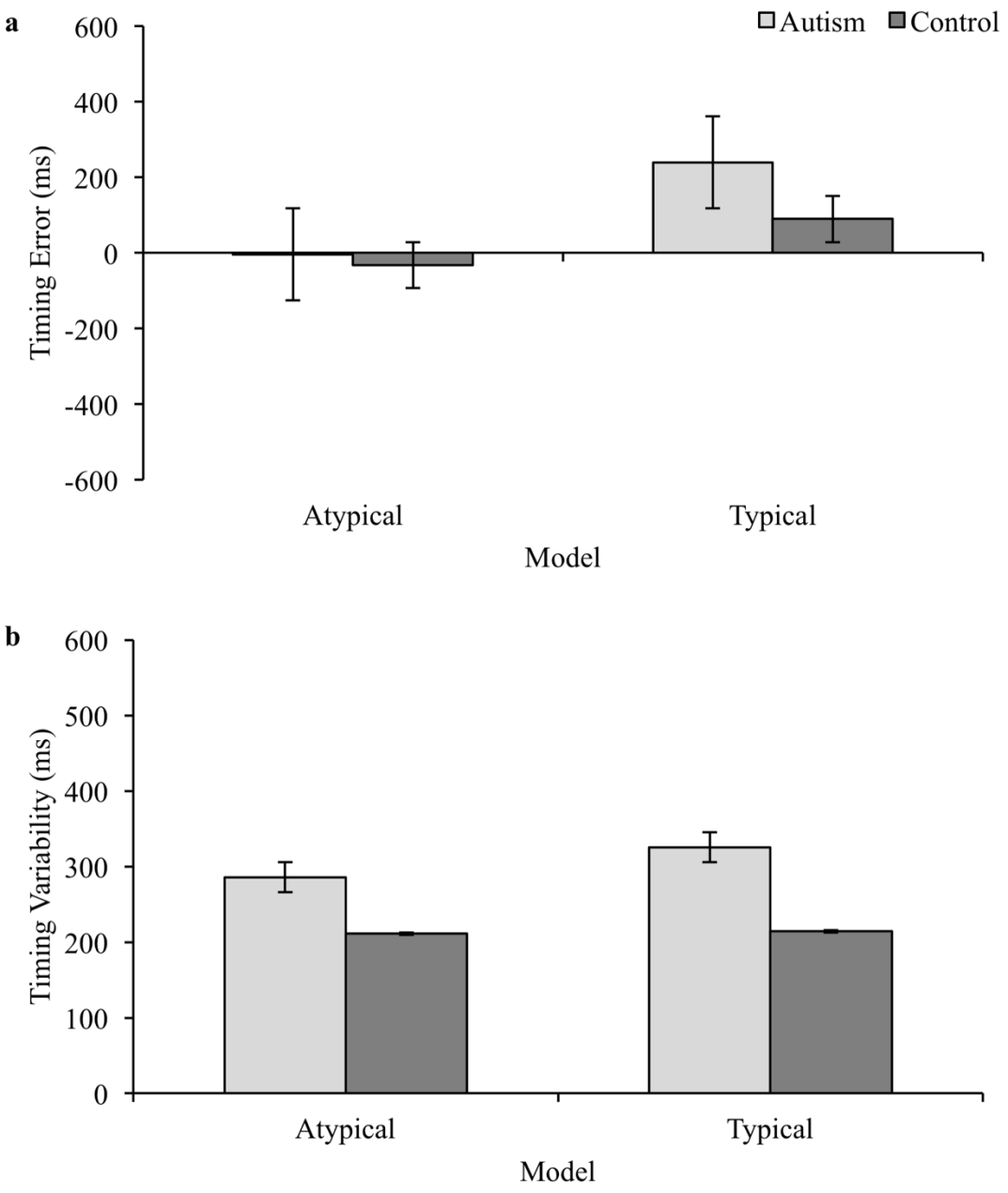

Figure 4.5 (a) Timing error and **(b)** timing variability for the imitation task (error bars represent standard error of the mean) in the Interference experiment presented as a function of group and model.

0.370, $p = 0.548$, $\eta_p^2 = 0.013$], overall the autism group (M = 118 ms; SD = 701 ms) performed to the same extent as the control group (M = 0 ms; SD = 377 ms).

4.9.1.1 Timing Variability

A group main effect [F(1, 28) = 5.627, $p = 0.024$, $\eta_p^2 = 0.167$] indicated timing variability was significantly lower for the control (M = 211 ms; SD = 61 ms) than autism (M = 309 ms; SD = 181 ms) group (**Figure 4.5b**).

4.9.2 Kinematic Data

4.9.2.1 Peak Velocity

A main effect of model [F(1, 28) = 31.586, $p < 0.001$, $\eta_p^2 = 0.530$] for peak velocity indicated magnitude was higher when imitating *atypical* (M = 0.269 mm/ms; SD = 0.043 mm/ms), compared to *typical* velocity model (M = 0.189 mm/ms; SD = 0.030 mm/ms) (**Figure 4.6a**).

4.9.2.2 Time-to-Peak-Velocity

There was a main effect of model [F(1, 28) = 24.668, $p < 0.001$, $\eta_p^2 = 0.468$], where peak velocity occurred significantly earlier when imitating *atypical* (M = 31 %; SD = 7 %), compared to the *typical* velocity models (M = 42 %; SD = 10 %) (**Figure 4.6b**). This was superseded by a model x group interaction [F(1, 38) = 7.969, $p = 0.009$, $\eta_p^2 = 0.222$], which indicated peak velocity occurred significantly earlier when imitating *atypical* velocity model (**Figure 4.6b**) for the control (M = 28 %; SD = 5%) than autism group (M = 35 %; SD = 9 %). The early occurrence of peak velocity in the control group was more reflective of *atypical* velocity

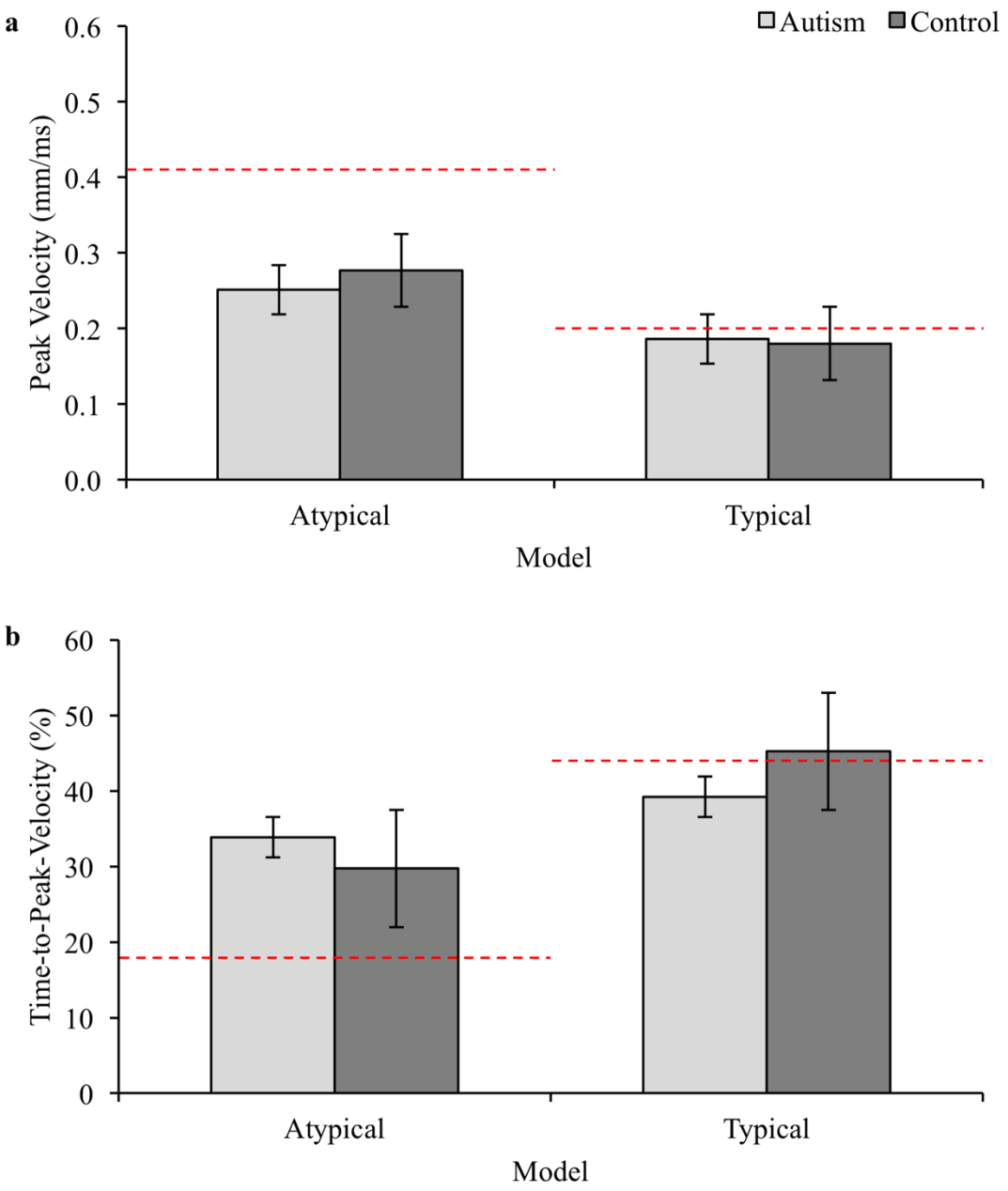

Figure 4.6 (a) Peak velocity and **(b)** time-to-peak-velocity for the imitation task (error bars represent standard error of the mean) in the Interference experiment presented as a function of group and model. The dashed-red lines in **a** represent the magnitude of peak velocity for the *typical* (i.e., 0.410 mm/ms) and *atypical* (i.e., 0.200 mm/ms) models. In **b**, they represent the time-to-peak-velocity for the *typical* (i.e., 44 %) and *atypical* (i.e., 18 %) models.

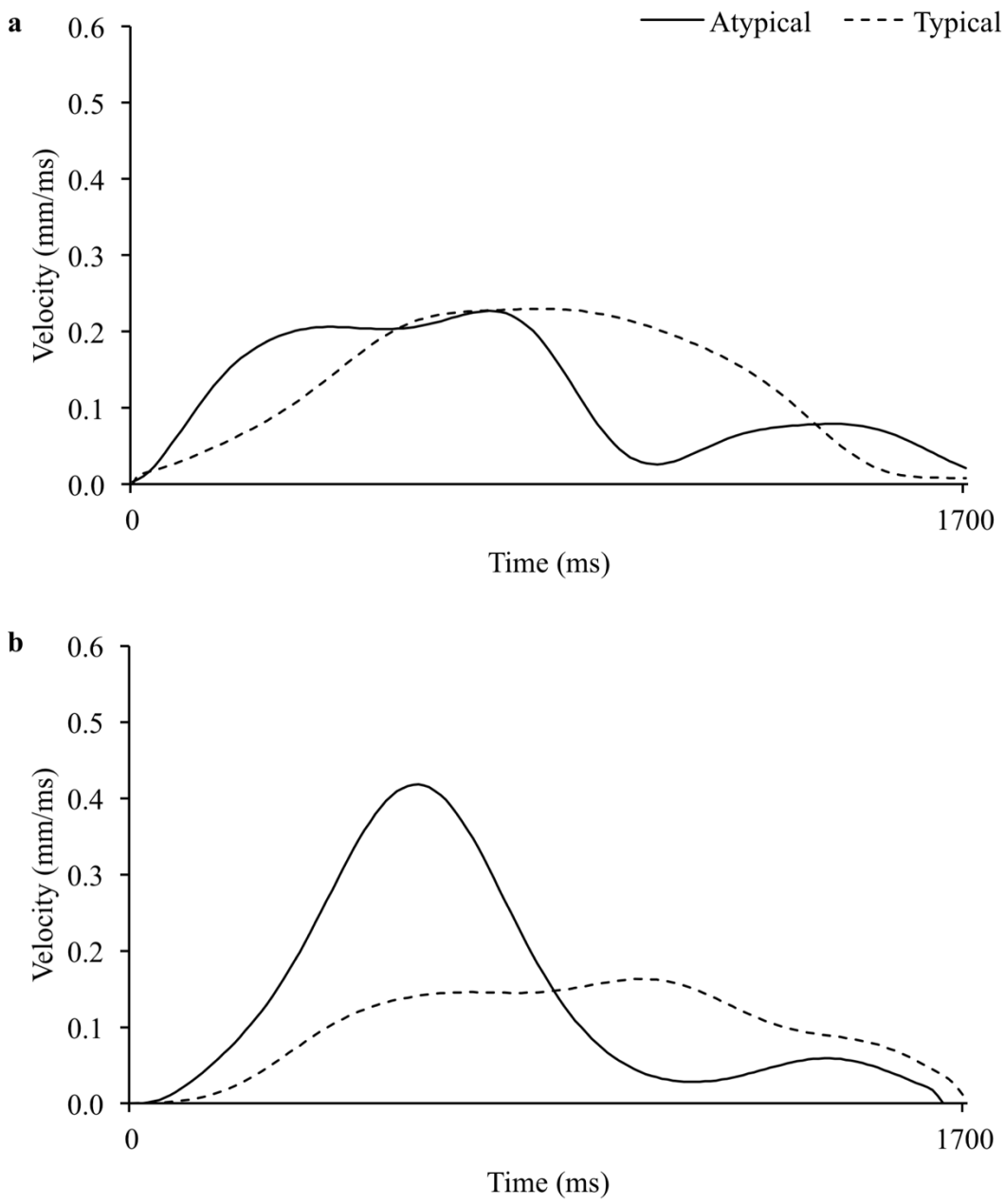

Figure 4.7 Velocity traces displaying exemplar kinematic data for the **(a)** autism and **(b)** control groups during imitation of the *typical* (dashed-black trace), *atypical* (solid-black trace) velocity models in the Interference experiment presented as a function of time.

Table 4.4 Mean (standard deviation) timing and kinematic data for the imitation task in the Interference experiment presented as a function of group and model.

		Autism	Control
Timing Data			
Timing Error (ms)	**Atypical**	10	-71
	Typical	206	47
Timing Variability (ms)	**Atypical**	286	207
	Typical	326	216
Kinematic Data			
Peak Velocity (mm/ms)	**Atypical**	0.251 (0.046)	0.286 (0.040)
	Typical	0.193 (0.033)	0.185 (0.027)
Time-to-Peak-Velocity (%)	**Atypical**	35 (9)	28 (5)
	Typical	39 (10)	45 (10)

model (18 %; dashed-red line), than the *typical* velocity model (44 %; dashed-red line). There was no significant difference ($p > 0.050$) between the groups when imitating *typical* velocity model (autism: M = 39 %; SD = 10 %, control: M = 45 %; SD = 10 %). Correlation analysis revealed no relationship between time-to-peak-velocity when imitating the *atypical* velocity model and ADOS total score (Pearson's r(15) = -0.112, $p = 0.690$) or *typical* velocity model and ADOS total score (Pearson's r(15) = -0.310, $p = 0.261$).

These above effects can be seen in the exemplar velocity traces illustrated in **Figure 4.7**. When imitating the *atypical* velocity model, peak velocity occurred significantly earlier in the movement for the control group (**Figure 4.7b**) than the autism group (**Figure 4.7a**). When imitating the *typical* velocity model, peak velocity occurred toward the midpoint of the movement for both groups (dashed-black trace; **Figure 4.7**). For a full breakdown of each dependent variable see **Table 4.4**.

4.10 Discussion

To further examine the suggestion that the increase in imitation fidelity of *atypical* biological kinematics observed in the Fixed experiment was facilitated by integrating sensorimotor information during the inter-trial delay, a secondary motor task (Mattar & Gribble, 2005; Brown et al., 2009) was included during the inter-trial delay. The secondary visuomotor task was implemented to experimentally interfere with the processing of sensorimotor information from trial *n*. As expected, and in line with the findings from *Chapters Two* (autism: M = 33 %; control: M = 24 %) and *Three* (autism: M = 31 %, control: M = 27 %) the control group were significantly more

accurate (M = 28 %) than participants in the autism (M = 35 %) group at imitating *atypical* biological motion (18 %). In effect, imitation performance in the autism group when imitating the *atypical* model was statistically similar to imitating the *typical* model (M = 39 %). Moreover, imitation performance was qualitatively different to the autism group from the Fixed experiment (M = 27 %) where the opportunity for sensorimotor consolidation during the inter-trial delay was not perturbed (Bandura, 1977; Byrne & Russon, 1998; Wolpert et al., 2011).

4.11 Random Experiment

One of the most important findings from the Fixed experiment is that through presenting the to-be-imitated stimulus in a fixed order, adults with autism imitated biological motion kinematics to a similar level as matched neurotypical controls (**Figure 4.2b**). Through repeated attempts at imitating the *atypical* model in a fixed condition it is likely the refinement of the sensorimotor representation was facilitated by increasing the opportunity across similar trial types to process error through comparisons between the expected and actuals sensory signals during the inter-trial delay. The suggestion that imitation fidelity was facilitated by integrating sensorimotor information during the inter-trial delay was supported by the findings from the Interference experiment (**Figure 4.6b**) where participants performed a secondary motor task in the inter-trial delay. The secondary visuomotor task was implemented to experimentally interfere with the processing of sensorimotor information from trial *n*. As expected, imitation performance in the autism group was statistically similar to imitating the *typical* model and importantly significantly different to the neurotypical control group.

Considering the aforementioned it could be suggested that previous difficulties in imitation of kinematics in autism may be attributed to the random structure which does not allow the opportunity for integration and consolidation of sensorimotor information on a trial-by-trial basis. Instead during the random condition different sensorimotor representations (e.g., Task 1, followed by Task 2) are required to be constructed, deconstructed, and reconstructed across trials leading to an increase in sensorimotor interference within the inter trial delay during the learning process (Shea & Morgan, 1979). In *Chapter Two* adults that were naïve to the protocol showed poor imitation of *atypical* biological motion. Therefore, the aim of the third study it to expand upon this finding and confirm the suggestion that imitation impairments are associated with processes that integrate and consolidate sensorimotor information adults with and without autism that were familiar to the protocol (i.e., they took part in the Fixed and Interference experiment) took part. The to-be-imitated model(s) were the same as previous, yet was be presented in a randomised structure, where the information from trial *n* (e.g., *atypical* biological motion) is expected to be different to trial *n*+1 (e.g., *typical* biological motion).

It can be predicted that if the previous difficulties in imitation of kinematics in autism may be attributed to the random structure, then adults with autism would show low-fidelity imitation of *atypical* velocity model, exhibiting kinematics significantly different to the control group, yet similar to those reported in *Chapters Two*, *Three* and the Interference experiment in this chapter. On the contrary, if impaired imitation is not associated with the random structure, then it can be expected that adults with autism will who high-fidelity imitation of *atypical* velocity model, exhibiting kinematics similar to the control group and those reported in the Fixed experiment of this chapter.

4.12 Method

4.12.1 Participants

Volunteers were identical to that in the Interference experiment (in order to reduce any retention effects associated with imitation learning, participants completed the Random experiment three months following the Interference experiment). Sample characteristics are presented in **Table 4.5**.

4.12.2 Apparatus, Procedure, Data Reduction and Data Analysis

The apparatus, procedure and data reduction and data analysis were identical to that in the Fixed experiment except trial order within a block, as well as block order, was randomised across volunteers.

4.13 Results

4.13.1 Timing Data

4.13.1.1 Timing Error

The ANOVA did not reveal any significant main effects of group or model or any interactions for timing error (*ps* > 0.050) (**Figure 4.8a**).

4.13.1.2 Timing Variability

A group main effect [F(1, 28) = 6.248, $p = 0.019$, $\eta_p^2 = 0.182$] indicated timing variability was significantly lower for the control (M = 234 ms; SD = 89 ms) than autism (M = 322 ms; SD = 152 ms) group (**Figure 4.8b**).

Table 4.5 Participant characteristics of the autism and control groups in the Random experiment.

	Autism (n= 15)		**Control (n = 15)**		
	Mean (SD)	**Range**	**Mean (SD)**	**Range**	***P* Value**
Chronological Age	22 (2) years	18 - 26	21 (2) years	18 - 27	0.068
IQ:					
Full Scale	101 (11)	82 - 118	108 (11)	92 - 124	0.516
Verbal	101 (12)	87 - 127	110 (11)	95 - 130	0.154
Performance	101 (13)	79 - 119	103 (11)	82 - 127	0.922
ADOS:					
Total	8 (1)				
Communication	3 (1)				
Social Interaction	5 (1)				
Gender	13 M: 2 F		13 M: 2 F		

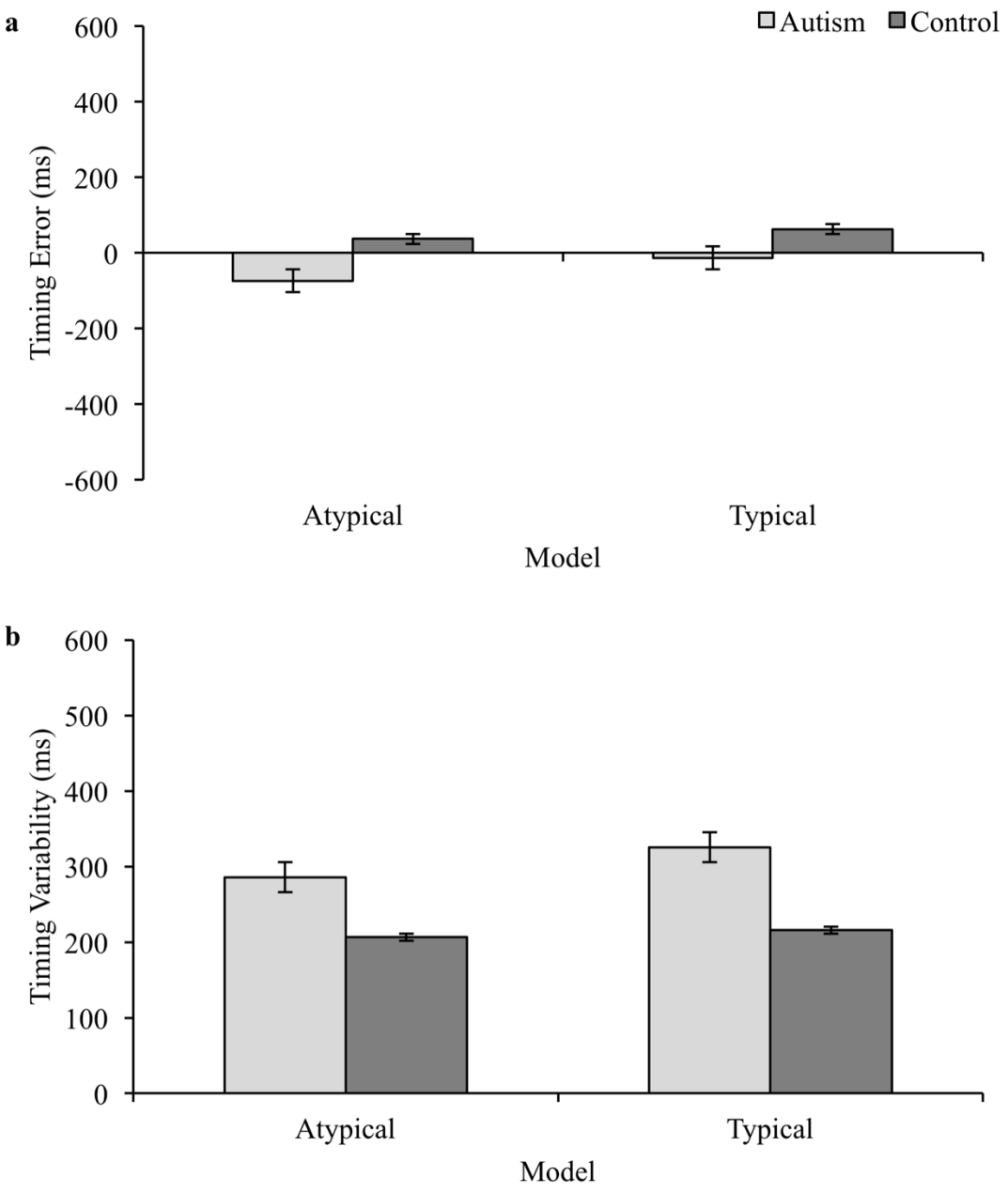

Figure 4.8 (a) Timing error and **(b)** timing variability for the imitation task (error bars represent standard error of the mean) in the Random experiment presented as a function of group and model.

4.13.2 Kinematic Data

4.13.2.1 Peak Velocity

A main effect of model [F(1, 28) = 39.937, $p < 0.001$, $\eta_p^2 = 0.588$] indicated magnitude was higher when imitating *atypical* (M = 0.247 mm/ms; SD = 0.043 mm/ms), compared to *typical* velocity model (M = 0.196 mm/ms; SD = 0.030 mm/ms) (**Figure 4.9a**). This was superseded by a model x group interaction [F(1, 38) = 4.324, $p = 0.047$, $\eta_p^2 = 0.134$], which indicated peak velocity was higher when imitating the *atypical* velocity model (**Figure 4.9a**) for the control (M = 0.262 mm/ms; SD = 0.040 mm/ms) than autism group (M = 0.233 mm/ms; SD = 0.046 mm/ms). There was no significant difference ($p > 0.050$) between the groups when imitating *typical* velocity model (autism: M = 0.199 mm/ms; SD = 0.033 mm/ms, control: M = 0.186 mm/ms; SD = 0.027 mm/ms). Correlation analysis revealed no relationship between peak velocity when imitating the *atypical* velocity model and ADOS total score (Pearson's r(15) = 0.318, $p = 0.248$) or *typical* velocity model and ADOS total score (Pearson's r(15) = 0.371, $p = 0.173$).

4.13.2.2 Time-to-Peak-Velocity

There was a main effect of model [F(1, 28) = 30.021, $p < 0.001$, $\eta_p^2 = 0.517$], where peak velocity occurred significantly earlier when imitating *atypical* (M = 29 %; SD = 8 %), compared to the *typical* velocity model (M = 38 %; SD = 10 %) (**Figure 4.9b**). This was superseded by a model x group interaction [F(1, 38) = 4.263, $p = 0.048$, $\eta_p^2 = 0.132$], which indicated peak velocity occurred significantly earlier when imitating *atypical* velocity model (**Figure 4.9b**) for the control (M = 26 %; SD = 7 %) than autism group (M = 31 %; SD = 10 %). The early occurrence

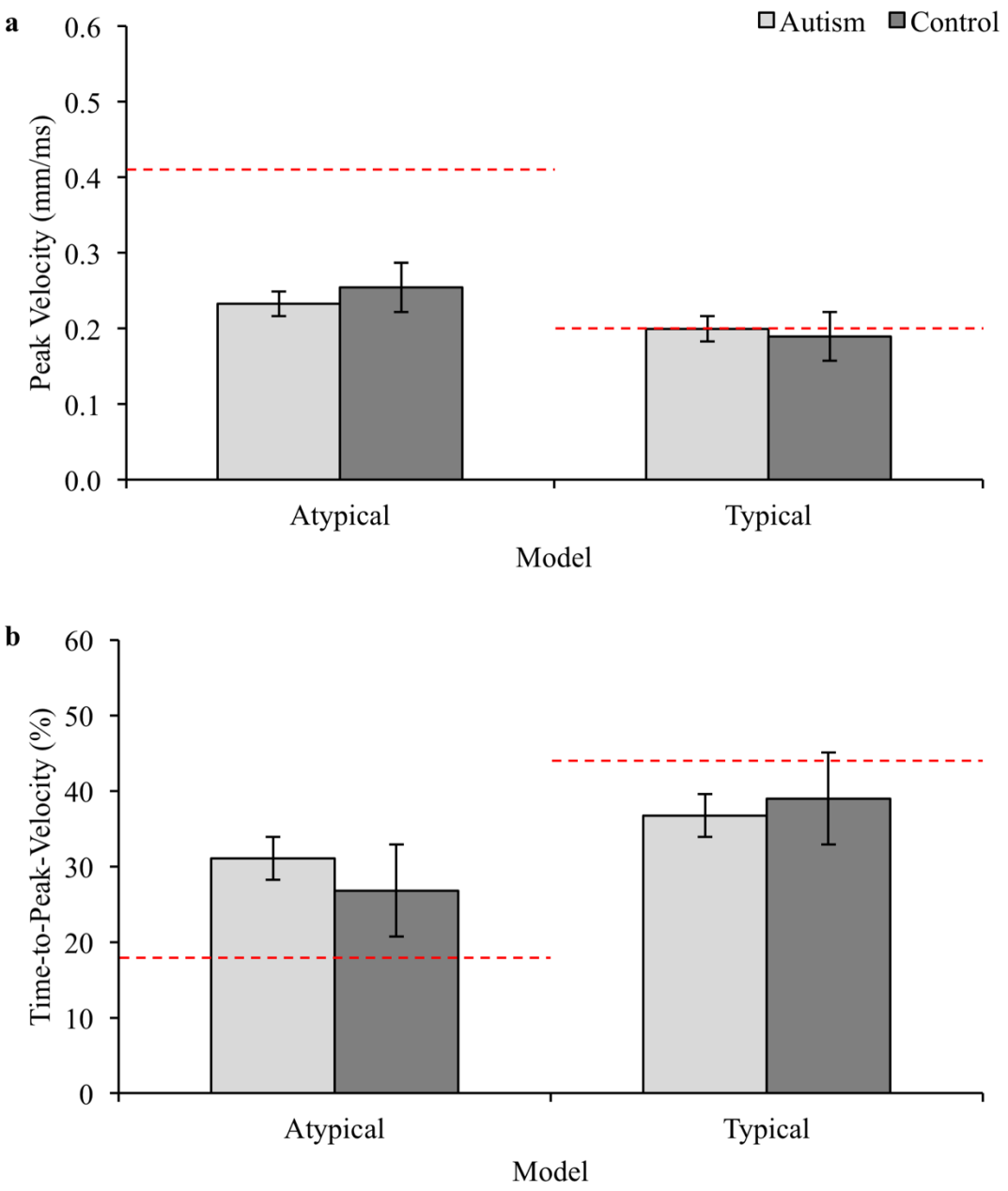

Figure 4.9 (a) Peak velocity and **(b)** time-to-peak-velocity for the imitation task (error bars represent standard error of the mean) in the Random experiment presented as a function of group and model. The dashed-red lines in **a** represent the magnitude of peak velocity for the *typical* (i.e., 0.410 mm/ms) and *atypical* (i.e., 0.200 mm/ms) models. In **b**, they represent the time-to-peak-velocity for the *typical* (i.e., 44 %) and *atypical* (i.e., 18 %) models.

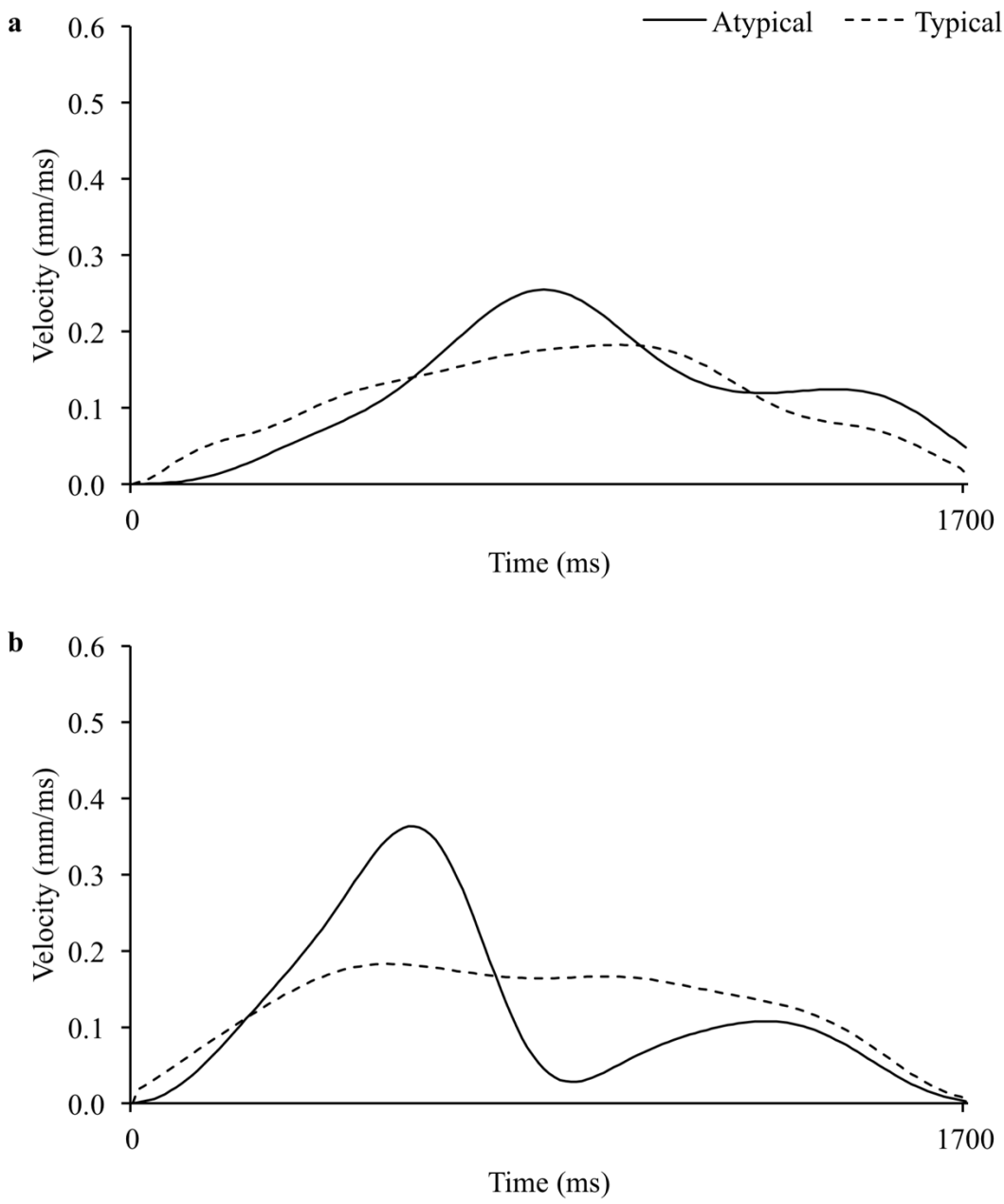

Figure 4.10 Velocity traces displaying exemplar kinematic data for the **(a)** autism and **(b)** control groups during imitation of the *typical* (dashed-black trace), *atypical* (solid-black trace) velocity models in the Random experiment presented as a function of time.

Table 4.6 Mean (standard deviation) timing and kinematic data for the imitation task in the Random experiment presented as a function of group and model.

		Autism	Control
Timing Data			
Timing Error (ms)	**Atypical**	-49	17
	Typical	-41	67
Timing Variability (ms)	**Atypical**	320	207
	Typical	324	262
Kinematic Data			
Peak Velocity (mm/ms)	**Atypical**	0.233 (0.044)	0.262 (0.047)
	Typical	0.199 (0.036)	0.191 (0.038)
Time-to-Peak-Velocity (%)	**Atypical**	30 (10)	27 (7)
	Typical	36 (11)	39 (10)

of peak velocity in the control group was more reflective of *atypical* velocity model (18 %; dashed-red line), than *typical* biological motion (44 %; dashed-red line). There was no significant difference ($p > 0.050$) between the groups when imitating *typical* velocity model (autism: M = 37 %; SD = 11 %, control: M = 39 %; SD = 10 %). Correlation analysis revealed no relationship between time-to-peak-velocity when imitating the *atypical* biological motion and ADOS total score (Pearson's r(15) = 0.132, $p = 0.639$) or *typical* biological motion and ADOS total score (Pearson's r(15) = -0.057, $p = 0.841$).

These above effects can be seen in the exemplar velocity traces illustrated in **Figure 4.10**. When imitating the *atypical* velocity model, peak velocity occurred significantly earlier in the movement for the control group (**Figure 4.10b**) than the autism group (**Figure 4.10a**). When imitating the *typical* velocity model, peak velocity occurred toward the midpoint of the movement for both groups (dashed-black trace; **Figure 4.10**). For a full breakdown of each dependent variable see **Table 4.6**.

4.14 Discussion

In this Random experiment, participants that were not naïve to the task (i.e., they took part in the Fixed or Interference experiment) observed and subsequently imitated the two models that were presented in an unknown structure (i.e., analogous to *Chapter Two*). As expected, and consistent with the findings from *Chapter Two* (autism: M = 33 %; control: M = 24 %) when the trial order was also randomised, imitation performance in the autism group was statistically similar to imitating the *typical* model (M = 39 %), and importantly significantly different to the neurotypical

control group. This finding supports the suggestion in the Fixed experiment low-fidelity imitation of *atypical* biological kinematics in autism is attributed to complications in integrating sensorimotor information on a trial-by-trial basis where there is opportunity for consolidation occur due to the unknown presentation structure. Furthermore, these results not only replicate those reported in *Chapter Two* but are also consistent with previous work that has indicated differences in imitating style (Rogers et al., 1996; Hobson & Lee, 1999) and action speeds (Wild et al., 2012; Stewart et al., 2013).

4.15 General Discussion

The primary aim of the current study was to examine imitation of *atypical* biological kinematics in adults with autism. A behavioural protocol was used that required participants to observe and subsequently imitate models that displayed distinctly different but biologically plausible kinematics (Hayes et al., 2014; Andrew et al., 2016). One of the most important findings from these studies is that through presenting the to-be-imitated stimulus in a fixed order, adults with autism imitated biological motion kinematics to a similar level as matched neurotypical controls. Here then, after observing the *atypical* model, participants in the autism group imitated movements with a time-to-peak-velocity that occurred at 27 % of the movement trajectory (**Figure 4.2b**). The early occurrence of peak velocity was reasonably similar to that displayed by the *atypical* model (18 %), and importantly similar to the control group (M = 25 %), and significantly different to the time-to-peak-velocity exhibited after imitating the *typical* model (M = 37 %). The increase in imitation fidelity suggests that imitation of *atypical* biological kinematics occurred

as a function of an increased opportunity for sensorimotor integration and consolidation across repeated trials of the same model. Over repeated attempts at imitating the *atypical* model in a fixed condition it is likely the refinement of the sensorimotor representation was facilitated by increasing the opportunity across similar trial types to process error through comparisons.

The suggestion that imitation fidelity was facilitated by integrating sensorimotor information during the inter-trial delay was supported by the findings from the Random experiment (**Figure 4.9b**) when the presentation structure was randomised and the Interference experiment (**Figure 4.6b**) where participants performed a secondary visuomotor task in the inter-trial delay. The secondary visuomotor task was implemented to experimentally interfere with the processing of sensorimotor information from trial *n*. As expected, and in line with previous chapters where the trial order was randomised, imitation performance in the autism group (M = 35 %) was statistically similar to imitating the *typical* model (M = 39 %), and importantly significantly different to the neurotypical control group (M = 28 %). Moreover, imitation performance was qualitatively different to the autism group from the Fixed experiment (M = 27 %) where the opportunity for sensorimotor consolidation during the inter-trial delay was not perturbed (Bandura, 1977; Byrne & Russon, 1998; Wolpert et al., 2011; Heyes, 2013). Further, similar findings were also reported in the Random experiment as again imitation performance in the autism group (M = 31 %) was statistically similar to imitating the *typical* model (M = 37 %), and importantly significantly different to the neurotypical control group (M = 26 %). Similar motor performance decrements have been reported from learning studies that examined contextual interference (for a review see Magill & Hall, 1990). For example, Shea and Morgan (1979) instructed participants to learn a three-segment

movement sequence under blocked (low contextual interference) or random (high contextual interference) practice conditions. Data indicated that motor performance was more accurate in the blocked condition, compared to random condition. During the random condition different sensorimotor representations (e.g., Task 1, followed by Task 2) are required to be constructed, deconstructed, and reconstructed across trials leading to an increase in sensorimotor interference within the inter-trial delay during the learning process (Li & Wright, 2000; Cross, Schmitt, & Grafton, 2007). While this may lead to benefits in long term retention (e.g., performance after 10 days) the interference effect was suggested to have an immediate influence on motor variability (Lee & Magill, 1983) and motor consolidation. The interference effect found from the secondary task in the Interference experiment is similar to data reported from an observational learning study (Brown et al., 2009) that induced motor interference using rTMS. Consistent with previous work (Mattar & Gribble, 2005), reaching performance was facilitated by observational learning. However, during the consolidation period repeated transmagnetic stimulation was applied to the primary motor cortex, learning was significantly reduced. This finding demonstrates sensorimotor information is consolidated in primary motor cortex, which is also known to be active during imitation learning (Nishitani et al., 2004).

This interpretation of sensorimotor integration being associated with impaired imitation of biological motion kinematics in autism is consistent with a recent collection of work that has shown that individuals with autism process and integrate visual and proprioceptive information differently (Sharer et al., 2015; Nebel et al., 2015; Hayes, Andrew, Foster, Elliott, Gowen, & Bennett, under review). When learning novel movements children with autism show a bias in the integration of sensorimotor feedback, favouring proprioception over visual (Haswell

et al., 2009: Marko et al., 2015). Furthermore, the finding of children with ADHD demonstrating a learning pattern indistinct from typically developing children points towards this being autism specific (Izawa et al., 2012). This bias towards proprioceptive feedback signals has also been evidenced in a ball catching task as part of the movement assessment battery for children (Brown & Lalor, 2009). The ball catching task requires a demand for the integration of temporal and spatial characteristics of the movement that requires online adjustments for successful task completion. Work employing the ball catching task (Whyatt & Craig, 2012) showed children with autism had difficulties in performing the task when compared to matched (age) typically developing children. Notably, these difficulties were not only dissimilar to typically developing controls, but also children with ADHD (Ament et al., 2015) indicating the adaptation of motor skills that require the coupling of visual and temporal feedback operate differently in autism. It was concluded that rather than general motor abilities (Green, Brennan, & Fein, 2002), motor skill deficits in autism are suggested to be allied with the ability to integrate visual spatial and temporal characteristics of an action (Ament et al., 2015).

In addition to the behavioural level differences in sensorimotor integration in autism, neurophysiological work has also shown that brain activity underpinning the development of sensorimotor representations differ compared to neurotypical controls (Müller, Pierce, Ambrose, Allen, & Courchesne, 2001; Müller, Kleinhans, Kemmotsu, Pierce, & Courchesne, 2003; Marko et al., 2015). For instance, using an fMRI paradigm fifty children with autism, and fifty matched (age, IQ and handiness) typically developing children were scanned during three gesture imitation tasks: (1) gesture imitation; (2) gestures to verbal command; (3) gesture involving tool use. Results showed that children with autism had increased intrinsic asynchrony in

neural activation between visual (lateral occipital cortex) and motor (pre- and post-central gyrus) regions which correlated with more severe traits of autism (measured via ADOS). Furthermore, children that exhibited greater intrinsic synchrony showed greater imitation accuracy. This altered synchrony could influence the integration of visuomotor information (Nebel et al., 2015). Based on these findings given that sensorimotor representations form part of a mechanism that facilitates the processing of biological motion for action-understanding and motor-execution (Blakemore & Decety, 2001), it may be that neural specificity of sensorimotor representations regulate how subsequent observed visual information is processed in social visuomotor contexts (Nebel et al., 2015). In the context of the current thesis, it could be proposed that during the Fixed experiment these brain mechanisms (lateral occipital cortex; pre- and post-central gyrus) occurring during imitation are operating effectively due to the consistent structure of the models leading to the sensorimotor representation being refined on a trial-by-trial basis (Wolpert et al., 2011). However, in the Interference experiment these neural processes are being interfered with, resulting in asynchronic brain activity which would be similar to those reported in the autism participants (Nebel et al., 2015). Finally, though the main emphasis of the study was self-other mapping, Williams et al. (2006) also suggested that problems with sensorimotor integration could account for problems in imitation in autism.

4.16 Summary

To conclude, adults with autism can imitate *atypical* biological motion kinematics to the same extent as matched neurotypicals by presenting the to-be-imitated models in a fixed structure which facilitates greater integration and consolidation of

sensorimotor information during the inter-trial delay. This was supported by the findings from the Interference experiment where the processing of sensorimotor information from trial *n* was experimentally interfered with. Imitation performance in the autism group was attenuated when required to perform a secondary motor task in the inter-trial delay. In a Random experiment similar to *Chapter Two* adults with autism exemplified differences in imitation of *atypical* biological kinematics when the presentation structure was fully-unknown.

5 Epilogue

5.1 Aim of the Chapter

The epilogue will summarise and synthesise the key findings observed across the program of work. There will be a critical evaluation with respect to current literature on imitation in autism, as well as implications for theoretical accounts of impaired imitation in autism and sensorimotor control processes in imitation. Future considerations and translational research will be discussed, with the intention of offering prospective social rehabilitation protocols in autism.

5.2 Aims of Thesis

The main aim of the present thesis was to examine imitation of biological motion in adults with autism, and to investigate whether adults with autism can adapt and learn to imitate and represent biological motion following specific manipulations to the imitation context (e.g., instructions, feedback, practice type). Across the three experimental chapters, a novel behavioural protocol was adopted that required adults with autism and matched (age, gender, handiness, IQ) adults without autism to observe and subsequently imitate models that displayed movements that had identical spatial and temporal outcomes, but with a *typical*, *atypical,* or *constant* (*Chapter Two* only) velocity profiles. The *atypical* model ensured the observer configured the sensorimotor system to represent the novel movement kinematics, as opposed to the *typical* model that could be achieved by rescaling an existing representation of a typical upper-limb aiming movement (Vivanti et al., 2008; Hayes et al, 2009; 2012). To control for specific top-down influences of coding biological motion (Kilner et al., 2007), and thereby minimised processes known to regulate

social modulation (Cook & Bird, 2012; Wang & Hamilton, 2012; Stewart et al., 2013) a non-human agent model (white-dot) was presented that had limited social context. To control for visual attention towards the goal-directed features of the task environment (Vivanti et al., 2008; Wild et al., 2012), target goals were only displayed in half of the imitation trials in *Chapter Two.* Targets were removed in *Chapters Three* and *Four* to encourage attention towards the trajectory of the model.

The aim of *Chapter Two* was to examine whether adults with autism can imitate *atypical* biological motion kinematics. Furthermore, to examine adaptation during imitation, performance was evaluated across the early-phase and late-phase of imitation. Based on the findings, there were two possible processes that could account for the underlying differences in imitating biological motion in autism. Firstly, it was suggested that visual attention away from the kinematic features of the model (i.e., movement trajectory) could lead to differences in sensorimotor information extracted for imitation (Vivanti et al., 2008; Wild et al., 2012; Gowen, 2012; Vivanti & Dissanayake, 2014). Secondly, individuals with autism may have had difficulties integrating sensorimotor information across trials that do not promote an opportunity for consolidation and representation development (Williams et al., 2006; Marko et al., 2015; Ament et al., 2015; Nebel et al., 2015).

In *Chapter Three*, the aim was to explore the first possible processing account of impaired imitation in autism by examining the effects of manipulating overt visual attention and intention during imitation of *atypical* biological motion kinematics in autism. Here, using a similar protocol as *Chapter Two*, adults with and without autism were provided with selective-attention verbal instructions prior to imitation, (Bach et al., 2007; Hayes et al., 2014) that directed visual attention towards the trajectory profile (i.e., kinematics) of the model(s). To determine if

visual attention was directed to the model stimuli during action-observation phase of imitation, and whether visual there were any differences in visual attention following selective-attention instructions, eye movements in all participants were recorded in both across both instruction conditions.

In *Chapter Four*, the aim was to explore the second possible processing account of impaired imitation in autism by examining sensorimotor integration and consolidation during imitation of *atypical* biological motion kinematics across a series of three studies. In the first study, a similar protocol as previous chapters were used, but now the to-be-imitated model(s) were presented in a fixed trial (i.e., blocked) order, thus increasing the opportunity for sensorimotor integration and consolidation during the inter-trial delay. In a second study, the same participants (autism: n = 9, control: n = 9) in the Fixed experiment imitated *atypical* biological motion kinematics (in a fixed presentation structure) while completing a secondary visuomotor task (drawing circles on the tablet) during the inter-trial delay, with the intention of interfering with sensorimotor integration and consolidation processes. Finally, the third study used the same participants as the Interference experiment and imitated using a similar protocol the Fixed experiment (i.e., no secondary visuomotor task) yet reverted to a random trial order. This was important in order to replicate the findings of *Chapter Two* and thereby confirm that trial order, and not simply individual differences, compromises sensorimotor consolidation.

5.3 Summary of Key Findings

Figure 5.1 summarises the key findings from the timing and kinematic data for each of the experimental chapters.

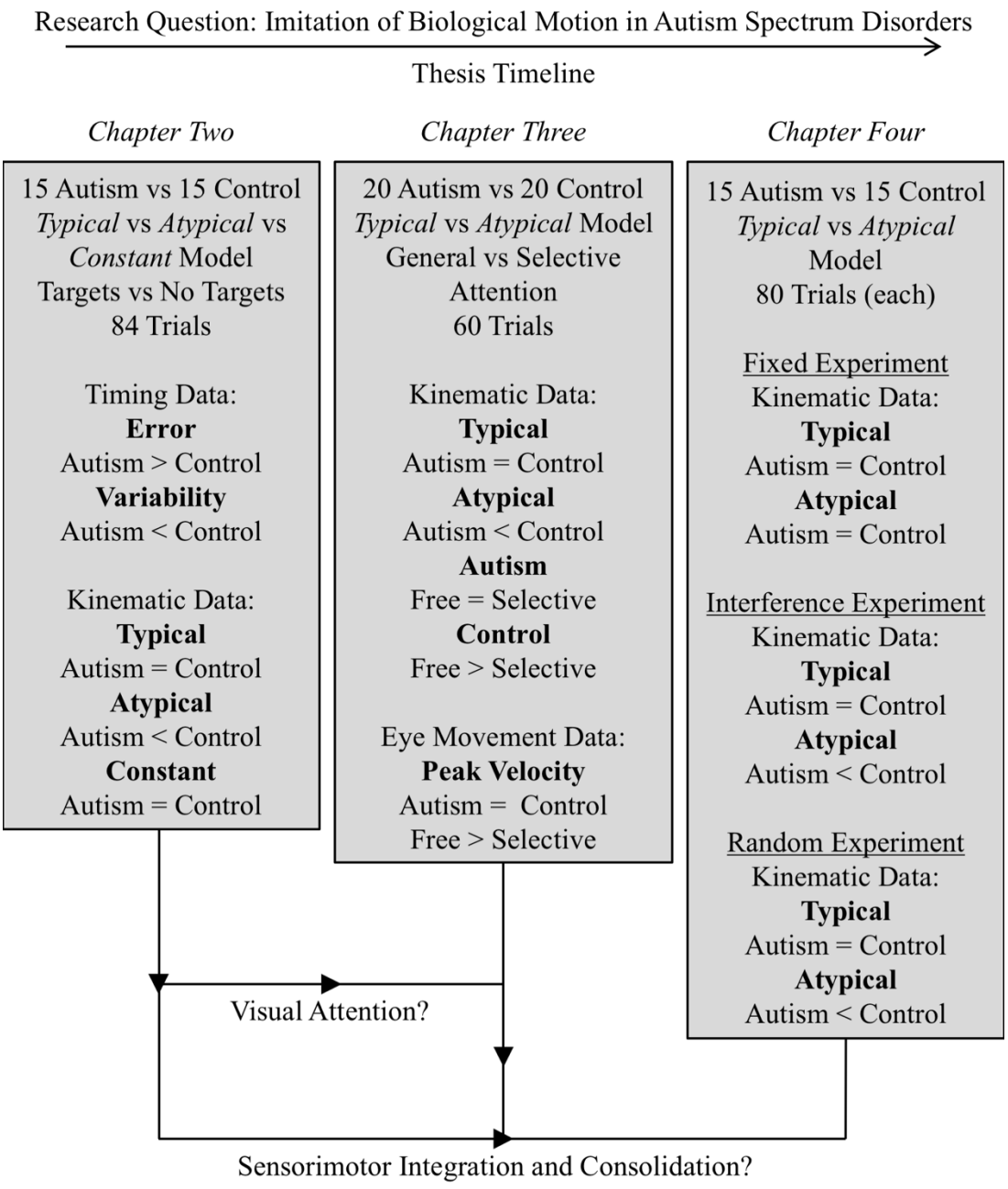

Figure 5.1 Schematic representation of the key findings across the three experimental chapters in the present thesis.

5.3.1 Chapter Two

As detailed in **Table 2.2**, examination of the kinematic data indicated that individuals in the autism group exhibited similar magnitudes of peak velocity as individuals in the control group, with both groups successfully modulating between velocity models. As after observing the *atypical* model, both groups imitated movements with a peak velocity (M = 0.238 mm/ms; SD = 0.037 mm/ms) that was significantly higher to the peak velocity when imitating the *typical* (M = 0.192 mm/ms; SD = 0.045 mm/ms) and *constant* (M = 162 mm/ms; SD = 0.030 mm/ms) velocity models (**Figure 2.3a**). Though there were no variances in magnitude, there were differences when imitating the timing of peak velocity, as after observing the *atypical* model, participants in the control group imitated movements with a time-to-peak-velocity that occurred at 24 % (SD = 8 %) of the movement trajectory. This early occurrence of peak velocity was similar to that displayed by the *atypical* model (18 %; **Figure 2.3b**), and significantly different to the time-to-peak-velocity exhibited after imitating the *typical* (M = 33 %; SD = 10 %) and *constant* (M = 38 % SD = 13 %) velocity models. This high-fidelity imitation of biological motion in the control group was not found for participants in the autism group. As after observing the *atypical* model, time-to-peak-velocity occurred at 33 % (SD = 10 %) of the movement trajectory, which was significantly different from the control group, but statistically similar to the time-to-peak-velocity exhibited when imitating the *typical* (M = 37 %; SD = 9 %) and *constant* (M = 38 %; SD = 11 %) velocity models.

Importantly, when performance was evaluated across the early-phase and late-phase of imitation an adaptation effect was found where individuals with autism became significantly more accurate at representing movement time by 35 % (175 ms), reducing movement time variability by 24 % (99 ms), and increasing the

magnitude of peak velocity by 12 % (0.024 mm/ms). This specific adaptation effect was not observed in the control group where neurotypical adults became significantly less accurate at representing movement time by 44 % (139 ms), and showed a non-significant change in movement variability (27 ms; 11 % change) and magnitude of peak velocity (0.009 mm/ms; 4 % change). The fact that adults with autism became significantly more accurate at imitating movement time, and exhibited a magnitude of peak velocity that was similar to the control group demonstrates that autistic participants are actively engaging true imitation (Carroll & Bandura, 1982; Byrne & Russon, 1998). Moreover, they also suggest that during action-observation visual attention may have been orientated towards the temporal properties of the movement (e.g., movement time) displayed by the non-human agent model (Vivanti et al., 2008; Wild et al., 2012; Gowen, 2012) at the expense of the kinematic properties. In contrast, reasonable high-fidelity imitation of the *atypical* biological kinematics in the control group may have been associated with visual attention towards the movement trajectory, which was examined in *Chapter Three* (black solid-line in **Figure 3.1**).

5.3.2 Chapter Three

As detailed in **Table 3.2,** examination of the kinematic data indicated when provided with general-attention instructions that guided observers to "*watch and copy the dot as it moves across the monitor*", participants in the control group exhibited movements with a time-to-peak-velocity that occurred at 29 % (SD = 8 %) of the movement trajectory. This early occurrence of peak velocity was significantly different to the time-to-peak-velocity exhibited after imitating the *typical* biological kinematics (M = 37 %; SD = 10 %). Low-fidelity imitation, and thus poor

representation of *atypical* biological kinematics was found for participants in the autism group. Consistent with *Chapter Two*, while they imitated movement time error (autism: M = 271 ms; SD = 355 ms, control: M = 71 ms; SD = 232 ms) and magnitude of peak velocity (autism: M = 0.217 mm/ms; SD = 0.051 mm/ms, control: M = 0.254 mm/ms; SD = 0.047 mm/ms) to a similar level of accuracy as the control group, time-to-peak-velocity in the *atypical* condition occurred at 32 % (SD = 10 %) of the movement trajectory, which was significantly different from the control group. Moreover, this time-to-peak-velocity was similar to that exhibited when imitating the *typical* biological kinematics (M = 36 %; SD = 11 %).

When provided with selective-attention instructions that explicitly guided participants to "*watch and pay attention to the dot's trajectory, with the intention to then copy the trajectory*", participants in the control group imitated with a time-to-peak-velocity that occurred significantly early in the movement after observing the *atypical* biological motion compared to *typical* biological motion. Although imitation of *atypical* biological motion was not modulated by selective-attention instructions for either group, imitation of *typical* biological motion kinematics became significantly 11 % (4 units) more accurate, and closer to the model in the neurotypical control group. The modulation of biological motion via task instructions has been shown in previous work (Hayes et al., 2014) and is suggested to be underpinned by a top-down mechanism that regulates (input modulation; Heyes & Bird, 2007) the lower-level visuomotor processes that code biological motion. A modulatory effect was not found in the autism group that exhibited little change in time-to-peak-velocity (4 %; 1 unit).

As detailed in **Table 3.3**, Examination of eye movements during action-observation indicated that peak and timing of smooth pursuit eye velocity was

similar between groups and instruction conditions, as well as being influenced in as a similar way by the biological motion kinematics. Also, while saccade amplitude was generally greater in the autism group (M = 4.84 saccades; SD = 1.58 saccades) compared to the control (M = 4.34 saccades; SD = 1.37 saccades) group, there was no increase in saccade amplitude nor number of saccades as a function of biological motion kinematics. The eye movement data imply that adults with autism and neurotypical controls maintained overt visual attention on the observed model irrespective of attention instructions (e.g., the velocity profiles of the eye during action-observation was similar to the velocity profile exhibited by the models; **Figures 3.8** and **3.9**). Accordingly, it is possible that eye movements could provide extra-retinal input (Barnes & Asselman, 1991) that is involved in configuring the upper-limb motor response required in imitation (Byrne & Russon, 1998; Hayes et al., 2014). Importantly, however, even though the autism group allocated overt visual attention to motion trajectory information, imitation was still attenuated.

Because gaze location does not necessarily coincide with the focus of attention, participants completed a series of debriefing questions designed to determine their thoughts and engagement with the studies. These data (Questions 1-4) indicated the autism group understood the instruction to pay more attention, and intend to imitate the trajectory following selective-attention instructions. For example, Participant 5 responded to Question 3 with the answer: "*Yes I think that you meant to watch the dot more closely, to notice the dot a bit more. I think that I noticed the dot more, like the way that it (the dot) moved and where the dot sped up and slowed down, and I tried to copy it (the dot) the same way*". In addition, Participant 11 reported their imitation strategy changed after receiving selective-attention instructions (Question 4): "*Yes I think so, I think I was better than the time*

before, I think I was faster, I think I sped up and slowed down like the fast then slow video that I watched". Finally, the group reported they could differentiate the two models, as exemplified by the response of Participant 4 to Question 1: "*Yes, one of the movements was fast and jagged then slowed right down, and the other one (the dot) was kind of like a similar speed all the way through*".

5.3.3 Chapter Four

As detailed in **Table 4.2**, in the first of three studies the kinematic data indicated that individuals in the autism group exhibited similar magnitudes of peak velocity to individuals in the control group, with both groups successfully modulating between velocity models. As after observing the *atypical* model, both groups imitated movements with a peak velocity (M = 0.274 mm/ms; SD = 0.047 mm/ms) that was significantly higher to the peak velocity when imitating the *typical* (M = 0.186 mm/ms; SD = 0.033 mm/ms) velocity model (**Figure 4.2a**). Furthermore, in line with *Chapters Two* and *Three*, examination of the timing of peak velocity showed the control group exhibited reasonably high-fidelity imitation of *atypical* biological kinematics. As after observing the *atypical* model, participants in the control group imitated movements with a time-to-peak-velocity that occurred at 25 % (SD = 7 %) of the movement trajectory. The early occurrence of peak velocity was similar to that displayed by the *atypical* model (18 %; **Figure 4.2b**), and significantly different to the time-to-peak-velocity exhibited after imitating the *typical* model (M = 39 %; SD = 9 %). One of the most significant findings across the present thesis is that participants in the autism group imitated movements with a time-to-peak-velocity that occurred at 27 % (SD = 9 %) of the movement trajectory, which was significantly different to the time-to-peak-velocity exhibited after

observing *typical* (M = 37 %; SD = 12 %) biological motion (**Figure 4.2b**). These timings of peak velocity were statistically similar to that of participates in the control group, hence individuals with autism also exhibited reasonably high-fidelity imitation of *atypical* biological kinematics. This effect was achieved by presenting the to-be-imitated models in a fixed trial order. which led to the suggestion that imitation of *atypical* biological kinematics occurred as a function of an increased opportunity for sensorimotor integration and consolidation across repeated trials of the same model. However, it was unclear whether this increased sensorimotor integration and consolidation occurred online during motor-execution (Burke et al., 2010), and/or offline during the inter-trial delay (Wolpert et al., 2011).

As detailed in **Table 4.4**, in the Interference experiment, the kinematic data indicated temporal correspondence between control participants and the *atypical* biological kinematics. As after observing the *atypical* model, participants in the control group imitated movements with a time-to-peak-velocity that occurred at 28 % (SD = 5 %) of the movement trajectory. The early occurrence of peak velocity was similar to that displayed by the *atypical* model (18 %; **Figure 4.6b**), and significantly different to the time-to-peak-velocity exhibited after imitating the *typical* model (M = 45 %; SD = 10 %). Though they showed reasonable high-fidelity imitation of *atypical* biological motion, it is notable that neurotypical controls exhibited a time-to-peak-velocity that occurred later than those reported in the Fixed experiment, thus indicating that the secondary visuomotor task leads to an increase in sensorimotor interference that consequently impacts the fidelity of a movement representation. Notably, while participants in the autism group imitated the magnitude of peak velocity (autism: M = 0.222 mm/ms; SD = 0.083 mm/ms, control: M = 0.236 mm/ms; SD = 0.087 mm/ms) to a similar level as the controls. The

reasonably high-fidelity imitation of the timing of peak velocity in the Fixed experiment deteriorated, and instead consistent with *Chapters Two* and *Three*, they imitated movements with a time-to-peak-velocity occurred at 35 % (SD = 9 %) of the movement trajectory. This was significantly different from the control group, but statistically similar to the time-to-peak-velocity exhibited when imitating the *typical* (M = 39 %; SD = 10 %) velocity model. This decline in imitation fidelity of the *atypical* biological kinematics through experimentally interfering with the processing (i.e., integration) of sensorimotor information indicates that this increased sensorimotor integration and consolidation in the Fixed experiment occurred offline during the inter-trial delay.

As detailed in **Table 4.6**, in the Random experiment, the kinematic data indicated that in accordance with *Chapter Two* were the presentation structure of the models was also randomised, participants in the control group imitated movements with a time-to-peak-velocity that occurred at 27 % (SD = 7 %) of the movement trajectory. The early occurrence of peak velocity was similar to that displayed by the *atypical* model (18 %; **Figure 4.9b**), and significantly different to the time-to-peak-velocity exhibited after imitating the *typical* model (M = 39 %; SD = 10 %). Furthermore, the kinematic data further supported the notion that increased sensorimotor integration and consolidation in the autism group in the Fixed experiment occurred as a function presenting the model in a blocked presentation structure. As unlike the Fixed experiment and consistent with the Interference experiment, they imitated movements with a time-to-peak-velocity occurred at 30 % (SD = 10 %) of the movement trajectory. This was significantly different from the control group, but statistically similar to the time-to-peak-velocity exhibited when imitating the *typical* (M = 36 %; SD = 11 %) velocity model.

5.4 Implications for Processing Accounts of Imitation

As evidenced through a review of the literature within the introductory chapter of the thesis, there is strong evidence of impaired imitation of biological motion in autistic children, adolescents and adults (Williams et al., 2001; Vanvuchelen, Roeyers, & De Weerdt, 2011; Hamilton, 2013; Edwards, 2014; Vivanti & Hamilton, 2014). As a result, many researchers endeavoured to ascertain the mechanism(s) associated with these atypicalities in imitation in autism. Given the experimental manipulations and results observed across this programme of work, the subsequent sections discuss the implications for current theories of imitation in autism.

5.4.1 Mirror Neuron System; Self-Other Mapping Processing

One of the most central findings reported in *Chapter Two* was that although both groups performed similarly when imitating the *typical* (autism: M = 37 %; SD = 9 %, control: M = 33 %; SD = 10 %) and *constant* (autism: M = 38 %; SD = 11 %, control: M = 38 %; SD = 13 %) velocity models that could achieved by rescaling and existing sensorimotor representation (Hayes et al., 2009). When required to imitate a model by configuring the sensorimotor system to represent the novel kinematics, adults with autism performed significantly worse (M = 33 %; SD = 10 %) than matched neurotypical adults (M = 24 %; SD 8 %) (**Figure 2.3b**). These imitation findings observed where using an object-movement re-enactment protocol (OMR; Whiten, Horner, Litchfield, & Marshall-Pescini, 2004) that does not display the human model physically executing the action to achieve the end product and controls the influence of social-affective processes known to modulate imitation (Whiten et al., 2009). A comparable study by Stewart et al. (2013) that also used an OMR task

too found attenuation in imitating movement kinematics in adolescents with autism. Here then, when compared to neurotypical adolescents that showed no differences in imitating shapes using a stylus on a graphics tablet (i.e., similar to the present thesis) following action-observation of a human or non-human model, adolescents with autism showed significantly less imitation accuracy in action duration and path length in both conditions. It was suggested that impaired imitation in autism was underpinned by differences in the lower-level visuomotor system that maps the biological motion onto the motor system, also more commonly referred to as the mirror neuron system hypothesis (Williams et al., 2001; 2004; Ramachandran & Oberman, 2005).

The mirror system hypothesis suggests that impaired imitation of biological motion kinematics in autism is associated with lower-level processes that integrate sensorimotor information (Oberman et al., 2005; Théoret et al., 2005; Dapretto et al., 2006; Williams et al., 2006). These processes are part of a functional network that represents an observed movement by mapping the biological motion characteristics directly onto the motor system (Iacoboni et al., 1999; Rizzolatti & Craighero, 2004). Using various brain imaging techniques (e.g., PET; EEG; TMS; fMRI), there is a large body of research has shown differences in cortical activity in regions associated with the lower-level processes during imitation and action-observation in autism (Nishitani et al., 2004; Théoret et al., 2005; Oberman et al., 2005; Dapretto et al., 2006; Bernier et al., 2007; Oberman, Ramachandran, & Pineda, 2008; Martineau, Cochin, Magne, & Barthelemy, 2008; Enticott et al., 2012). Importantly, *Chapter Two* isolated whether the imitation deficit in autism is attributable to imitating specific lower-level properties (e.g., velocity) of biological motion kinematics. This was achieved by employing a novel protocol that required participants to imitate

movements that had distinctly different, but still biologically plausible, movement kinematics (Hayes et al., 2014; Andrew et al., 2016). Albeit the findings in *Chapter Two* were behavioural and not neurophysiological, given the protocol isolated lower-level processing as well as the resemblances in task (i.e., OMR; Stewart et al., 2013) and findings of the abovementioned study, the attenuated imitation of *atypical* biological kinematic in *Chapter Two* could also be impaired by differences in the lower-level visuomotor processes (Williams et al., 2006; Dapretto et al., 2006; Haswell et al., 2009; Izawa et al., 2012; Nebel et al., 2015).

It is well accepted that lower-level processes associated that underpin imitation of biological motion kinematics are regulated by top-down attentional (end-state goals) and social (human form; eye contact) factors (Kilner et al., 2007; Stanley et al., 2007; Southgate & Hamilton, 2008). One way to regulate the lower-level processes in by providing specific instructions to direct overt visual attention to the movement trajectory enhances imitation fidelity of *atypical* biological kinematics in neurotypicals (Hayes et al., 2014). Therefore, to examine whether low-fidelity imitation of biological kinematics in autism is due to reduced attention to the trajectory (Wild et al., 2012), or processes associated with visuomotor integration in *Chapter Three*, participants were provided selective-attention instructions directed towards the movement trajectory. As can be seen in **Figure 2.3b**, these top-down selective attention instructions did not modulate input to the lower-level system mirror system. Similar to the findings of *Chapter Two* (M = 33 %; SD = 10 %), imitation of *atypical* biological kinematics was still attenuated (M = 32 %; SD = 9 %) compared to neurotypical adults (M = 28 %; SD = 8 %) and was not modulated by top-down selective-attention instructions (autism: 5 % change; 2 units, control, 8 % change; 2 units). These findings are consistent with previous studies (Stewart et

al., 2013) and may suggest that rather than impaired imitation in autism being related to the focus of attention during action-observation (Wild et al., 2012; Gowen, 2012), it could be based on a basic dysfunction in lower-level sensorimotor processes that integrate sensorimotor information control self-other mapping (Williams et al., 2001; 2004; Rogers & Williams, 2006).

Similar dysfunction in lower-level processes were promoted to describe differences in brain activity in an imitation study that used functional magnetic resonance imaging (fMRI) to examine the neural mechanism of imitation (Williams et al., 2006). For instance, sixteen adolescents with and without autism (matched for age, gender and IQ) were scanned while ask to observe, execute or imitate index finger movements. While both groups exhibited similar imitation task performance, in line with previous studies (Iacoboni et al., 1999) the control participants showed activation within the right parietal lobe and the right temporo-parietal junction. In comparison, activity in this area was less extensive in individuals with autism. It was suggested that the altered brain activity patterns during motor imitation could stem from poor integration between brain areas serving sensorimotor integration that would impact the development of a representation (see also Dapretto et al., 2006).

One way to increase sensorimotor integration is by providing blocked practice structures which allows for greater opportunity for response-produced error and variability to be reduced over similar trial types leading to a more refined sensorimotor representation (Wright & Shea, 2001). As can be seen from **Figure 4.2**, in contrast to kinematics observed in *Chapters Two* (M = 33 %; SD = 10 %) and *Three* (M = 31 %; SD = 10 %), in the Fixed experiment of *Chapter Four* when in a context that presented the stimulus in a fixed trial order, participants in the imitated the *atypical* model with kinematics (M = 27 %; SD = 9 %) that were similar to the

neurotypical control group (M = 25 %; SD = 7 %) and importantly were significantly different from the *typical* model (M = 37 %; SD = 12 %). Based upon these findings observed, it seems the fixed trial order allowed the underlying lower-level visuomotor processes within the autism group to be effectively engaged in order to integrate and process sensorimotor information. Moreover, the findings also indicate that imitation of biological kinematics may not be fundamentally impaired in autism (Williams et al., 2001; 2004; Théoret et al., 2005; Oberman et al., 2005) but rather the processes underlying this mechanism need to be operationalised in a specific imitation learning context to overcome the sensorimotor integration problems that underpin imitation in autism (Williams et al., 2006; Haswell et al., 2009; Izawa et al., 2012; Marko et al., 2015; Ament et al., 2015; Nebel et al., 2015). The issue of sensorimotor integration and lower-level processes are directly discussed in a following section.

5.4.2 Visual Attention Processes

As discussed within *Chapter* One, overt visual attention during the action-observation phase of imitation has previously been suggested to underpin imitation impairments in autism (Hobson & Hobson, 2007; Vivanti et al., 2008; Vivanti & Dissanayake, 2014). Although eye movements were not directly examined in *Chapter Two*, behavioural data could be interpreted to suggest that impaired imitation of *atypical* biological kinematics in adults with autism may have been accompanied by differences in overt visual attention. Here then, timing data (**Figure 2.3b**) showed that adults with autism adapted movement time from the early- to late-phase of imitation, becoming more accurate by 35 % (175 ms), compared to the control group increased timing error by 44 % (139 ms). Though adults with autism

accurately represented movement time, this may have been at the expense at imitating in the kinematics. As illusatred in **Figure 2.3b** only the neurotypical controls imitated with time-to-peak-velocity (M = 24 %; SD = 8 %) that was similar to that displayed by the *atypical* model (18 %; **Figure 2.3b**). In comparison, adults with autism exhibited a time-to-peak-velocity (M = 33 %; SD = 10 %) that statistically similar to the *typical* (M = 37 %; SD = 9 %) model. These findings suggest low-fidelity imitation of *atypical* biological kinematics in adults with autism may have been associated with bias in orientation of visual attention towards movement time over the kinematics, whereas on the contrary neurotypical controls may have orientated visual attention towards movement kinematics over movement time. This interpretation of the data is consistent with differences in eye movements reported in a study by Wild et al. (2012) during imitation of hand actions. As compared to neurotypical controls that spent more time pursuing the hand of the model leading to successful imitation of action speeds, adults with autism spend more time shifting attention towards the action end-point. This focus away from the hand may have resulted in the movement kinematics not being perceived and processed (Wild et al., 2012; Gowen, 2012).

While the findings form *Chapter Two* show partial support previous suggestions for differences in visual attention in autism. *Chapter Three* directly examined visual attention in autism by recording eye movements in the action-observation phase of imitation. As illustrated by the solid-black trace in **Figures 3.6** and **3.7**, in an initial phase where general-attention instructions were provided ("*watch and copy the dot as it moves across the monitor"*) that did not specify what aspects of the model to imitate (i.e., similar to *Chapter Two*), both groups exhibited similar magnitudes and time-to-peak smooth pursuit eye velocity (**Figure 3.8**),

combined with fewer saccades of greater amplitude, when observing *atypical* compared to *typical* velocity model (**Figure 3.9**). However, although the eye movements indicated that adults with autism successfully tracked the movement trajectory, this did not result in successful imitation of the *atypical* biological kinematics. As consistent with *Chapter Two*, only the neurotypical adults (M = 28 %; SD = 8 %) exhibited a time-to-peak-velocity similar to the *atypical* model (**Figure 3.3b**), compared to adults with autism (M = 32 %; SD = 9 %) that exhibited timing of peak velocity similar to the *typical* (M = 36 %; SD = 10 %) model. These findings demonstrate not only that visual attention was maintained on the observed model during action-observation, but also that participants had comparable retinal and extra-retinal input for the configuration of the upper-limb motor response required in imitation.

One way to experimentally manipulate the orientation of visual attention is by providing specific instructions that direct attention towards the movement trajectory and have previously been shown to enhance imitation accuracy kinematics in neurotypicals (Hayes et al., 2014). Therefore, in a second phase of *Chapter Three*, the same participants were provided with instructions that specifically instructed all participants to pay attention to, and intend to imitate the stimulus' movement trajectory. As illustrated in **Figures 3.6** and **3.7** the selective-attention instructions had a modulatory effect on eye movements. Compared to the solid-black trace which represents the general-attention instructions, the dashed-black trace which represents selective-attention instructions indicated that both groups eye movements became significantly closer to the observed model. Further analysis signified that for both group the magnitude of peak velocity increased by 7 % (0.795 deg/s; **Figure 3.8a**) and the timing of peak velocity occurred significantly earlier and closer to the model

in the selective-attention condition by 18 % (8 units; **Figure 3.8b**). Notably, while the selective-attention instructions modulated visual attention, there was no significant change in the accuracy of imitating *atypical* biological kinematics in the autism group following selective-attention instructions (**Figure 3.3b**). By using a non-human agent (white dot) to control for the influence of social attention (Vivanti & Hamilton, 2014), it is difficult to directly compare the eye movements reported in *Chapter Three* to previous work on imitation that used a human social setting (e.g., Hobson & Hobson, 2007; Vivanti et al., 2008; Wild et al., 2012; Vivanti & Dissanayake, 2014). However, because of adults with autism showing similar eye movement patterns as matched neurotypicals, it seems unlikely that impaired imitation in autism reported in *Chapter Three* is related to poor tracking of the models trajectory. In addition, while eye-movements were not recorded, given the similarities experimental protocol and finings in the general-attention phase in *Chapter Three* and other experimental chapters, it could also be suggested that the eye movements may have been similar across this thesis and hence are also unlikely to be attributed towards differences in visual attention (Gowen, 2012). From the findings reported in *Chapter Three*, as an alternative to visual attention it was concluded that rather than impaired imitation of *atypical* biological kinematics in autism being associated with visual attention, it could alternatively be attributed to altered 'input modulation' associated with how the lower-level sensorimotor processes are controlled during the encoding of biological kinematics (Southgate & Hamilton, 2008; Welsh, Ray, Weeks, Dewey, & Elliot, 2009), and/or the integration of sensorimotor information across imitation (Mostofsky & Ewen, 2011; Hannant, Tavassoli, & Cassidy, 2016). This latter point is discussed in more detail later in this chapter.

5.4.3 Processing Biological Kinematics

Perception of biological motion is important for early development of social cognition and if an individual is unable to perceive differences between biological motion kinematics, then it can be expected that consequently they would also not exhibit differences when imitating the kinematics. In order to determine whether participants had engaged in the experiment and understood the task instructions a post-experimental debrief was developed. When participants were asked "*Did you notice anything about the movements you observed?*", overall individuals in the autism group reported they could differentiate the two models, as exemplified by the response of Participant 4 to Question 1: "*Yes, one of the movements was fast and jagged then slowed right down, and the other one (the dot) was kind of like a similar speed all the way through*". This suggestion from the participants in the autism group that they could differentiate between the biological models was supported by findings from the judgement task which was included as a control measure in *Chapter Three*. In this task, participants observed two models (identical to the action-observation phase of imitation) and were instructed to indicate whether the models had either similar (i.e., *atypical*; *atypical*) or diverse (i.e., *atypical*; *typical*) movement trajectories. As can be seen in **Figure 3.10b**, the autism group accurately perceived differences between *atypical*, *typical*, and *constant* velocity kinematics. As from a possible total of 45 correct responses, the autism group made 30 (SD = 9) correct responses which was similar to the control group (M = 27; SD = 7), and at a level significantly greater than chance (66 %). These above findings make it unlikely that impaired imitation of biological kinematics observed in the autism group in *Chapter Three* (**Figure 3.3b**) were due to an inability to perceive differences in biological motion.

Moreover, the intact biological perception effect found in the judgement task in *Chapter Three* is similar to data reported from a study that used point-light displays (Cusack et al., 2015). In this study, adolescents with autism and matched neurotypical adolescents completed a battery of action-perception tasks that involved: (1) differentiating between biological and non-biological motion; (2) discrimination between robotic and natural motion; (3) discrimination of one form of action from another; (4) integration of limbs into full-body agents; (5) discrimination of two agents that are temporally synchronous or not; (6) attending to biological motion signals. Results indicated that consistent with previous work signifying intact biological motion perception (Freitag et al., 2008; Saygin et al., 2010; Wild et al., 2012; Cook et al., 2013), across all six experiments autistic adolescents exhibited similar performance scores as neurotypical controls. Though the findings from the judgement task show intact biological motion perception in autism, work also using point light displays have shown difficulties in perception of biological motion in autism (Blake et al., 2003; Freitag et al., 2008). For example, in a study by Nackaerts et al. (2012), typically developed controls were more accurate than individuals with autism in recognising emotions from point light displays. While these studies show differences, this occurred when examining emotion and social interaction. In comparison, in the present judgement task a single white dot (indistinguishable to the imitation task) that controlled for social interaction (Spengler et al., 2010; Cook & Bird, 2012) was utilised. Therefore, though the findings from the judgement task in *Chapter Three* cannot be directly compared to these studies, it can be suggested that participants with autism could successfully perceive differences between the *typical*, *atypical*, and *constant* models, and did not influence the imitation findings across this thesis. One limitation across this thesis is that the judgement task, as well as the

post-experimental debrief was only utilised in *Chapter Three*, for this reason future work examining imitation in autism should always look to control for perception of biological motion.

5.5 Implications for Sensorimotor Control Processes

In the introductory chapter of the present thesis a review of the sensorimotor control processes in imitation in an attempt to isolate possible impairments which may contribute to the difficulties in imitation observed in individuals with autism (Gowen & Hamilton, 2013). Given the experimental manipulations and results observed across this programme of work, there are several implications for the sensorimotor control processes discussed in *Chapter One*. These will be returned to in the proceeding subsections of the epilogue. Given that action-observation and visual attention have been discussed in detail previously, this will not be revisited. This will be the same for motor planning as no experimental manipulations examined motor-planning.

5.5.1 Motor-Execution

Previous studies examining how individuals with autism execute movements have shown a consistent finding that despite they can execute actions that show similar accuracy levels as neurotypical counterparts, their underlying movement kinematics seem to be far more variable (Mari et al., 2003; Rinehart et al., 2006; Glazebrook et al., 2006). Standard deviations reported from the kinematic data in *Chapters Two* and *Three* are in agreement with these studies. As can be seen in **Tables 2.2** and **3.2**, overall when imitating the *atypical* kinematics, though autism

group had similar variability in the magnitude of peak velocity (autism: *Chapter Two* = 0.045 mm/ms; *Chapter Three* = 0.051 mm/ms, control: *Chapter Two* = 0.046 mm/ms; *Chapter Three* = 0.046 mm/ms), the timing of peak velocity (*Chapter Two* = 10 %; *Chapter Three* = 10 %) was more variable than controls (*Chapter Two* = 8 %; *Chapter Three* = 8 %). Similar differences in variability in kinematics in motor-execution in autism have also been shown in a study whether individuals with autism move with an unnatural kinematic profile (Cook et al., 2013). When performing sinusoidal arm movements, compared to controls adults with autism performed movements that were more 'jerky' with greater acceleration and velocity compared to matched neurotypical adults. These jerky movements positively correlated with autism severity (ADOS) suggesting that they may be autism specific. Furthermore, it was implied that these kinematic results may have due to individuals with autism having poor anticipation of the subsequent part of the action sequence (Fabbri-Destro et al., 2009) thus leading to a compromised ability to predict when to change direction (i.e., from a left to right arm movement).

Though the abovementioned suggestion seems plausible, in the context of the current thesis this seems unlikely. As compared to these studies that used actions that required multiple movements, as can be seen in **Figure 1.2** the imitation task required a single movement which would not involve participants to anticipate a second action. An alternative and more probable proposition is that in *Chapters Two* and *Three*, participants in the autism group were not provided appropriate time to integrate sensorimotor information (Rinehart et al., 2001; Gowen et al., 2007; Nazarali et al., 2009). This suggestion that greater sensorimotor information is facilitated by providing more adequate time is supported by findings from performance evaluations in *Chapter Two*, as can be seen in **Table 2.2** when imitating

the *atypical* model, participants in the autism group decreased variability in timing of peak velocity by 21 % (2 units). Furthermore, in addition to the kinematics as can also be illusatred in **Figure 2.2b** the autism group also significantly decreased timing variability by 24 % (99 ms), becoming closer in the late-phase of imitation (M = 314 ms) as the control group (M = 227 ms). Similar sensorimotor integration explanations have been forwarded to explain differences in overall motor-execution times in manual aiming studies that examined vision. Glazebrook et al. (2009) had participants perform eye movements and/or manual aiming movements with or without vision. Results signified that in general individuals with autism used sensorimotor information to execute the movements, however they took significantly more time to execute movements that required greater sensorimotor integration. Importantly, variability in the movements kinematics in the Fixed experiment in *Chapter Four* support this suggestion that motor-execution variability is associated with sensorimotor integration. As can be seen in **Table 4.2**, although the autism group are overall more variable than neurotypical controls (SD = 8 %), the standard deviations when imitating the *atypical* model were similar to those in the late-phase of imitation of *Chapter Two* (SD = 9 %) and different from the early-phase (SD = 11 %). Therefore, it could also be suggested that in addition to providing adequate opportunity, this decrease in motor-execution variability may be associated with the blocked practice structure which allows for increased sensorimotor integration. The influence of sensorimotor integration on imitation in autism is discussed in more information in the proceeding sections:

5.5.2 Sensorimotor Consolidation

Many of the previous studies examining imitation in autism have collapsed

analysis over all trials (Wild et al., 2012; Stewart et al., 2013). While this is a suitable approach, this may mask important information about imitation adaptation. An alternative approach that was utilised in *Chapter Two* was to evaluate performance in the early-phase (i.e., first five trials) and late-phase (i.e., last five trials) of imitation, which is akin to that used in observational learning studies (Hayes et al., 2008; Andrew et al., 2016). As can be seen in **Figure 2.2**, this analysis revealed that adults with autism became significantly more accurate at imitating movement time by 35 % (175 ms), while also reducing movement time variability by 24 % (99 ms) from early-phase to late-phase of imitation. Compared to the control group that became significantly less accurate at imitating movement time by 44 % (139 ms). Still the kinematic data revealed that imitation of biological kinematics was attenuated in individuals with autism (M = 33 %; SD = 10 %) compared to controls (M = 24 %; SD = 8 %), which did not change from early-phase to late-phase of imitation (**Figure 2.3b**). Therefore, these findings, more specifically the timing data suggest that individuals with autism can successful refine and adapt a sensorimotor representation (Wolpert et al., 2011). Similar intact sensorimotor adaptation and consolidation has been reported in autism through motor adaptation studies that examined performance changes following perturbations in the environment (Mostofsky et al., 2004; Gidley Larson et al., 2008). In the latter study, autistic children were required to throw a ball at a target wall while wearing prism goggles that shifted vision. Compared to a baseline condition (no goggles), autistic children adapted their motor behaviour to meet the demands of the new environment to the same extent as neurotypical children. Moreover, when the goggles were worn during training, both autism and control groups adapted their movements across from early to late adaptation, again becoming closer to the baseline.

The adaptation of movement time reported in *Chapter Two* is also similar to data reported from a recent motor learning study (Hayes et al., under review). Here then, adults with autism and matched (age, gender, IQ) neurotypical control adults physically practiced a three-segment movement sequence in accordance with a timing goal (for more information see Andrew et al., 2016) by moving a using a stylus such that the cursor passed through the sequence to achieve a movement time goal. To facilitate learning, knowledge-of-results (Winstein & Schmidt, 1990) were provided following every trial. As can be seen in **Figure 5.2**, albeit motor performance in the autism group was generally less accurate than the control group (Ghaziuddin & Butler, 1998; Fournier et al., 2010). In line with findings from *Chapter Two* that evaluated performance form early- to late-phase (**Figure 2.2**) adults with autism significantly adapted timing accuracy (66 % change; 843 ms) and timing variability (66 %; 363 ms) similar to the controls (accuracy, 67 % change; 532 ms, variability, 61 % change; 281 ms), becoming closer to the goal. These findings further demonstrate intact creation and refinement of a sensorimotor representation (Mostofsky et al., 2004; Gidley Larson et al., 2008) by integrating self-generated efferent sensorimotor commands, afferent sensorimotor information, and visual consequences of a performed action (Wolpert et al., 2011).

Though this study as well the data from *Chapter Two* show successful motor adaptation in autism, this is only for timing and not kinematics which was examined in the present thesis. As can be seen in **Figure 4.2b**, adults with autism represented *atypical* biological kinematics (M = 27 %; SD = 9 %) to the same extent as neurotypical controls (M = 25 %; SD = 7 %). This high-fidelity in autism is different from those reported in *Chapters Two* (M = 33 %; SD = 10 %) and *Three* (M = 31 %; SD = 10 %). This increase in imitation fidelity occurred by providing a blocked

practice structure, which provides an increased opportunity for response-produced error and variability to be reduced over similar trial types leading to a more refined sensorimotor representation (Wright & Shea, 2001). Thus, it could be suggested that some sensorimotor adaptation may have taken place across imitation during the Fixed experiment in *Chapter Four*. To examine motor adaptation of kinematics in the Fixed experiment, in a similar vein as *Chapter Two*, the data from the Fixed experiment was reanalysed by evaluating performance in the early-phase and late-phase of imitation. As can be interpreted from **Figure 5.3**, when imitating the *atypical* model, adults with autism increased the magnitude of peak velocity by 21 % (0.047 mm/ms) and decreased timing of peak velocity by 19 % (6 units). This change in timing of peak velocity was comparable to the control group that decreased by 16 % (5 units), with both groups becoming closer to the *atypical* model (**Figure 5.3b**). It was concluded from the Fixed experiment that imitation of *atypical* biological kinematics occurred as a function of an increased opportunity for sensorimotor integration and consolidation. In addition to the fixed structure, it may also be suggested that this increased opportunity for sensorimotor integration was facilitated by an increase in the amount of practice (Hannant et al., 2016). In comparison with *Chapters Two* and *Three* where participants imitated the *atypical* model per condition for 14 in 15 trials respectively, in *Chapter Four* participants imitated the *atypical* model in 40 trials. This suggestion of providing increased amounts of practice have also been forwarded to explain performance increases in a texture discrimination task. Here then, in an initial assessment the autism group scored lower than controls, however, performance in the autism group improved when repeating the task two additional times (Vandenbroucke, Scholte, van Engeland, Lamme, & Kemner, 2009).

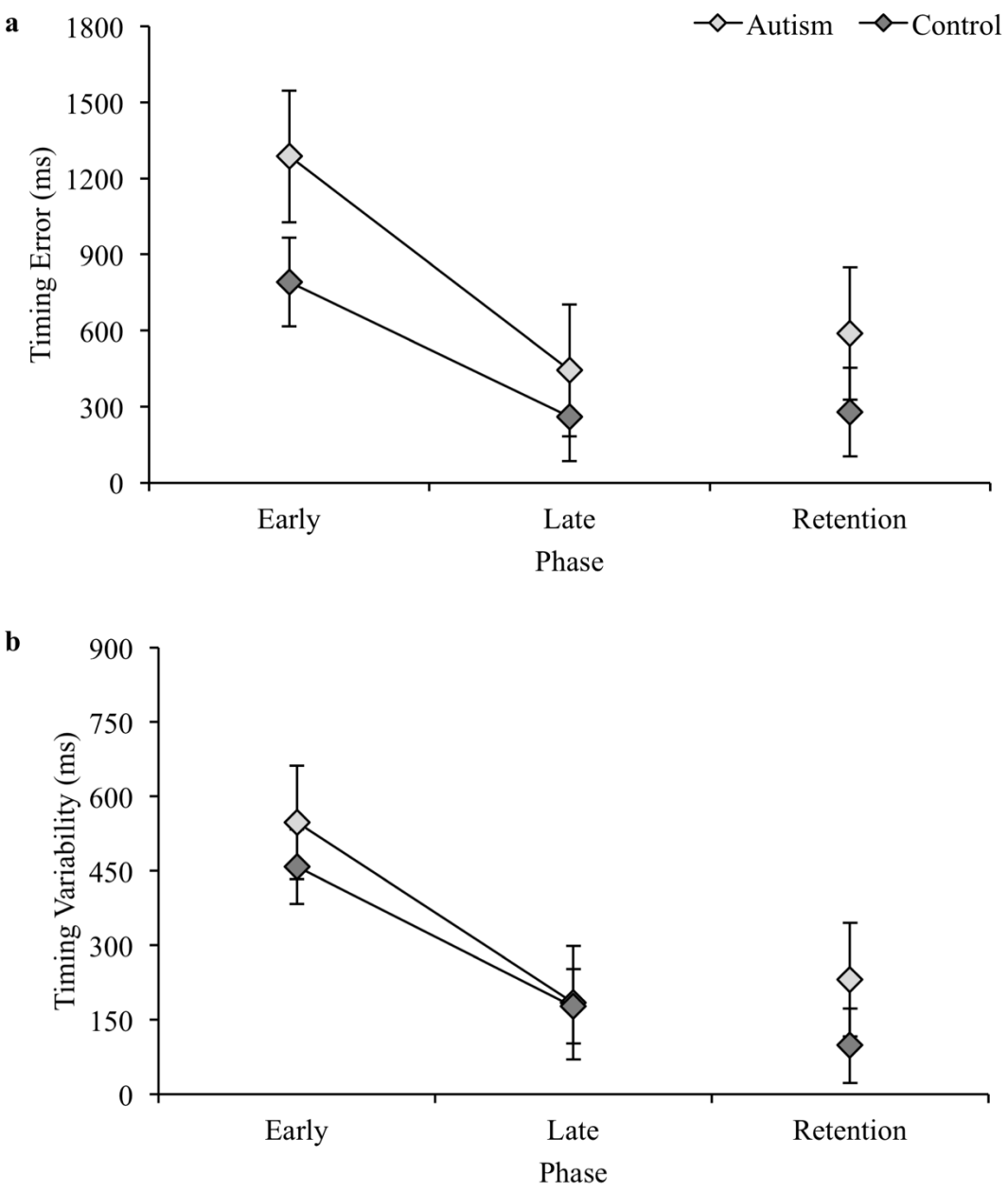

Figure 5.2 (a) Timing error and **(b)** timing variability for the motor learning task (error bars represent standard error of the mean) presented as a function of group phase (adapted from Hayes et al., under review).

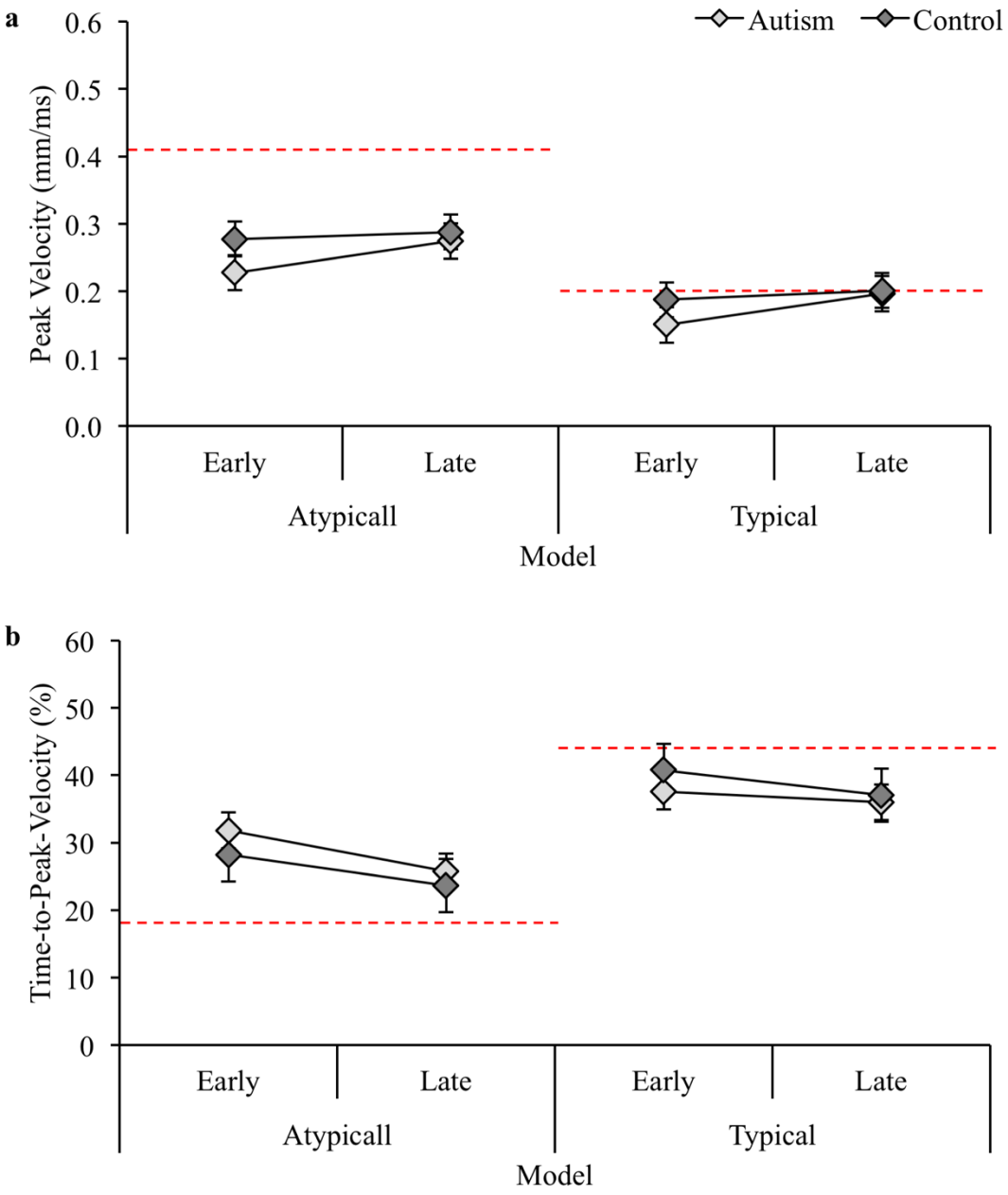

Figure 5.3 (a) Peak velocity and **(b)** time-to-peak-velocity for the imitation task (error bars represent standard error of the mean) presented as a function of group, model and phase in the Fixed experiment. The dashed-red lines in **a** represent the magnitude of peak velocity for the *typical* (i.e., 0.410 mm/ms) and *atypical* (i.e., 0.200 mm/ms) models. In **b**, they represent the time-to-peak-velocity for the *typical* (i.e., 44 %) and *atypical* (i.e., 18 %) models.

5.6 Sensorimotor Integration in Autism

An important finding from *Chapter Four* was the fact that adults with autism imitated *atypical* biological motion kinematics to a similar level of accuracy as matched neurotypical controls. After observing the *atypical* model in a context that presented the stimulus in a fixed trial order, participants in the autism group imitated movements with a time-to-peak-velocity that occurred at 27 % (SD = 9 %) of the movement trajectory (see **Figure 4.2b**). The early occurrence of peak velocity was reasonably similar to that displayed by the *atypical* model (18 %), and that of the control group (M = 25 %; SD = 7 %). Importantly, time-to-peak-velocity was significantly different from that exhibited after imitating the *typical* model (M = 37 %; SD = 12 %). The increase in imitation fidelity suggests that imitation of *atypical* biological kinematics occurred as a function of an increased opportunity for sensorimotor integration and consolidation across repeated trials of the same *atypical* model. Over repeated attempts at imitating the *atypical* model in a fixed condition it is likely the refinement of the sensorimotor representation was facilitated by increasing the opportunity to process error through comparisons between expected (e.g., a plan of the efferent and afferent visual and motor information on trial *n*) and actual (efferent and afferent visual and motor information experienced on trial *n)* sensorimotor information (Wolpert et al. 2003; Iacoboni, 2005). The reduction in error occurs online during the ongoing motor response (Elliott et al., 2001) and offline during the inter-trial delay (Schmidt, 1975; Carroll & Bandura, 1982; Kilner et al., 2007; Burke et al., 2010; Wolpert et al., 2011).

The suggestion that imitation fidelity was facilitated by integrating sensorimotor information during the inter-trial delay was supported by the findings

from the Interference experiment where participants were instructed to perform a secondary visuomotor task. The secondary task was implemented in the inter-trial delay to experimentally interfere with the processing (i.e., integration) of sensorimotor information received from trial *n*. In line with findings from *Chapter Two* (M = 33 %; SD = 10 %) and *Three* (M = 31 %; SD = 10 %) when the trial order was randomised, imitation performance in the autism group was statistically similar to imitating the *typical* model (M = 39 %; SD = 10 %), and significantly different to the neurotypical control (M = 28 %; SD = 5 %) group. Moreover, and importantly, imitation performance was qualitatively different to the autism group from the Fixed condition (M = 27 %; SD = 9 %) where the opportunity for sensorimotor consolidation during the inter-trial delay was not perturbed (Bandura, 1977; Byrne & Russon, 1998; Heyes, 2013). Therefore, these findings, and specifically the interference effects induced by the secondary visuomotor task, suggest the inter-trial delay period during imitation is an important processing phase (Buccino et al., 2004) in autism where sensorimotor information following movement execution in a fixed context is integrated to develop and refine a sensorimotor representation that supports imitation.

Similar inter-trial processing explanations have been forwarded to explain decrements in motor performance in learning studies that examined contextual interference (for a review see Magill & Hall, 1990) and motor interference during observational learning (Brown et al., 2009). For example, Shea and Morgan (1979) instructed participants to learn a three-segment movement sequence under blocked (low contextual interference) or random (high contextual interference) practice conditions. Data indicated that motor performance was more accurate in the blocked condition (similar to the trial order in Fixed experiment in *Chapter Four*), compared

to random (similar to the trial order used in *Chapters Two* and *Three*) condition. During random trial orders different sensorimotor representations (e.g., Timing Goal 1, followed by Timing Goal 2) are suggested (Li & Wright, 2000; Cross e al., 2007) to be constructed, deconstructed, and reconstructed across different trial types. The process of planning different movement action plans across inter-trial delay periods leads to an increase in sensorimotor interference that consequently impacts the fidelity of a movement representation. Therefore, although benefits in long term motor learning (see Magill & Hall, 1990) and imitation learning (Blandin, Proteau, & Alain, 1994) have been facilitated from these interference effects, it has been consistently shown that motor performance accuracy decreases, and variability increases, across random practice trials (Lee & Magill, 1983).

Moreover, the interference effect found from the secondary task in *Chapter Four* is similar to data reported from an observational learning study (Brown et al., 2009) that induced motor interference using repetitive TMS. In this study, participants observed naïve learners perform reaching movements using a robotic arm. Following observational learning, participants either received repetitive TMS to primary motor cortex, or allowed to consolidate the processing of the observed stimulus. Consistent with many studies (e.g., Vogt 1995, Hayes, Elliott, & Bennett, 2010) participants that did not receive rTMS acquired the motor pattern via sensorimotor processes underlying observational learning. Importantly, and consistent with previous work (Mattar & Gribble, 2005), learning was significantly reduced in participants that received repetitive TMS to primary motor cortex during the consolidation period. This finding demonstrates sensorimotor information is integrated and consolidated across learning in primary motor cortex, which is also known to be active during imitation learning (Nishitani et al., 2004).

With the aforementioned in mind, the Fixed experiment in *Chapter Four* most likely offered similar motor performance and learning advantages as those consistently shown during blocked practice (Li & Wright, 2000; Wright & Shea, 2001). Blocked practice structures provide an opportunity for response-produced error and variability to be reduced over similar trial types leading to a more refined sensorimotor representation (Wright & Shea, 2001). Therefore, it seems the fixed trial order allowed the underlying lower-level visuomotor processes within the autism group to be effectively engaged in order to integrate and process sensorimotor information. Specifically, creating 'consolidation periods' within the imitation learning context most likely facilitated the *atypical* sensorimotor representation to be transformed from a fragile to a relatively permanent state (Caithness et al., 2004; Wolpert et al., 2011). Moreover, because other important imitation processes associated with mentalising and social regulation were controlled through the presentation of a non-human agent model (Cook & Bird, 2011; Stewart et al., 2013) indicates that this form of motor imitation may not be fundamentally impaired in autism but rather the processes underlying this mechanism need to be operationalised in a specific imitation learning context to overcome the sensorimotor integration problems that underpin imitation in autism (Williams et al., 2006).

The interpretation of sensorimotor integration being associated with impaired imitation of biological motion kinematics in autism is consistent with a recent collection of work that has shown that individuals with autism process and integrate visual and proprioceptive information differently (Sharer et al., 2015; Nebel et al., 2015; Hayes et al., under review). When learning novel movements children with autism show a bias in the integration of sensorimotor feedback, favouring proprioception over visual feedback (Haswell et al., 2009: Marko et al., 2015).

Furthermore, the finding of children with ADHD demonstrating a learning pattern indistinct from typically developing children points towards this being autism specific (Izawa et al., 2012). This bias towards proprioceptive feedback signals has also been evidenced in a ball catching task as part of the movement assessment battery for children (Brown & Lalor, 2009). The ball catching task requires a demand for the integration of temporal and spatial characteristics of the movement that requires online adjustments for successful task completion. Work employing the ball catching task (Whyatt & Craig, 2012) showed autistic children had difficulties in performing the task when compared to matched (age) typically developing children. Notably, these difficulties were not only dissimilar to typically developing controls, but also children with ADHD (Ament et al., 2015) indicating the adaptation of motor skills that require the coupling of visual and temporal feedback operate differently in autism. It was concluded that rather than general motor abilities (Green et al., 2002), motor skill deficits in autism are suggested to be allied with the ability to integrate visual spatial and temporal characteristics of an action.

In addition to the behavioural level differences in sensorimotor integration in autism, neurophysiological work has also shown that brain activity underpinning the development of sensorimotor representations differ compared to neurotypical controls (Müller et al., 2001; Müller et al., 2003; Marko et al., 2015). For instance, using an fMRI paradigm fifty autistic, and fifty matched (age, IQ and handiness) typically developing children were scanned during three gesture imitation tasks: (1) gesture imitation; (2) gestures to verbal command; (3) gesture involving tool use. Results showed that autistic children had increased intrinsic asynchrony in neural activation between visual (lateral occipital cortex) and motor (pre- and post-central gyrus) regions which correlated with more severe autistic traits (measured via

ADOS). Furthermore, children that exhibited greater intrinsic synchrony showed greater imitation accuracy. This altered synchrony could influence the integration of visuomotor information (Nebel et al., 2015). Based on these findings given that sensorimotor representations form part of a mechanism that facilitates the processing of biological motion for action-understanding and motor-execution (Blakemore & Decety, 2001), it may be that neural specificity of sensorimotor representations regulate how subsequent observed visual information is processed in social visuomotor contexts (Nebel et al., 2015). In the context of the current thesis, it could be proposed that during the Fixed experiment these brain mechanisms (lateral occipital cortex; pre- and post-central gyrus) occurring during imitation are operating effectively due to the consistent structure of the models leading to the sensorimotor representation being refined on a trial-by-trial basis (Wolpert et al., 2011). However, in the Interference experiment these neural processes are being interfered with, resulting in asynchronic brain activity which would be similar to those reported in the autism participants.

Although the findings from the Fixed and secondary visuomotor experiments provide good evidence that sensorimotor information was integrated and consolidated in the inter-trial delay between trials, it is possible that repeated exposures to the model in the Fixed experiment also allowed an opportunity to enhance the fidelity of the sensorimotor representation by engaging learning processes during action-observation. For example, sensorimotor representations are developed and refined without receiving response-produced sensory feedback from an effector (Mattar & Gribble, 2005; Brown et al., 2009). This form of learning is referred to as observational practice, which requires a learner to watch a model across a consecutive number of demonstrations without engaging in overt physical

practice (Maslovat, Hayes, Horn, & Hodges, 2010). It is now well accepted that novel actions, and underlying kinematics (i.e., magnitude and timing of peak velocity), can be learned by observational practice to the same extent as physical practice (Vogt, 1995; Mattar & Gribble, 2005; Hayes et al., 2010; 2012; Hayes, Elliott, Andrew, Roberts, & Bennett, 2012; Hayes, Elliott, & Bennett, 2013; for a review of observational practice see Vogt & Thomaschke, 2007). For example, in a study by Hayes, Timmis and Bennett (2009), participants either physically practised or observed a three-segment movement sequence to achieve one of three movement time goals. The data indicated the experimental groups learned the timing goals by increasing accuracy, and reducing variability, across practice. These findings suggest that in Fixed experiment increased imitation fidelity exhibited by the autism group may have been facilitated by engaging lower-level visuomotor processes that underlie learning through observational practice (Higuchi at al., 2012). This suggestion is further supported by data demonstrating that atypical biological motion is represented through observational practice (Andrew et al., 2016).

The findings of similar motor learning performance without engaging in overt physical practice (Hayes et al., 2009; 2010; 2012; 2013; 2014) are now known to occur through common underlying neural (i.e., mirror neuron) systems (Fadiga et al., 1995; 1996; Rizzolatti et al., 1996; Buccino et al., 2004; Calvo-Merino, Glaser, Grèzes, Passingham, & Haggard, 2005; Torriero, Oliveri, Koch, Caltagirone, & Petrosini, 2007; Vogt et al., 2007). For instance, fMRI data showed that similar changes in motor behaviour following a period of physical and observational practice of dance actions were underpinned by analogous activity in the premotor and parietal regions of the action observation network (Cross, Kraemer, Hamilton, Kelley, & Grafton, 2009). Furthermore, a more recent study has illustrated that

observational practice and imitation also share similar neural substrates (Higuchi et al., 2012). Using a fMRI paradigm naïve participants were required to imitate finger position of pictured guitar chords. Results indicated that successful imitation was underpinned by activity within the dorsolateral prefrontal cortex (DLPFC) as well as the frontoparietal mirror circuit. The second of two studies examined whether this neural circuits are also recruited during observational learning, or only physical practice. Again naïve participants observed new guitar chords yet were not required to immediately imitate. fMRI data illustrated that although prefrontal cortex activity was not constant in observational practice, prefrontal activation was correlated with behavioural practice effects indicating a crucial role of the prefrontal cortex in observational practice (Higuchi, Holle, Roberts, Eickhoff, & Vogt, 2012).

Further support that the sensorimotor representation is refined through action-observation comes from behavioural work that shows interference of observational practice effects through simultaneously completing a secondary motor task during action-observation (Mattar & Gribble, 2005). Here then, participants observed naïve learners perform reaching movements using a robotic arm. This robot was programmed to perturb the upper-limb dynamics by applying force fields to the learner's arm in a clockwise or counter-clockwise direction. When required to perform the action in a clockwise (i.e., retention test), those that observed the clockwise condition (i.e., congruent to practice) performed better than those that did not have the opportunity to practice (i.e., no observation). In contrast, those that observed the counter-clockwise condition (i.e., incongruent to practice) performed worse that the no observation group. Still, these positive effects of observational practice on reaching performance where attenuated when participants observed the counter-clockwise while simultaneously executing a secondary (i.e., incongruent arm

movement) motor task (Mattar & Gribble, 2005). It was concluded by the authors that the ability of visual information that drives motor learning through systems linking action with perception when the motor system is interfered with by the generation of unrelated movements. Though work examining observational practice in autism is limited, the suggestion that increase in imitation fidelity is associated with action-observation through repeated exposures to the model is supported by evidence of lower-level processing through automatic imitation in autism (Bird et al., 2007; Leighton et al., 2008; Press et al., 2010; Sowden et al., 2016). Such as when participants with autism were required to perform finger movements in response to a number or coloured square while observing congruent or incongruent finger movements, automatic imitation effects (faster responses when the observed action was congruent, rather than incongruent) exhibited were similar to matched neurotypical controls (Sowden et al., 2016). These findings indicate that the lower-level visuomotor processes that map the visual information onto the motor system (Iacoboni et al., 1999; 2001; Buccino et al., 2004) are operating effectively during automatic imitation.

5.7 Concluding Remarks

5.7.1 Limitations and Future Considerations

5.7.1.1 Human Stimuli

The experimental protocol used within the present thesis was designed in order to create an unmodulated condition that limited the influence of top-down factors that modulate lower-level processing of biological motion. As discussed in

the introductory chapter one such factor is social interaction. It has recently been suggested that impaired imitation in autism is associated with evaluation of social context (Southgate & Hamilton, 2008; Wang & Hamilton, 2012). Indeed, when primed with pro-social attitudes neurotypical adults exhibit greater levels of automatic imitation than when primed with anti-social attitudes (Cook & Bird, 2011). In comparison, adults with autism that do not show this modulation (Cook & Bird, 2012). Therefore, to control for the modulatory effects of social interaction, participants observed and subsequently imitated a non-human agent (white-dot) that had limited social context. By doing so, the findings reported in the present thesis cannot be directly comparable with those of imitation in human settings (Hobson & Hobson, 2007; Vivanti et al., 2008; Wild et al., 2012; Vivanti & Dissanayake, 2014). However, using an experiential protocol similar to that used in the present thesis, the influence of social interaction on imitation of biological motion in autism could be examined. Here then, using a stylus on a digital graphics tablet, participants with and without autism would observe and subsequently imitate *typical* and *atypical* biological motion kinematics (presented in a fixed presentation as per *Chapter Four*) following observation of non-human (i.e., a single white-dot; similar to the present thesis) or a human model (i.e., finger movement; similar to Wild et al., 2012). It could be expected that consistent with the findings reported in the Fixed experiment in *Chapter Four* (M = 27 %; SD = 9 %), when presented with a non-human stimulus in a blocked structure, adults with autism would show reasonably high-fidelity imitation of the *atypical* model, similar to matched neurotypical controls (M = 25 %; SD = 7 %). If then the processes underpinning impaired imitation in autism are associated with the social context (Spengler et al., 2010; Wang & Hamilton, 2012), then it could be expected that when presented with a human stimulus (i.e., human

hand), imitation fidelity in autism would be attenuated. Adults with autism would exhibit kinematics similar to those reported in *Chapters Two* (M = 33 %; SD = 10 %) and *Three* (M = 31 %; SD = 10 %). If, however, impaired imitation is not attributed to altered social top-down factors, then it could be expected that adults with autism would show no differences when imitating the human compared to non-human stimulus.

5.7.1.2 Autism Severity

There is a large body of evidence gathering demonstrating that imitation abilities in individuals with autism positively correlates with the severity of their disorder (Stewart et al., 2013; Nebel et al., 2015). For example, twenty-five autistic children that were evaluated for autism using the Autism Diagnostic Observation Schedule (ADOS) completed four imitation tasks (body movements and 'action on objects', using meaningful and non-meaningful tasks). Imitation abilities in all four tasks significantly correlated with autism severity where children with higher ADOS scores (indicating lower functioning) had lower imitation abilities (Zachor, Ilanit, & Itzchak, 2010). Consequently, additional correlation analyses were conducted within the current thesis to investigate whether the imitation dependent measure (i.e., timing accuracy and variability, magnitude and timing of peak velocity) in the present thesis correlated with autism severity (i.e., ADOS total score). These correlations revealed no relationship between any dependent variable and ADOS total score indicating that imitation abilities exhibited by the autism participants were not a consequence of the autism characteristics calculated via ADOS. The current thesis primarily examined only high-functioning individuals with autism which only represents a small proportion of individuals with autism. Many individuals with low-functioning

individuals have learning difficulties and other cognitive implications in addition to the core characteristics of autism which may modulate imitation of biological motion in the present thesis. Though these cognitive implications may influence imitation performance using the protocol in the present thesis. This protocol could be modified in order to accommodate and investigate imitation of atypical biological motion in low-functioning autistic children and/or adults.

5.7.1.3 Volunteer Age

In the present thesis imitation of biological motion was examined in autistic adults only. Although on the whole the adults with autism were of a relatively young age (*Chapter Two* = 26.4 years; *Chapter Three* = 21.7 years; *Chapter Four* = 21.7 years), these findings observed are difficult to directly compare to previous studies examining imitation in children with autism (e.g., Rogers et al., 1996; Hobson & Lee, 1999; Hamilton et al., 2007; Vivanti et al., 2008; Stewart et al., 2013). It is difficult to postulate whether similar findings would be observed in children with autism. Recent work has demonstrated that imitation performance is correlated with chronological age in both adolescents with autism (Stewart et al., 2013) as well as typically developing children (Williams et al., 2014). For instance, in a study by Stewart et al. (2013), participants were required to imitate different sized shapes using a stylus on a tablet. Results showed that although overall adolescents with autism aged between 11 and 17 years were less accurate than matched typically developing adolescents, a correlation analysis showed that as age increased the imitation error decreased for all shape sizes. As a matter of interest consistent with these studies (Stewart et al., 2013; Williams et al., 2014) in each experimental chapter the analyses for each dependent variable (timing error; timing variability;

peak velocity; time-to-peak-velocity) was re-run with age as a covariate. Similar to other work (Smith & Bryson, 2007) no effects of interest were found with respect to the age in adults with autism. Furthermore, in respect to imitation in children with autism, future work could use a similar protocol to examine imitation of biological motion.

5.7.2 Translational Research and Practical Recommendations

As a final point, the results across the present programme of work has translational research potential to promote social rehabilitation therapies in autism. Previous work has shown that video modelling is an effective tool when teaching children with autism social engagement (i.e., active participation in an activity with a peer; Bellini, Akullian, & Hopf, 2007), socially expressive behaviours such as gestures and facial expressions during social interaction (Charlop, Dennis, Carpenter, & Greenberg, 2010) as well as improving imitation skills (Cardon & Wilcox, 2011; Cardon, 2013). For instance, recent a recent study examined whether there is a relationship between imitation skills in four children with autism (2 male; 2 female) and caregiver implemented Video Modelling Imitation Training through the use of an iPad. Following minimal training (2 hours each) four caregivers were able to successfully create video models and implement Video Modelling Imitation Training that occurred three times a week for a total of 12 sessions (40 minutes). During each session the caregiver showed the child a pre-recorded clip of one-step actions (e.g., touch their nose, hand cup to caregiver) and the autistic child was given 10 seconds to imitate the action they had observed. Results indicated that children with autism significantly increased imitation performance (percent of actions imitated) across the 12 sessions which was not only maintained during the post-

intervention (one and three weeks after final session) but also to varying degrees' imitation following live modelling. In addition, analysis of language development after experience with the Video Modelling Imitation Training revealed expressive language skills increased for all participants (Cardon, 2012). These findings clearly demonstrate the positive influence of video modelling for social interaction in individuals with autism (Lindsay, Moore, Anderson, & Dillenburger, 2013; Cardon, 2016). In the context of the findings of the current thesis, more specifically the kinematic data in the Fixed experiment in *Chapter Four* where adults with autism imitated high-fidelity of *atypical* biological motion to the same extent as neurotypicals (**Figure 4.2b**), it could be suggested that the acquisition in everyday sensorimotor skills such as writing with a pen, tying shoes laces, or riding a bicycle could be acquired through the presentation of non-human agent models using video modelling in a fixed (i.e., predictable) structure, as this increases sensorimotor integration and consolidation, leading to greater imitation performance.

5.7.3 Summary

In conclusion, the present thesis examined imitation of biological motion in adults with autism, and to investigated whether adults with autism could adapt and learn to imitate and represent biological motion following specific manipulations to the imitation context (e.g., instructions, feedback, practice type). The thesis utilised a novel behavioural protocol which required adults with autism and matched (age, gender, handiness, IQ) neurotypical controls to observe and subsequently imitate models that displayed movements that had identical spatial and temporal outcomes, yet displayed distinctly different, but biological plausible kinematics.

Chapter Two extended upon previous studies (Wild et al., 2012; Stewart et

al., 2013) that manipulated speed or amplitude of a movement by isolating lower-level properties (e.g., velocity) of biological motion kinematics. As detailed in **Figure 2.3**, for the first time experimentally it was demonstrated that that adults with autism have difficulties imitating the velocity characteristics associated with *atypical* biological motion that ensured that the observer must configure the sensorimotor system to represent the novel kinematics. As though both groups performed similarly at imitating *typical* biological kinematics, but the control group was significantly more accurate than the autism group at imitating the *atypical* biological kinematics. Nonetheless, compared to neurotypical adults, adults with autism became significantly more accurate at representing movement time across trials thus illustrating that they actively engaged in the imitation task. This suggests the attenuation in imitating biological motion kinematics in autism is perhaps a compensatory strategy due to deficits in lower-level visuomotor processes associated with self-other mapping and/or motor ability, or that selective attention input to the processes that represent atypical biological motion kinematics.

Chapter Three investigated whether the impaired imitation findings in *Chapter Two* could be underpinned by altered visual attention. Here, selective-attention and eye movements in autism and controls were examined when imitating *atypical* and *typical* biological kinematics. To manipulate selective-attention, general instructions not specifying what aspects of the model to imitate were provided in the control phase. In the experimental phase, selective-attention instructions directed visual attention towards biological kinematics. As detailed in **Figure 3.3**, in the general-attention condition, kinematic data illusatred that both groups performed similarly at imitating *typical* biological kinematics, but the control group was more accurate than the autism group at imitating the *atypical* biological kinematics.

Moreover, as detailed in **Figures 3.8** and **3.9,** eye movement data showed the autism group had similar timing and peak smooth pursuit eye velocity, as well as a similar number, and amplitude, of saccades, during action-observation of both models as the control group. With selective-attention instructions, imitation of *atypical* biological kinematics remained unchanged, with the control group more accurate than the autism group. Only the control group became more accurate when imitating the *typical* biological kinematics. Eye movements were again similar between the groups, and modulated to become closer to the model after receiving selective-attention instructions. Adults with autism still have difficulties imitating t *atypical* biological kinematics, yet they are unlikely to be underpinned by difficulties tracking the model with the eye, and thereby the focus of visual attention. The lack of modulation following explicit instructions suggests altered imitation in autism could be associated with differences in 'input' modulation, where processes associated with attention do not effectively control lower-level sensorimotor processes that encode biological motion.

Chapter Four examined sensorimotor integration. In the Fixed experiment the models in a fixed structure which provides opportunity for the sensorimotor representation to be refined by updating error using and actual sensory consequences from trial *n* over similar trial types. It was demonstrated for the first time experimentally that adults with autism could imitate velocity characteristics associated with *atypical* biological motion to the same extent as matched neurotypicals. This suggests that imitation of biological motion in adults with autism may be associated increased sensorimotor integration and consolidation through presenting the model in a predictable structure. In the Interference experiment the models were presented in a fixed structure where participants completed a secondary

motor task during the inter-trial delay. It was demonstrated that adults with autism show difficulties imitating the velocity characteristics associated with *atypical* biological motion. This suggests that this increased sensorimotor integration and consolidation that results in imitation of *atypical* biological motion similar to matched neurotypicals may be taking place during the inter-trial delay. In the Random experiment the models in an unpredictable structure (similar to *Chapter Two*). It was demonstrated again that adults with autism show difficulties imitating the velocity characteristics associated with *atypical* biological motion. This finding confirms the suggestion that low fidelity imitation of *atypical* biological kinematics in autism is attributed to complications in integrating sensorimotor information on a trial-by-trial basis where there is opportunity for consolidation, or the previously reported problems in planning and execution occur due to the unpredictable presentation structure.

Overall, the results have extended the imitation in autism literature, have had both theoretical and practical implications, and provided a catalyst for future research within the area. Providing a strong suggestion that imitation in individuals with autism spectrum disorders is associated with the integration and consolidation of sensorimotor information.

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