Sensorimotor Processes Underpinning Imitation Learning of Biological Motion

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II. Contents

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Hayes, S. J., Dutoy, C. A., Elliott, D., Gowen, E., & Bennett, S. J. (2016). Atypical biological motion kinematics are represented by complimentary lower-level and top-down processes during imitation learning. *Acta Psychologica. 163.* 10-16.

V. Abstract

The aim of the present thesis was to examine the way in which biological motion is coded and imitated during imitation learning by improving upon methodologies currently used in the literature to examine imitation of underlying movement kinematics. Across four experiments, imitation of the kinematic structures of biological and non-biological motion models was examined to investigate the processes involved in imitation learning. The purpose of the first experimental chapter, *Chapter Two,* was to examine the way in which biological motion kinematics were coded during imitation learning by establishing whether imitation of biological motion kinematics was a function of lower-level visuomotor processing or top-down attentional modulation. Results showed that not only were imitations of typical and atypical biological motion different, but both models were imitated as accurately during spatially incompatible trials as compatible. Accurate imitation of spatially incompatible atypical biological motion confirmed biological motion coding is a function of lower-level visuomotor processing.

Following results from *Chapter Two, Chapters Three, Four* and *Five* assumed lower-level visuomotor processing of biological motion and were designed to further examine whether this lower-level visuomotor processing of biological motion was modulated by top-down attentional factors (e.g. end-state-targets, visual attention, social primes). The first of these top-down modulations was included in *Chapter Three, which* examined the influence of end-state-targets on biological motion coding during imitation learning. Although kinematics was not modulated by end-state-targets, movement time was less accurate when end-state-targets were present, which suggests that lower-level and top-down processes operate together during the processing of visual information during imitation learning. In addition to end-state target modulation, imitation data further confirmed the coding of atypical biological motion

by demonstrating differences in imitation of two relatively similar atypical biological motion models (atypical17 and atypical26). The top-down attentional factor examined In *Chapter Four* was visual attention, which was measured by recording eye movements during observation of the model stimuli. Analysis of eye movements demonstrated that visual attention was directed towards the model throughout the entirety of the observation phase during trials where end-state-targets were both present and absent. As goal-directed eye movements were not made during observation of the models, results suggest that the kinematic data contained within each of the models was observed and consequently featured in the representation formed for motor execution.

Chapters Two, Three and *Four* provide a fundamental understanding of how biological motion is coded during imitation learning by using robust protocol that improves upon the validity of those used in the current literature and specific modulations that discredit significant top-down modulatory explanations for biological motion coding. The way in which biological motion coding occurs in neurotypicals (no neurologically atypical patterns of thought or behaviour) is important when trying to understand where deficiencies in those with intellectual disabilities occur. The intellectual disability most closely associated with the current thesis is autism, where deficiencies in imitation are suggested to be linked to social components. Therefore, to establish a foundational understanding of how social context influences neurotypical imitation, *Chapter Five* examined the influence of social primes on the coding of biological motion. Results showed that social primes modulated the accuracy of imitation, where peak velocity was more like those of the models following observation of an anti-social prime. In addition, observation of both the pro- and anti-social primes was shown to reduce the variability of imitation relative to observing no social prime at all. These findings demonstrate that social primes are being coded and incorporated into the motor output such

that both the accuracy and consistency of imitation of biological motion are modulated. Together, the results presented in the current thesis demonstrate imitation of novel, atypical biological motion is a function of complimentary lower-level and top-down processes that facilitate the coding of both underlying kinematics and environmental context.

Chapter 1: Exploring the Processes Underpinning Biological Motion Coding During Imitation Learning

1.1 Aim of the Chapter

The following introductory chapter outlines the rationale and aims of this thesis, providing an overview of imitation of biological motion during imitation learning, as well as a review and discussion of the current theories relating to the factors that may modulate this process. In addition to discussing the current understanding of biological motion coding within existing literature, the introductory chapter will also outline how the methodology used in the present thesis will progress and expand upon this understanding to provide a more rigorous examination of biological motion coding during imitation learning and how that impacts wider research areas such as Autism Spectrum Condition (ASC).

1.2 Imitation

1.2.1 Definition of Imitation

Imitation is an important learning strategy for humans (van Gog, Paas, Marcos, Ayres & Sweller, 2009) and is largely defined as a method of acquiring novel actions or behaviours (Heyes, 2001). Research examining children's abilities to imitate suggest that imitation is one of the earliest forms of reciprocal interaction between infant and caregiver (Nadel, Guerini, Peze & Rivet, 1999) and facilitates the learning of fundamental abilities such as language and social skills (Adams, 1987). Successful imitation involves observing an action and coding the visual information into a representation that contains any perceived goals or means (the way in which the movement was executed i.e. movement velocity), such that an appropriate motor

response can be generated voluntarily when required (Blake, 1958; Bandura & Huston, 1961; Bandura, 1986; Heyes, 2001, 2011; Heyes et al., 2005). Imitation thus enables an individual to improve their response accuracy (termed 'motor control'; Wolpert, Doya, & Kawato, 2003) through a sensorimotor loop (Wolpert & Ghahramani, 2000) by updating the representation based on the afferent (i.e. visual; proprioceptive) and expected sensory signal information. This loop is continuous, as is the updating of the representation, such that prolonged exposure to the model combined with repeated imitation attempts results in reduced error as the motor response becomes more like the model (Schmidt, 1975; Bandura, Ross & Ross, 1963; Carroll & Bandura, 1982). It has been suggested that observation and imitation are part of a system in which they are linked by a common representational domain (Prinz, 1997), wherein visual information is processed using higher-order top-down (cognitive) and lower-level (visuomotor) mechanisms (Byrne & Russon, 1998; Brass & Heyes, 2005). Lower-level mechanisms refers to the direct engagement of neural processes through the stimulation of sensory receptors (Teufel et al., 2010), whereas top-down mechanisms elicit motor activity based on decomposed stimulus features such as goals or environmental context e.g. attention/ instruction/ social context (Csibra, 2007).

1.2.2 Importance of Imitation

While imitation is important in acquiring and developing motor skills (e.g. holding a fork, throwing a ball), it is also intricately linked with social interaction and the development of social skills such as facial expressions (Piaget, 1962), hand gestures (Fontaine, 1984; Vinter, 1986) and other socio-cognitive skills (Meltzoff & Decety, 2003) from infancy (Meltzoff & Moore, 1999). Imitation of these skills was initially found in infants between 8-

12 months of age (Piaget, 1962), however it was later discovered that imitation could be observed at an earlier age (Meltzoff & Moore, 1977; Heimann, Nelson & Schaller, 1989). This led to the question: what function does imitation at such an early age serve? Initially, the answer was 2-fold: the imitative responses were either 'reflexive' or a display of 'social cognition'. In terms of the former account, the suggestion was that a displayed model automatically triggers a preset motor program where the response is involuntary and unplanned (Bjorklund, 1987). The latter account suggests that infants interpret the intermodal equivalence, i.e., correlating a model's display with one of their own (Harvey & Johnston, 1973), such that imitation is used for social-communication purposes (Meltzoff & Moore, 1985). Further research on this suggestion found that infants used imitation to enrich their understanding of persons and actions used for communicative purposes, as well as to identify people by familiarising themselves with nonverbal behaviours to create identity (Meltzoff & Moore, 1992).

Adults, like infants, use imitation as a learning tool (van Gog et al., 2009). However, adults are also able to use imitation as a social medium to appear more affable by creating positive connections or a sense of familiarity. This was demonstrated when observers were shown two loosely related images and asked to highlight the similarities they shared (van Baaren, Janssen, Chartrand & Dijksterhuis, 2009). Participants who were being mimicked by an examiner reported a greater number of similarities between the images than those who were not being mimicked, suggesting that imitation had the power to change the way people think and perceive situations. As well as influencing perception, imitation has also been shown to increase behaviours such as rapport (Chartrand & Bargh, 1999), trust (Bailenson & Yee, 2005), memory (van Baaren, Horgan, Chartrand & Dijkmans, 2004), enjoyment (Tanner, Ferraro, Chartrand, Bettman & van Baaren, 2007), and likeability (Jacob, Guegeun, Martin &

Boulbry, 2011; Herrmann, Rossberg, Huber, Landwehr & Henkel, 2011; Stel, Mastop & Strick, 2011). Equally, a lack of imitation has been reported when people have a dislike or dissimilarity to those they are observing based on criteria such as obesity (Johnston, 2002), religion (Yabar, Johnston, Miles & Peace 2006) and attractiveness (van Leeuwen, Veling, van Baaren & Dijksterhuis, 2009) to name a few. Thus, if people want to disaffiliate with others, they are likely to imitate them less.

1.2.3 Types of Imitation

While the concept of imitation may seem straightforward due to its recognisability in day-to-day life, there are various types of imitation that are underpinned by different, and very complex, mechanisms. Therefore, it is important to consider the characteristics of imitation that are most relevant to this program of research and the processes that define and facilitate each type.

1.2.3.1 Emulation

Emulation is a goal-centric type of imitation in which the observer learns something about the environment but not about the behaviour of another (Tomasello et al., 1987). Emulation changes the saliency of certain goals such that during observation, the purpose or goal to which the demonstrator is striving is made obvious by its actions and as a result becomes the goal for the observer as well (Tomasello, 1996). The means by which the goal is achieved may be the same as those observed, however it is a matter of individual learning and not fundamental to emulation. Further, it has been suggested that goal emulation contains

elements of cognitive priming, where, providing goals are either familiar or identifiable, they are primed in the brain and are addressed before unprimed ones (Byrne, 1998). For example, studies have shown that chimpanzees who observed demonstrators collecting out-of-reach food with a rake, then used a similar tool themselves to obtain food and importantly, appeared to do so without using the same technique as the demonstrators (Call & Tomasello, 1994; Nagell, Olguin & Tomasello, 1993; Tomasello, Davis-Dasilva, Camak & Bard, 1987). By using dissimilar techniques but primarily obtaining the food, authors suggested the effects were a demonstration of emulation.

1.2.3.2 Simple Imitation

Simple imitation; also, termed 'mimicry' (Tomasello, 1996), 'automatic imitation' (Heyes et al., 2005), 'priming' and 'response facilitation' (Byrne & Russon, 1998), occurs when an observer copies the movements or actions of another that already exist as part of the observer's behavioural repertoire (Heyes, 2011). This is commonly seen in social interactions, where preconscious mimicry (Dijksterhuis & Bargh, 2001) of facial, vocal or postural cues (Hess, Philippot & Blairy, 1999) has been observed, e.g. ear touching (Bekkering, Wohlschlager & Gattis, 2000). However, simple imitation is considered dissimilar to emulation (Tomasello, 1996). An example of the distinction was demonstrated in a study that required observation of a demonstrator who touched their ear using ipsilateral (same side of the body) or contralateral (opposite side of the body) arm movements (Bekkering et al., 2000). When a demonstrator touched their right ear with their left hand, the goal of the movement was to touch the left ear and the contralateral arm movement defined the way in which the goal was attained. In this instance, emulation of the movement would have been to touch the right ear

regardless of which hand was used, whereas imitation of the movement required not only the touching of the right ear, but also the contralateral arm movement. Whilst the focus of this thesis is imitation learning (complex imitation), not automatic imitation (simple imitation), automatic imitation research is regularly referenced due to the commonalities it shares with complex imitation in relation to the underlying mechanisms, as outlined by the ASL theory (Heyes & Ray, 2000), that mediate imitation (Heyes, 2005).

1.2.3.3 Complex Imitation

Complex imitation, also termed 'imitation learning' (Tomasello, 1996), 'true imitaiton' (Zentall, 2006; Cook & Bird, 2012), 'observational learning' (Carroll & Bandura, 1982), occurs when an observer copies a novel sequence of movements. Importantly, these are movements that are not already part of the motor repertoire (Heyes, 2011). Throughout the thesis, the common term used will be imitation learning. Imitation learning is believed to depend on complex psychological processes whereby visual information from a model is translated into a motor output that looks the same. Like simple imitation, imitation learning is concerned with imitation of both the goal and the way in which the goal was obtained. The important distinction though, is that because the to-be-imitated movement is novel, it requires an element of learning, e.g. performing a semaphore-like movement with the hand and arm (Carroll & Bandura, 1982). As such, it is initially unclear how the cognitive system works out which potential action corresponds to the one that was observed as it is one that will not have been performed before (Bird & Heyes, 2007). Moreover, a recent review article of imitation posited that the processes underpinning the early stages of imitation learning are 'far from

obvious' due to the complexities and unknowns regarding the available solutions (Heyes, 2011).

1.2.4 Theoretical Models of Imitation

1.2.4.1 EP-M Model

The EP-M model is a dual route model concerned with how actions are processed and is like other dual route models proposed for imitation of actions (Tessari & Rumiati, 2002), language processing (Lichtheim, 1885) and stimulus-response compatibility, subsequently referred to as SRC (Heyes, 2011). The EP-M model is associated with the perception-action system and is made up of two components: the 'EP' and the 'M' routes (Hamilton, 2008). The EP route is involved in understanding, planning and achieving the goal of an observed action, which is generated using pre-existing sensorimotor representations (e.g., picking up a fork), which are scaled relative to the task-demands. The EP route is suggested to originate in the middle temporal gyrus (MTG) and ends at the inferior frontal gyrus (IFG) travelling via the inferior parietal lobule (IPL), the region that is associated with identifying the goal of an action. The M route is involved in the processing of movement kinematics and makes direct associations between the visual and motor components, termed 'visuomotor mapping', such that automatic mimicry is feasible without cognitive interpretation. It is generally used when observing actions that are novel, or are lacking any obvious goals, e.g. accuracy of imitation. The M route is also suggested to originate in the MTG, however visual information is sent directly to the IFG, leading to direct associations between visual and motor representations. Often during observation, these routes are used together to provide a complimentary understanding of the goal of the movement and the way in which the goal was obtained

(Hamilton, 2008). A similar dual route model was generated in relation to SRC (Heyes, 2011) that discussed an intentional and automatic route in relation to stimulus-response association. The intentional route was suggested to generate short-term stimulus-response association between the sensory and motor representations based on task instructions that generally lasted for the duration of the task (Barber & O'Leary, 1997). Conversely, the automatic route is modelled as a long-term stimulus-response association between the sensory and motor representations, which has recently been shown to be a function of learning (Oostenbroek, Suddendorf, Nielsen, Redshaw, Kennedy-Costantini, Davis et al., 2016).

1.2.4.2 Goal-Directed Theory

Goal-directed imitation refers to a type of top-down modulation that is suggested to influence the direct visuomotor mapping associated with lower-level processing. Broadly speaking, goal-directed imitation has largely been examined in terms of meaningful (MF) and meaningless (ML) actions (Rumiati & Tessari, 2002; Press & Heyes, 2008), or transitive (object-related) and intransitive (non-object related) actions (Bekkering et al., 2000; Press, Bird, Walsh & Heyes, 2008). While MF and ML actions are not examined directly in the current thesis, their examination and subsequent contribution to goal-directed imitation is noteworthy. Imitation accuracy (Rumiati & Tessari, 2002) and reaction time (Press & Heyes, 2008) are improved following observation of MF (goal-directed) compared with ML (not goal-directed) actions, which authors have postulated are a function of a dual-route model of imitation (Rumiati & Tessari, 2002). The dual-route model proposed a direct route for imitation of unknown actions, which relies on direct visuomotor mapping of an action and a semantic route for imitation of known actions that utilises long-term memory. MF actions are

stored in the long-term, semantic memory and therefore engage the semantic route, while ML actions could only be reproduced through the direct route as they had no pre-stored goals in the semantic memory. It was suggested that the less accurate imitation of ML actions was a result of using the direct route, as it bypassed the semantic route and created greater demands on the short-term and working memory systems (Rumiati, Weiss, Tessari, Assmus, Zilles, Herzog et al., 2005).

Another interpretation of goal-directed imitation suggests that goals are embedded within observed and executed actions (Bekkering et al., 2000). It posits that the motor representation constructed for imitation is decomposed during observation of an action into its constituent components and later reconstructed based from these components. Importantly, this process is guided by the viewer's perception and interpretation of the motor program in relation to its goal-directed features. If a tangible goal is present, it is generally considered to be of more importance than the way in which the goal is achieved. For instance, touching the ear is more important than using the correct ipsilateral (i.e. left hand to touch left ear) or contralateral (i.e. right hand to touch left ear) arm movement to touch the ear (Gordon, 1920; Head, 1923; Schofield, 1976; Bekkering et al., 2000). However, if there was no obvious goal of a movement, e.g. moving a limb into a space (Bekkering et al., 2000; Wild, Poliakoff, Jerrison & Gowen, 2010), then the way in which the movement was performed would likely be recognised as the primary goal. Within the interpretation of goal-directed features, GOADI suggests that goals are represented as a hierarchical structure where specific goals are encoded as having greater importance (Bekkering, Wohlschlager & Gattis, 2000; Wohlschlager, Gattis & Bekkering, 2003). Consequently, the goals that are prescribed greater importance are likely to generate a greater accuracy of imitation. The goals that are selected then elicit the motor program with which they are most strongly associated and therefore, do not necessarily lead

to matching movements. Consistent with the EP route of the dual route model (Hamilton & Grafton, 2006; 2007), neuroimaging research examining the mirror neuron circuitry has demonstrated observation and imitation of goal-directed tasks activate parietal regions of the brain, specifically the IPL (Rizzolatti, Fogassi & Gallese, 2004; Iacoboni, 2005), which are controlled and regulated by the prefrontal cortex, subsequently referred to as PFC (Quintana & Fuster, 1999; Miller, 2000).

1.2.4.3 Associative Sequence Learning

The associative sequence learning (ASL) theory suggests imitation occurs because the sensory and motor representations generated during observation and imitation are connected by direct 'vertical associations' that are highly experience-dependent (Heyes, 2001). Some of these vertical associations are innate, that is, executed autonomously without cognition, e.g. yawning, smiling (Meltzoff & Moore, 1977). However, the majority are formed because of correlated experience of observation and execution of the movement, where humans use self-reference after imitating to gauge the accuracy of the representation. As well as direct vertical associations, there are also indirect vertical associations between the sensory and motor representations, whereby the representation is mediated by an additional representation, e.g. a word or phrase. The indirect association can occur during the observation or execution of the movement, which then informs the direct association. Vertical associations characterise sensorimotor representations and the execution of an imitative movement may be a combination of multiple representations. These connections are made through horizontal associations to form a sequence or sensorimotor representations. It is suggested that the acquisition of novel motor skills assumes that activation of motor representations via vertical

associations provides input to task general processes of motor learning, and results in imitative performance unless inhibited by mechanisms regulating intentional action. These mechanisms then connect the vertical associations to an output in relation to goals that formulate the execution of imitation (De Renzi, Cavalleri & Facchini, 1996).

1.2.4.4 Direct-Matching Hypothesis

The direct-matching hypothesis has been suggested to operate using a 'resonance' mechanism (Rizzolatti, Fadiga, Fogassi & Gallesse, 1999), whereby the visual information observed (e.g. a picture; movement kinematics) is mapped onto a motor representation of the same action in the nervous system (Rizzolatti, Fogassi & Gallesse, 2001). Moreover, this hypothesis predicts that cortical areas where matching occurs must contain neurons that discharge during observation and that some of them should also receive input based on the action they are encoding. Therefore, these areas should have motor properties that are likely to be more active when the imitation is primed by observation of the to-be-executed action (Iacoboni, Woods, Brass, Bekkering, Mazziotta & Rizzolatti, 1999).

Several theories have been outlined in this review of current literature to demonstrate the complex and specific processes underpinning the ability to imitate. It is important to note that no one theory is entirely correct, or incorrect – depending on the feature of imitation that is examined, each can assume varying applicability. Therefore, the current thesis is not designed to corroborate one particular theory, but to consider all theories and their relevance to the findings discussed throughout. That withstanding, certain theories are expected to be more relevant to the current thesis based on the research questions examined. The primary

research question in the current thesis concerns whether atypical biological motion is coded during imitation learning. If results demonstrate, as expected, that atypical biological motion is coded and imitated differently to typical biological motion, these findings would support the ASL theory by evidencing direct associations between the observed and executed movements to produced different visual representations formed during observation that resulted in scaled imitation of the respective models.

1.3 Perception-action System

The perception-action system is believed to contain the neural circuitry that is active during imitation learning. The term 'mirror' is a reference to the connection between neuron activation during observation and imitation respectively; that is, regions of the brain believed to activate during observation also activate during imitation of the same stimulus. The first example of these 'mirror neurons' was discovered in monkeys (di Pellegrino, Fadiga, Fogassi, Gallese & Rizzolatti, 1992), specifically in the IFG located in the F5 region, and forms a circuit with the posterior parietal area, also containing neurons with mirror properties (Gallesse, Fadiga, Fogassi & Rizzolatti, 2002; Fogassi, Ferrari, Gesierich, Rozzi, Chersi & Rizzolatti, 2005). Subsequently, there have been many neuroimaging studies examining the perception-action system in the human brain. However, it is still uncertain which regions of the brain 'consistently contribute' to observation and imitation processing (Caspers, Zilles, Laird & Eickhoff, 2010). Early neuroimaging studies involving humans suggested mirror neuron activation was in the inferior frontal (Iacoboni, 2001), premotor and parietal (Decety, Chaminade, Grezes, Meltzoff & Grezes, 2002; Buccino et al., 2004) regions of the brain. Yet,

a more recent review of 20 studies found discrepancies based on the regions that were consistently activated (Molenberghs, Cunnington & Mattingley, 2009). The first strong evidence of a perception-action system in humans was found using motor evoked potentials (MEPs) that were activated by single-pulse transcranial magnetic stimulation (TMS) while observing upper-limb movements (Fadiga, Fogassi, Pavesi & Rizzolatti, 1995). The study found that activation of MEPs during observation of another person's movement were similar to those recorded during execution of the same movement. Neuroimaging studies have also reported a mirroring effect between observation and imitation in tasks such as observing goal-directed hand-grasping movements (Gangitano, Mottaghy & Pascual-Leone, 2001; see Rizzolatti & Craighero, 2004), playing guitar chords (Vogt, Buccino, Wohlschlager, Canessa, Shah, Zilles et al.., 2007) and compatible finger movements (Biermann-Ruben et al., 2008).

Further research using functional magnetic resonance imaging (fMRI), electroencephalography (EEG), magnetoencephalography (MEG) and TMS methods, indicated a contribution from the primary motor cortex (Nishitani & Hari, 2002; Jarvelainen, Schurmann & Hari, 2004) and the dorsal premotor cortex (Grezes, Armony, Rowe & Passingham, 2003; Leslie, Johnson-Frey & Grafton, 2004) in the mirror-circuitry due to their activation during action observation. In addition to the core circuitry, it had also been suggested that other regions of the brain not containing mirror properties are active during action observation. For example, the posterior part of the superior temporal sulcus (pSTS) has been shown to act as a visual relay to the IPL and frontal lobe structures (Nishanti & Hari, 2002; Iacoboni et al., 2001). Specifically, it was suggested that the visual information provided by the pSTS to the fronto-parietal mirror regions facilitates goal-directed information processing in the IFG and ventral premotor cortex (vPM), as well as kinematic

information processing in the rostral part of the inferior parietal lobule (aIPL) (Iacoboni, 2005).

More recently, it has been posited that the frontal regions of the perception-action system code the kinematics, whereas the parietal regions code the goal(s) of an observed movement (Hamilton, 2008). Moreover, it has also been suggested that these regions not only code the kinematics or goal of an observed movement, but also contribute to the understanding of actions and their consequences (Hamilton & Grafton, 2008). This was demonstrated using a repetition suppression (RS) protocol to measure blood oxygen level-dependent signal (BOLD), which highlights active regions during visual information processing, after viewing videos of novel or repeated kinematics, goals and outcomes. A selection of RS studies have reported suppression of the lateral occipital cortex (LOC) and IFG when constructing the visual and motor representations of kinematics (Hamilton & Grafton, 2007; Kilner, Neal, Weiskopf, Friston & Frith, 2009), as well as in the IPL when processing the goals or outcomes of a movement (Hamilton & Grafton, 2008; Chong, Cunnington, Williams, Kanwisher & Mattingley, 2008). However, RS studies have also indicated that while these regions are active, they may not in fact contain neurons with mirror properties. Rather, motor areas have distinct, segregated populations of visual and motor neurons where the visual neurons discharge during observation and the motor neurons fire during imitation (Dinstein, Thomas, Behrmann & Heger, 2008). It was proposed that if mirror neurons existed in humans, they should adapt at a synaptic level based on repetitive exposure to the same information through common pathways (Sawamura, Orban & Vogels, 2006). However, it was suggested that adaptation effects were difficult to interpret as common pathways are often lacking (Bartels, Logothetis & Moutoussis, 2008). For example, frontoparietal regions receive visual information during observation from the superior temporal sulcus (STS) (Rizzolatti &

Luppino, 2001; Iacoboni, 2005), whereas during imitation it mostly comes from the frontal lobes (Fuster, 2008). Moreover, this inference of separate neural regions involved in visual and motor processing is unlikely based on the volume of literature demonstrating congruency between the regions and neurons involved in observing and imitating motor acts (Fadiga et al., 1995; Strafella & Paus, 2000; Maeda, Kleiner-Fisman & Pascual-Leone, 2002). Therefore, if visual information reaches neurons that encode the same motor act such that imitation is achieved, the neurons involved in these processes have mirror properties (Rizzolatti & Sinigaglia, 2010).

1.4 Biological Motion

1.4.1 Definition of Biological Motion

'Biological motion' refers broadly to the movement of an animate object. Relative to human movement, it refers more specifically to body movements (e.g. hands, eyes, face) that may provide information about specific actions or intentions (Allison, Puce & McCarthy, 2000). The original methodology designed to measure biological motion processing used a rudimentary version of point-light displays (PLD), which allowed for studying of the movement without interference from the form (Johansson, 1973). It was suggested that 10-12 light bulbs located on the major joints in the human body, observed against a contrasting background (often in a darkened room) were sufficient to be able to convey enough visual information to be able to distinguish between the highly specific movement patterns that comprise actions such as walking, running and dancing (Johansson, 1973). The theory is founded on the basis of physics principles, which state the motion of a single point is

characterised by its position and the forces applied to it. That is, the movement of a single point, as well as it's interaction with the other visible points within a PLD, contain enough relative information to represent motion structure. It is suggested that recognition occurs due to the implicit recognition that the visual information follows the laws of human motion. One law of human movement is the minimum jerk (MJ) velocity profile, which describes a 'bell-shaped' velocity curve during a point-to-point movement (e.g. drawing a horizontal line across a piece of paper). A MJ velocity profile starts with a slow acceleration from a stationary position where the magnitude of acceleration gradually increases until it reaches a peak velocity (typically between 40%-60% of the movement displacement; Elliott, Helsen & Chua, 2001), before a gradual decrease in magnitude of velocity as the movement reaches its conclusion (Abend, Bizzi & Morasso, 1982; Flash & Hogan, 1985). Relative to the current thesis, the MJ velocity profile is representative of one of the model data that was observed by participants as it complies with the laws of human motion. That is, it is a movement that humans make implicitly when moving between two points (Abend et al., 1982).

1.4.2 Importance of Biological Motion

The functional implications of being able to detect biological motion have developed over time. From an evolutionary standpoint, it has been shown to facilitate the identification of predators, prey and those of one's own kind (Ewert, 1987). Specifically, within humans, observation of biological motion has allowed for the distinction between animals and humans (Mather & West, 1993), genders (Kozlowski & Cutting, 1977), identities/ familiarity (Cutting & Kozlowski, 1977; Troje, Westhoff & Lavrov, 2005), facial expressions (Bassili, 1978) and actions (Dittrich, 1993) to name a few. These effects are largely consistent when observing

static or dynamic biological motion. However, it has been suggested that the depth of movement recognition is greater when observing dynamic biological motion (Mather & Murdoch, 1994; Troje, 2002). Importantly, the ability to process observed biological motion to infer actions and intentions (termed 'social perception'; Allison et al., 2000) is a function that defines humans and can be identified in infants as early as days old (Simion, Regolin & Bulf, 2008; Meary, Kitromilides, Mazens, Graff & Gentaz, 2007). Four-month-old infants were reported to stare at human motion sequences for longer duration than at random movements (Bertenthal, 1993; Fox & McDaniel, 1982). While it has been suggested that infants may process biological motion in a similar way to adults (Jokisch, Daum, Suchan & Troje, 2005; Reid, Hoehl & Striano, 2006), it has also been shown that children improve their abilities to perceive and process biological motion until they reach adult levels of performance at approximately age 5 (Pavlova, Krageloh-Mann, Sokolov & Birbaumer, 2001). Being socially cognisant is a feature that allows humans to thrive in complex social situations and is the cornerstone of human life (Amodio & Frith, 2006).

1.4.3 Neural Mechanisms and Application of Biological Motion Processing

In the adult brain, multiple areas are required for biological motion processing (for reviews, see Allison et al., 2000; Puce & Perrett, 2003) such as inferior occipital cortex (Bonda, Petrides, Ostry & Evans, 1996; Pelphrey, Mitchell, McKeown, Goldstein, Allison & McCarthy, 2003; Dayan, Casile, Levit-Binnun, Giese, Hendler & Flash, 2007), lingual gyrus (Vaina, Solomon, Chowdhury, Sinha & Belliveau, 2001; Pelphrey et al., 2003; Dayan et al., 2007), premotor cortex (Saygin, Wilson, Hagler, Bates & Sereno, 2004; Saygin, 2007) and primarily, the STS (Bonda et al., 1996, Pelphrey et al., 2003; Saygin et al., 2004; Safford,

Hussey, Parasuraman & Thompson, 2010). Each of these areas is suggested to process different components of the visual information such as motion and form (Vangeneugden, de Maziere, van Hulle, Jaeggli, van Gool & Vogels, 2011). However, the STS is considered the primary area associated with biological motion processing and is believed to integrate all components of biological motion processing to construct a complete visual representation of an observed movement (Puce, Allison, Bentin, Gore & McCarthy, 1998; Vangeneugden, 2011). STS activation in response to observed human movement was first discovered using single cell recordings in macaque monkeys (Oram & Perrett, 1994). More recently, neuroimaging studies have substantiated and elaborated upon this finding to show that it is specifically the pSTS that is most active when observing human movement, as demonstrated in PLD paradigms (Bonda et al., 1996; Grossman & Blake, 2002; Grossman, Blake & Kim, 2004). However, the breadth of regions that appear to be responsive to observed biological motion have meant it is unclear whether they are responding to general human movement or if they are specifically linked to biological motion (Troje, 2008). Nevertheless, neuroimaging studies spanning 20 years have sought to find definitive answers.

Some of the first studies on humans used positron emission tomography (PET) during observation of biological PLDs, such as hand-grasping actions and whole body movement. Increased activity was reported in the STS when observing biological motion compared to a non-biological control conditions that showed random motion (Bonda et al., 1996; Grossman et al., 2000) or scrambled motion (reconfigured biological PLD motion; Grossman et al., 2000). As well as PET, TMS has been used to temporarily disrupt cortical activity in specific areas of the brain. Applying repetitive TMS (rTMS) over the right pSTS prior to completing a biological motion discrimination task showed a decrease in detection of biological motion, suggesting normal function of the pSTS is required for the perception and processing of

biological motion (Grossman, Battelli & Pascual-Leone, 2005). Moreover, TMS has impaired biological motion coding when applied to the STS relative to control regions of the brain not associated with biological motion coding (vertex) and decreased sensitivity in the STS when observing biological, compared to non-biological (scrambled biological) motion (van Kemenade, Muggleton, Walsh & Saygin, 2012). These finding underscore the unique involvement of the STS in the processing of human movement.

More recently, neuroimaging studies have used fMRI as it is considered safer than PET and is more appropriate and accurate for determining the contribution from areas of the brain than TMS. Early fMRI studies demonstrated activations in portions of the STS region when observing facial movements, e.g. directed/ averted eye gaze that were both dynamic (Puce et al., 1998) and static (Haxby, Hoffman & Gobbini, 2000). The study using dynamic eye movements displayed human models that appeared to make eye movements from a central fixation to either the right or left side and then return to a central fixation while the head stayed in register. These eye movements were shown alongside clips of the same length, demonstrated by the same models, which demonstrated the eyes maintaining a central fixation. Brain activity in the STS region, specifically the pSTS was greater during clips where the eyes made a movement to either side, compared to when they maintained a fixation, which suggested the STS was active in the perception of biological motion contained in certain body movements (Puce et al., 1998). While the same conclusions were reported when using static images of eye movements and gaze direction, it was suggested that in addition to the perception of eye movements and gaze direction, the STS is also involved in higher-level representations of actions (Haxby, Hoffman & Gobbini, 2000), specifically recruiting the spatial-cognitive system to encode the direction in which eye movements or gaze were focussed. This is concurrent with a recent repetition suppression fMRI study that used the

repetition suppression (RS) design during observation of PLDs and suggested the STS generates higher-level representations of biological motion during action coding (Grossman, Jardine & Pyles, 2010). RS is based on the theory that consecutive activations of the same neuronal population create a reduced haemodynamic response compared to consecutive activations of different neuronal populations (Buckner et al., 1998; Grill-Spector & Malach, 2001). The experiment consisted of observation of pairs of images; the first animation was of a PLD showing one of twenty-five action sequences, e.g. walking, running etc., and the second was either (a) the same animation repeated, (b) a PLD of a different animation, or (c) the same animation mirror-reversed. Results showed that activation in the pSTS was suppressed after observation of the same animation and of the mirrored animation but not after observing different animations. The implication is that regions of the STS not only detect biological motion, but also generate higher-level representations to form a more complex understanding of actions and movements.

In addition to the wealth of neuroimaging research demonstrating a connection between the STS region and the perception of biological motion, this association is made even more robust by the implementation of the theory in behavioural studies that observe interactive performance (Saygin, 2007; Lange & Lappe, 2006; van Kemenade et al., 2012). One example of biological motion processing affecting behavioural performance was demonstrated when participants performed linear arm movements while concurrently observing orthogonal arm movements. Online imitation of the vertical human arm movement produced involuntary movement deviation in the execution of horizontal arm movements whereas observation of the vertical robot arm movement did not. The suggestion is that the observation of incompatible biological properties to those of execution create an interference effect during the processing of the visual information (Kilner, Paulignan & Blakemore, 2003).

This effect has also been replicated when observation was of a robot arm reproducing biological motion (Chaminade, Franklin, Oztop & Cheng, 2005), inferring it is the underlying nature of the movement, not the form in which the movement is presented, which creates the interference. This was recently corroborated when a curvilinear arm movement was included in addition to the same vertical and horizontal arm movements used in the above studies (Roberts, Hayes, Uji & Bennett, 2015). Although no interference was reported during observation of the congruent, horizontal arm movements, observing the curvilinear arm movement produced a significant interference resulting in vertical deviations when trying to execute the horizontal movements. As the end-points of the movement were the same, authors concluded that the interference was a result of the observed biological motion being different to that being executed and represented the overlap in processing of different representations of biological motion. These perceived interference effects during online imitation have been termed 'motor contagion' (Blakemore & Frith, 2005) based on the theory that during observation, the observed biological properties directly activate a corresponding representation in the observer's motor repertoire.

Similarly, during imitation learning, observers have been shown to imitate movement kinematics such as peak velocity and differences in velocity of biological motion performed by both human (Wild, Poliakoff, Jerrison & Gowen, 2010) and non-human (Bisio Stucchi, Jacono, Fadiga & Pozzo, 2010) agents. Moreover, the imitation of biological motion kinematics appears to be more accurate when environmental context facilitates 'true imitation' such that there is no social or goal-based context (Iriki, 2006; Wild et al., 2010). It has been suggested that the imitation of movement kinematics is representative of the ability to code kinematic markers repeatedly during observation. This temporal coding has been demonstrated using MEPs during single TMS pulses while observing a reaching-grasping

action (Gangitano et al., 2001). The TMS pulses were administered relative to specific kinematic landmarks linked to certain phases of the reaching-grasping movement such as maximal finger aperture. It was reported that in addition to coding the features or goals of biological motion to form a motor plan, this process also automatically codes visual information relative to the temporal features, which accounts for the ability to code kinematic features such as changes in velocity. These findings have all been amalgamated in a recent study that showed biological motion kinematics are coded during imitation learning and moreover, and that imitation is more accurate when there are no target goals present during the task (Hayes, Dutoy, Elliott, Gowen & Bennett, 2016). Participants were tasked with imitating novel biological motion stimuli with different kinematic profiles relative to peak velocity and the time at which peak velocity occurred (percentage-time-to-peak-velocity). There were two atypical biological motion models wherein the peak velocities were greater and occurred earlier than 'normal' and a non-biological constant velocity model. Imitating accurately but also relative to the respective models, showed participants could distinguish between different types of biological motion to accurately code and imitate specific features of the movements. This finding suggests the coding of biological motion involves lower-level sensorimotor systems to produce high-level, complex representations of observed visual stimuli.

In the review of imitating biological motion, there is relative consistency within neuroimaging research that identifies certain regions of the brain that are activated when identifying biological motion (STS) and coding the underlying kinematics of an observed stimulus (IFG, mPFC), compared to coding the cognitive, attentional features of the environmental context e.g. goals (IPL). Although there appears to be a general consistency in

how the visual information is processed, the way in which the visual information influences physical imitation seems more varied. For example, there is evidence to suggest that spatial compatibility between observation and imitation of transitive hand movements is both required (Sturmer et al., 2000; Brass et al., 2001) and not required (Brass et al., 2005) to facilitate successful imitation of the observed action. While there are not always behavioural discrepancies in the research, inconsistencies in behavioural research provide an insight into how complex imitation can be.

Similarly, it is difficult to identify any right or wrong regarding the theories that are suggested to underpin imitation of biological motion. Each theory discussed previously has been experimentally proved and disproved, which further suggests the environmental context can heavily influence how people imitate certain features of an observed stimuli. Instead, these discrepancies allude to the complexities involved in imitating that may, by definition, not be explained by one single theory. Instead, it is predicted that the various modulations included in each of the experimental chapters may corroborate different theories of imitation relative to the purpose of the experiment e.g. the end-state-target modulation in *Chapter Three* may confirm GOADI in the context of goal-directed imitation of biological motion kinematics during imitation learning. While the experiments examining top-down modulations may support their corresponding theories, the theory behind coding of biological motion kinematics is predicted to be more straightforward. The ASL theory suggests that direct associations between the visual and motor representations are formed during processing of the visual stimulus, which facilitates accurate imitation. Therefore, in line with ASL theory, it is expected that imitation of novel atypical biological motion will be scaled to the model and different to that of the other models used in the respective experiments e.g. typical biological motion/ constant velocity.

1.5 Other Factors That May Modulate Imitation

While the effect goals, or the absence thereof, on imitation have been alluded to above (see Goal-directed Theory of Imitation; pg 9), it is important to discuss some of the other factors that influence the processes underlying imitation. While there are numerous factors that could be discussed, those addressed below are central to some of the studies presented in this thesis, namely spatial compatibility, visual attention and social primes.

1.5.1 Spatial Compatibility

SRC refers to the situation where the selection of a response is directly related to the position of the related stimulus. When the relationship between stimulus and response is natural and direct, it is described as natural. When it is unnatural and indirect, it is described as incompatible (Proctor & Vu, 2006). Basic understanding of this subject has used simple tasks involving finger pressing of keys. Faster responses were generated in both horizontal (left and right; Proctor & Reeve, 1990) and vertical (top and bottom; Chan, & Chan, 2005) axes when the stimulus was compatible, compared with incompatible, to the response required, e.g. left light stimulus paired with left button rather than right button response. From an imitation perspective, spatial SRC protocols have been used to extensively to explore action representation and control, seminal among which were the Simon effect (1969) and spatial Stroop effect (1935).

In a Simon task, the relevant stimulus is a non-spatial physical feature (e.g. colour, shape) that is assigned to left or right manual responses often controlled by key-presses, and

the location in which the stimulus occurs (left or right) is irrelevant. A spatial Stroop task is largely the same, however the relevant stimulus is a word or feature that conveys spatial information, e.g. the word "LEFT". Measurements are often recorded in relation to reaction time and error scores, where compatible and incompatible trials are directly compared. In both the Simon and spatial Stroop tasks, responses are faster when the stimulus appears at a congruent position (e.g. "LEFT appearing on the left side of the screen), than at an incongruent position. More recently, this effect has been examined in automatic imitation where protocols based on the original spatial Stroop and Simon tasks were used. Largely, they were choice reaction time SRC protocols where the cues were photographic images of human models performing the actions required in the response set. For example, Sturmer, Aschersleben and Prinz (2000) examined hand-opening and hand-closing. As the hand started to either open or close, the hand would change colour to signal either the opening (blue) or closing (red) of the observer's hand as quickly as possible. Results showed that reaction time was quicker when the stimulus hand was compatible with the correct response (e.g. open hand stimulus to open hand response), compared to when the stimulus and response were incompatible (e.g. close hand stimulus to open hand response). Similar results have been demonstrated when lifting movements of the index or middle finger were examined (Brass, Bekkering, Wohlschlager & Prinz, 2000). A number would appear to signify the lifting of either the index ("1") or middle ("2") finger, which coincided with the lifting of either the index or middle finger of the stimulus hand. Again, reaction time was quicker when the stimulus finger was compatible with the correct response e.g. index finger stimulus and index finger response.

While similar studies (Gillmeister, Catmur, Liepelt, Brass & Heyes, 2008; Bach & Tipper, 2007; Leighton & Heyes, 2010) have contributed to robust findings regarding this automatic imitation effect within SRC protocols, the protocols themselves were not designed

such that the effects could be definitively attributed to imitation. Relevant to observation of body movement, the term "imitation" is often used to describe actions that are topographically similar (Heyes, 2001). However, it is possible that the tendency to produce a topographically similar response, e.g. "imitatively compatible" (Catmur & Heyes, 2011), could be confused with the tendency to respond in the same relative position as the stimulus, e.g. "movement compatible" (Brass, Bekkering & Prinz, 2001). For example, observing a hand move quickly to a target could prompt the response to also move quickly to a target (imitatively compatible) but it could also have prompted the response to simply reach the target and thus finish the movement in the same location (spatially compatible) regardless of speed (Wild et al., 2010). The bigger issue however, is that when considering a multitude of action types, such as power/ precision grip (Chong, Cunnington, Williams & Mattingley, 2009), index finger movements (Brass et al., 2000; Brass et al., 2001; Bertenthal, Longo & Kosobud, 2006), and hand and/ or mouth opening/ closing (Heyes et al., 2005; Press et al., 2008; Leighton & Heyes, 2010), it is not always possible to dissociate imitation effects from spatial compatibility effects.

1.5.2 End-state-targets

The theory underpinning meaningfulness and transitive goal-directed imitation was discussed earlier in the chapter. This section will discuss some of the research that incorporated end-state-targets as the specific transitive goal, designed to induce top-down modulation of imitation. When end-state-targets are present, they provide a tangible, achievable goal that defines the movement and acts as a definitive form of completion of an imitation attempt. The goal-directed theory of imitation suggests that when there are obvious

goals of a movement, these goals are incorporated into the to-be-used motor representation and moreover, arranged within a hierarchy based on their perceived importance.

One of the most thoroughly examined paradigms used to explore this theory required participants to imitate a model making either ipsilateral or contralateral arm movements to touch their ears (Head, 1920; 1926; Gordon, 1923; Schofield, 1976; Bekkering et al., 2000). In these instances, touching the ear represented the goal of the trial and was deemed the 'end-state-target' that concluded the imitation attempt. Results have consistently shown more ipsilateral than contralateral responses are produced, regardless of the way in which the model demonstrated the task, to touch the correct ear as demonstrated by the model. The most recent of these studies (Bekkering et al., 2000) extended these findings by including two further manipulations. Firstly, only one ear was used as an end-state and thus reduced the number of objects at which the arm movements were directed. This resulted in more correct arm movements being made during imitation. Secondly, an additional trial type was included where the model made the same ipsilateral or contralateral arm movements, but in addition to touching the ear and thus being goal-directed, also moved to a nondescript space by the side of the head. There trial types were classified as being goal-less, and reduced the preference to make predominantly ipsilateral arm movements. Instead, the type of arm movement used by the model in goal-less trials was more accurately imitated. Results primarily corroborated previous research that showed that imitation is mediated by goals. They also showed that when multiple goals are available, they are organised into a perceived hierarchy that inform what elements of an action are imitated. Further, if there are no obvious goals present during observation, the way in which the action is achieved becomes the primary goal and is thus imitated with more accuracy.

The structure of goal-directed imitation was further explored using a modified version of the classic SRC protocol mentioned earlier in the chapter (Brass et al., 2000). Participants were shown clips of a finger movement that produced either a biomechanically possible (flexion) or impossible (hyperextension) movement but were not given instruction as to their physical nature (Longo et al., 2008; Experiment 1). Even though participants were largely aware of the biomechanical differences between the movements, comparable automatic imitation was elicited from both actions, which suggested the actions were coded in relation to their respective goals (e.g. tapping a surface) rather than their constituent movements. In a subsequent study (Longo et al., 2008; Experiment 2), participants were informed they would see both possible and impossible actions prior to completing the same task. Results indicated that the automatic imitation effects from Experiment 1 were only found during imitation of biomechanically possible actions. In line with previous suggestions (Bekkering et al., 2000), the authors agreed that goal-directed coding appears to be the default response when generating imitative responses. Further, these results suggest that the proposed hierarchy of goals appears to incorporate additional top-down factors such as attention and instruction.

In addition to automatic imitation, top-down modulation has also been examined relative to imitation learning and more complex movements. For example, participants were required to imitate a model that moved between two points and displayed different movement kinematics (Wild et al., 2010). The kinematics represented either ‘fast’ or ‘slow’ speeds and were displayed either between beginning and end-state-targets (goal-directed), or two nondescript points in space (goal-less). Results showed that the presence of end-state-targets modulated the accuracy of kinematic imitation such that it was less accurate compared with when there were no-end-state-targets present. In line with GOADI, these results suggest that goals have a modulatory effect on imitation and moreover, that these top-down modulations

apply to all forms of imitation. Further, they demonstrate that in the absence of any obvious goals during observation, the way in which a movement is completed becomes the primary goal of the movement, as demonstrated by the more accurate imitation of movement kinematics during goal-less trials.

1.5.3 Visual Attention

Visual attention is defined by two basic phenomena: limited capacity for processing information; and selectivity. Limited capacity refers to amount of information available on the retina that can be processed to influence behaviour, while selectivity is the ability to filter out unwanted information (Desimone & Duncan, 1995). Taken together, these phenomena suggest that at some point between input and response, objects in the visual input compete for representation, analysis or control (Treisman, 1960; 1993). This competition has been demonstrated several times in experiments where two objects are presented in the visual field that require property identification of both stimuli, with separate responses for each. These types of experiments highlighted several important facts: dividing attention almost always results in poorer performance than focussing attention on one object (Duncan, 1984); interference and subsequent performance limitation only occurs when multiple stimuli are presented simultaneously (Duncan, 1980); interference is independent of eye movements (e.g. fixation or periphery) and spatial separation (Sagi & Julesz, 1985). Once there is competition in the visual field, the next issue is how selectivity is coordinated across the different systems so that a target object is selected for perceptual and spatial analysis as well as for motor control.

Selectivity refers to the ability to screen out unwanted or irrelevant information, but this process is complex. Depending on the difficulty of filtering out a non-target, responses such as reaction time can be affected by as much as 50 ms (Treisman & Gelade, 1980), although this number varies continuously depending on the task (Treisman & Gormican, 1988). The biased competition model (Desimone & Duncan, 1995) suggests a bottom-up bias can influence selectivity where features such as inhomogeneity (Sagi & Julesz, 1984), new objects (Jonides & Yantis, 1988) or objects that are larger, brighter or faster (Treisman & Gormican, 1988) are naturally biased towards. However, this natural, instinctive bias would be impractical unless there was also a way to bias the visual competition towards whatever is relevant to current behaviour. That is, bottom-up, stimulus driven biases need top-down attentional control. Because the spectrum of behaviourally relevant input can be so broad, it is argued that some form of description of the information is required to control the competitive bias in the visual system such that matching inputs are favoured by having attention directed towards them (Wolfe, Cave & Franzel, 1989).

Attention refers to the ability to focus one's cognitive resources on information (e.g. motion, goals), which from an imitation perspective could subsequently influence performance. While it has been demonstrated that intentionally directed attention to a stimulus or feature is not a requirement to imitate (Leighton & Heyes, 2010), there is evidence to suggest that intentionally mediated attention and feature selection can influence the magnitude of imitation effects. For example, when required to imitate an index/ middle finger tapping motion, instructions slowed reaction time down when it was believed the movement being observed was either impossible (Longo, Kosobud & Bertenthal, 2008) or represented non-biological motion (Longo & Bertenthal, 2009). When instructions did not mention any stimulus features, both possible and impossible, biological and non-biological motions

produced an effector compatibility effect. Using the same paradigm, instructions also modulated imitation based on the belief of the origins of the observed stimuli (Liepelt & Brass, 2010). When observing a gloved human hand, those who were told the hand was wooden showed smaller imitation effects than those who were told the hand was human. In all three instances, authors concluded that any reduced imitation effects (impossible, non-biological motion, wooden hand) could have been a result of attention being on the kinematics of the stimuli, rather than the end-point of each of the movements. Importantly, even when attention was not directed to a specific feature of the stimuli, some feature of the observed stimuli was acknowledged as the goal (i.e., the end-point of the movement). This demonstrates that while attention can be directed to purposefully allude to task-relevant or irrelevant information for experimental purposes, attention can also be non-consciously self-focussed to imitate or achieve a goal (Chartand & Baugh, 1999).

Neurophysiological studies have shown that relevant and irrelevant items to task goals both enhance and suppress cortical activity respectively (Reynolds & Chelazzi, 2004). For successful top-down modulation to occur, attention should be focussed on task-relevant stimuli while irrelevant distractions are ignored (Gazzaley & Nobre, 2012). This subsequently activates the 'control' regions of the brain, such as the PFC and the parietal cortex (Curtis & D'Esposito, 2004; Gazzaley & D'Esposito, 2007), both of which are known to be involved in information processing within the perception-action system. Moreover, the ability to observe and then imitate a task is dependent on certain stages of processing and neural representations, which are also linked to attention e.g. expectation, encoding, maintenance and retrieval. These stages contribute in some part to forming a representation that facilitates imitation, although expectation and encoding are the key contributors, and all are enhanced when attention is specifically directed (Gazaley & Nobre, 2012).

The expectation phase arrives before any visual stimuli is visible. Indeed, specifying where or what to attend to can enhance perceptual performance, most notably to improve the speed or accuracy of a response (Posner, 1980). Pre-stimulus enhancement has been demonstrated in regions of the brain associated with visual information following cues that attend to task features such as location (Kastner, Pinsk, De Weerd, Desimone & Ungerleider, 1999) and stimulus features (Giesbrecht, Weissman, Woldorff & Mangun, 2006). Demonstration of the latter effect was shown in an automatic imitation study that presented pre-cues of either a finger-tapping or finger-lifting movement prior to having to execute a finger tap movement (Brass, Bekkering & Prinz, 2001). Results showed that when the stimulus features of the pre-cues were task-relevant (finger tap) as opposed to task irrelevant (finger lifting), reaction time was significantly quicker and more accurate.

The encoding phase follows the presentation of any visual stimulus, where the information is initially processed to construct a representation of what has been observed, usually in terms of any goal-related tasks (see GOADI; Bekkering et al., 2000). Goal-related tasks can occur at any point during the observation of stimuli (Hillyard, Vogel & Luck, 1998) and have been shown to activate specific regions of the brain associated with goal-directed attention (visual association cortex; Gazzaley, Cooney, McEvoy, Knight & D'Esposito, 2005). For example, identifying goals early in movement observation (within 200 ms of stimulus onset) can improve performance (Gazzaley, 2011). Still, optimal performance requires the addition of filtering irrelevant information, thus allowing for increased attention to the task-relevant information (Zanto & Gazzaley, 2009). This was demonstrated by children who were required to imitate a model that made goal-directed and goal-less movements (Bekkering et al., 2000). The goal-directed task was to imitate a model who touched their ear using either ipsilateral (same side e.g. left hand touched left ear) or contralateral (e.g. left hand

touched right ear) arm movements. The goal-less task used the same arm movements but instead of touching an ear, the model moved the hand to the space at either side of the head. Results showed that when children were required to touch their ears (make a goal-directed movement), they generally did so using ipsilateral arm movements regardless of how the model had executed the task. This demonstrated identification of goals within an observed stimulus (touching the ear) and the filtering of irrelevant information (how the ear was touched) to focus attention on what was perceived as the goal of the movement.

1.5.4 Social Primes

Social interaction in humans is ubiquitous and often involuntary or spontaneous (van Baaren et al., 2009), but as with attention it can influence imitation. Social interaction is complex and dynamic. It defines humans (Hari & Kujala, 2009) and is conveyed through multiple verbal and non-verbal behaviours. It is widely understood in social psychology that humans will communicate through these unintentional social interactions that form the basis of relationships. For example, people have exhibited imitative behaviours connected to postures, gestures, facial expressions, emotions and language (Chartrand & van Baaren, 2009). Social psychology suggests that imitation has positive social consequences on social interaction whereby liking and affiliation are created between those involved (Chartrand & Bargh, 1999) through processes linked to the perception-action system (Catmur, Gillmeister, Bird, Liepelt, Brass & Heyes, 2008; Catmur, Walsh & Heyes, 2009; Heyes, 2011). The perception-action system creates a direct link between perception and action where the sensory input automatically activates a motor response (Brass & Heyes, 2005), which has led

to the suggestion that this link is formed through ASL (Heyes, 2001; Catmur et al., 2008; Catmur et al., 2009).

While imitation is often subconscious and involuntary, there is also the theory that imitation within social interaction may be strategically implemented to change the social context for self-advancement, termed the social top-down response modulation theory (STORM; Lakin & Chartrand, 2003), or perhaps to simulate theory of mind (Pickering & Garrod, 2004; Gallese, 2006). In relation to the latter, the suggestion is if I were to imitate someone, that gives me a better understanding of their feelings or intentions. The STORM theory suggests when I consciously choose to imitate another person, they unconsciously detect my behaviour such that they positively change their attitude towards me. Therefore, if it was beneficial for someone to like me, I could imitate them more to improve my social standing. This has been evidenced in studies demonstrating greater liking (Chartrand & Baugh, 1999), feelings of closeness (van Baaren, Holland, Kawakami & van Knippenberg, 2004), trust (Bailenson & Yee, 2005) and levels of persuasion (Maddux, Mullen & Galinsky, 2008) between people who imitate strangers.

From a neurological standpoint, the STORM model has two core components: visuomotor mapping and a top-down modulation system. The visuomotor mapping contains a series of connections running from higher-order visual systems through the inferior parietal cortex to premotor and motor cortices (Cisek & Kalaska, 2010). Generally, for imitation to occur, connections are formed between visual and motor representations after actions are observed and performed (see ASL theory; Heyes, 2011). While imitation is a very natural and instinctive response (e.g., seeing an action automatically activates the ability to imitate in the perception-action system), the tendency to imitate must obviously be restrained to avoid over-imitating. Therefore, the suggestion is that social imitation is governed and inhibited by top-

down control from regions in the prefrontal lobes (Teufel, Fletcher & Davis, 2010). For example, patients with prefrontal lesions have difficulty with tasks that require the inhibition of dominant responses, e.g. go/ no-go paradigm and will often over-imitate, displaying traits of echolalia or echopraxia (Lhermitte, Pillon & Serdaru, 1986). Similarly, the mPFC and TPJ display stronger activations when the natural tendency to imitate something incongruent needs to be suppressed (Wang, Newport & Hamilton, 2011; Kampe, Frith & Frith, 2003; Teufal et al., 2010). This top-down control has been demonstrated in modulation of imitation using various social cues, such as eye contact (Wang, Newport & Hamilton, 2011), word scrambles (Leighton et al., 2010; Cook & Bird, 2011) and social status (Lakin & Chartrand, 2003).

Of these well researched social cues, most pertinent to the current thesis is eye gaze. Eye gaze is considered a critical social cue as it expresses social knowledge of imitative behaviour and, based on the STORM theory, direct eye contact is sufficient for an interactive partner to detect imitative behaviours and thus improve performance (Wang & Hamilton, 2012). For example, a study examined the reaction time of executing a hand-opening or hand-closing movement in response to either a congruent or incongruent stimuli, while concurrently playing a video of a model displaying either direct or averted gaze (Wang, Newport & Hamilton, 2011). Results showed that direct gaze improved the speed of hand action imitation relative to the averted gaze, demonstrating the positive affect social cues can have on features of imitation. Similar research showed that by moving the position of the hand stimuli such that it was besides, not directly in front of, the model displaying direct or averted gaze (Wang & Hamilton, 2014), direct gaze again enhanced imitation whereas the averted gaze did not. This finding demonstrated that attention is not necessary for social primes to influence imitation, and subsequently that it may be the social elements of the primes that directly influence or control imitation.

While imitation can be used to form positive social connections and improve performance, it has also been shown to generate negative responses and reduce performance following engagement in a social priming task prior to completion of the hand-opening/ hand-closing stimulus response compatibility paradigm discussed previously (Leighton et al., 2010). The social priming task required the formation of sentences from a series of scrambled words that included either pro-social, neutral or anti-social words. Results showed an automatic imitation effect, where reaction time was faster while imitating compatible, rather than incompatible hand movements (Sturmer et al., 2000; Heyes et al., 2005). In addition to an automatic imitation effect, results showed a pro-social priming effect (Wang & Hamilton, 2011a; Wang & Hamilton, 2014) where reaction time was faster after completing the pro-social word scrambles compared with the neutral and anti-social word scrambles. In addition, results also showed that the anti-social word scrambles decreased performance such that the both the automatic imitation effect and reaction time were decreased following the anti-social primes, relative to the neutral primes. It was suggested that anti-social primes may have increased the inhibition of imitation responses relative to a neutral baseline of inhibition and subsequently decreased performance.

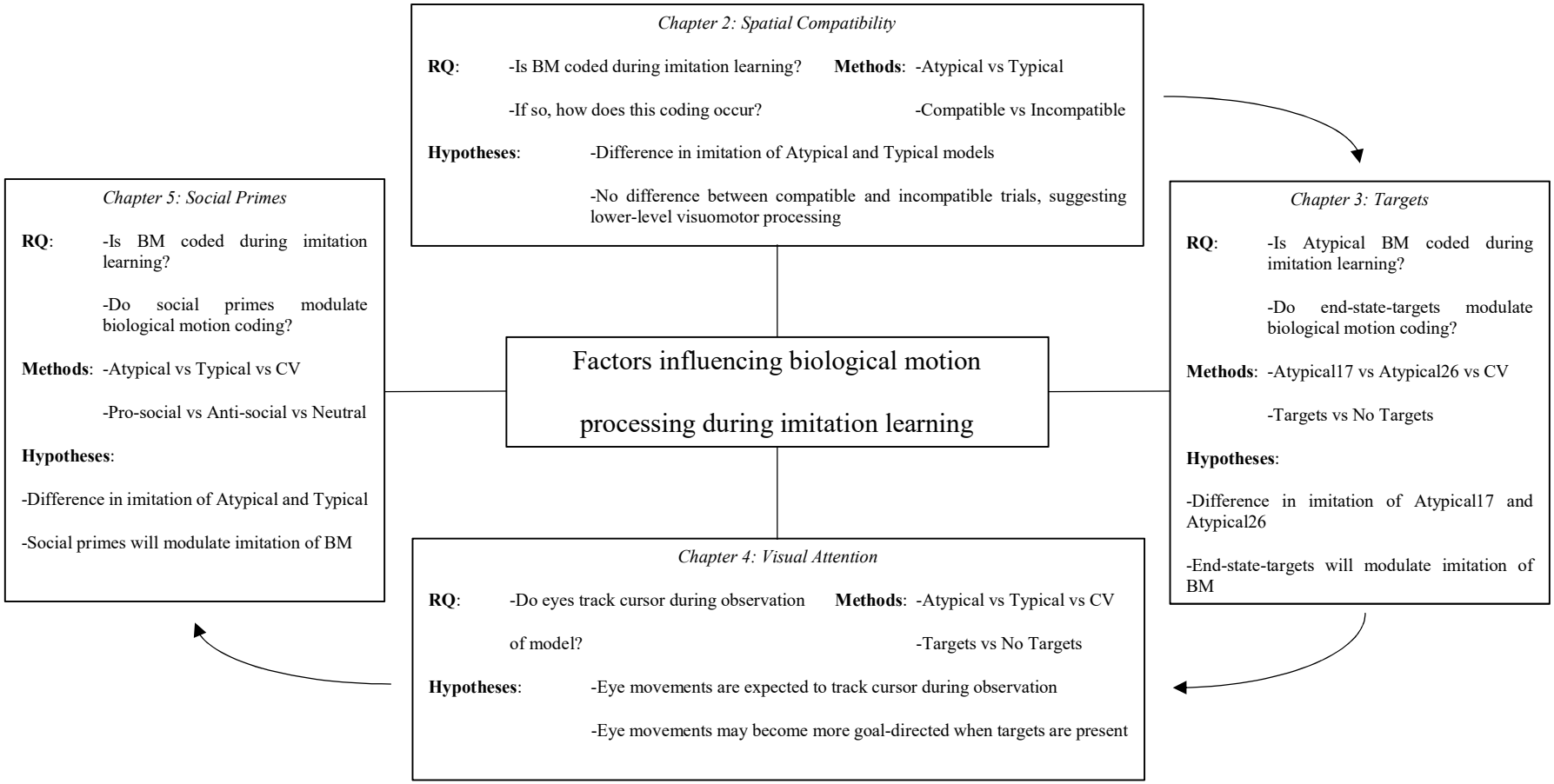


Figure 1.1. A schematic showing the structure, purpose and hypotheses of the four experimental chapters in the current thesis.

1.6 Summary of Research and Current Thesis Aims

The aim of the introductory sections was to give an account of imitation of biological motion kinematics and the processes that underpin it, as well as ways in which the processing can be modulated. It appears to date that although the way in which biological motion is processed is largely lower-level (Iacoboni et al., 1999; Brass & Heyes, 2005), there are numerous top-down factors that can modulate this process (Kilner et al., 2007; Stanley, Gowen & Miall, 2007). However, explicit measurement of the coding of biological motion kinematics and the processes that underpin them during imitation learning are not currently well understood. Therefore, the purpose of the present thesis is to examine a methodology designed to develop the current understanding of biological motion coding. Principally, the methodology in the present thesis defines the to-be-observed models through discrete variables (peak velocity and percentage-time-to-peak-velocity) that are considered appropriate kinematics markers to differentiate model stimuli (Hayes et al., 2014), which are also used to determine imitation accuracy during data analysis.

In addition to developing the fundamental methodology, the present thesis will also seek to clarify the underlying processes associated with biological motion coding. Within the current literature, it has been suggested that some imitation effects may be the result of reproducing spatial properties of observed stimuli, rather than the underlying kinematics contained within the observed stimuli (Heyes, 2011). Therefore, *Chapter Two* will spatially decouple the observed and imitated stimuli to isolate the coding of biological motion to either lower-level or top-down processing. Once a baseline for unmodulated imitation has been established in *Chapter Two*, subsequent experimental chapters will examine additional top-

down modulatory effects (end-state-targets, visual attention) to expand upon the information processing contained within imitation of biological motion during imitation learning.

Finally, as imitation is widely shown to be influenced by social context (Bandura, 1986; Blake, 1958; Chartrand & Bargh, 1999; Wang & Hamilton, 2012), *Chapter Five* examined the way in which biological motion coding was modulated by social priming during imitation learning. In addition to developing the current understanding of social priming on biological motion coding by integrating it with an improved methodology, the social manipulation in *Chapter Five* was included to provide a baseline for social modulation of imitation learning in neurotypicals, which would facilitate future research in people with ASC. Imitation is a well-established issue for people with ASC (Ritvo & Provence, 1953; Ramachandran & Oberman, 2006), so *Chapter Five* was the first in a series of experiments using a similar methodology (Hayes et al., 2016; Hayes, Andrew, Foster, Elliott, Gowen & Bennett, 2017), to examine the sensorimotor processing of biological motion in people with ASC.

1.6.1. Aims of Thesis

The primary aim of the present thesis is to examine whether biological motion kinematics is processed during imitation learning. If biological motion coding is established, the way in which the biological motion is processed will be examined, as well as the ways in which that processing can be modulated. The following section describes the specific hypotheses pertinent to each of the chapters individually:

1.6.1.1. Chapter Two

The primary aim of *Chapter Two* is to examine whether biological motion is coded during imitation learning by examining the imitation of atypical and typical biological motion models. Further, by decoupling the spatial properties of the observed and executed stimuli, *Chapter Two* will examine the underlying processes associated with biological motion coding. Participants will observe the biological motion models in trials that are either spatially compatible or incompatible. Imitation of the model stimuli will always be from left to right and spatially compatible trials will be observed in the same direction and spatial orientation. Spatially incompatible trials will require observation of the model either in the opposite direction (right to left), or orthogonal plane (top to bottom/ bottom to top). Firstly, if biological motion is coded during imitation learning it is expected that there will be differences in imtation between the atypical and typical models. Secondly, if biological motion coding is a function of function of lower-level visuomotor processing (Iacoboni et al., 1999; Heyes, 2001), the kinematics of atypical biological motion should be imitated as accurately during spatially incompatible trials as compatible trials. Conversely, if biological motion coding is mediated by top-down attentional control related to the spatial coordinates of the model kinematics (Proctor & Vu, 2006; Bisio et al., 2010; Heyes, 2011), spatially incompatible trials will be less accurate than spatially compatible trials.

1.6.1.2. Chapter Three

The imitation task in *Chapter Three* presents the visual stimuli in a single horizontal trajectory for both observation and imitation, thus removing any spatial incompatibility. Biological motion coding will be further examined by examining two structurally similar

atypical biological motion models (atypical17 and atypical26) rather than atypical and typical models. In addition to the coding of atypical biological motion, the modulatory effect of goals on biological motion processing will be measured by including the presence of end-state-targets in 50% of the trials (Wohlschlager et al., 2003; Wild et al., 2010). If biological motion kinematics are coded during imitation learning, it is expected that imitation of atypical17 and atypical26 models will be scaled relative to the respective models. Further, if imitation is goal-directed, it is expected that the reproduction of biological motion kinematics will be less similar to those of the models when end-state-targets are present during the imitation task (Bekkering et al., 2000).

1.6.1.3. Chapter Four

In *Chapter Four*, eye movements will be recorded to confirm the direction of visual attention during observation of the model stimuli. The imitation task will examine the coding of biological motion through imitation atypical, typical and constant velocity models, as well as the modulatory effect of end-state-targets on both imitation and eye movements (Wild et al., 2010). It is expected that if biological motion is coded during imitation learning, there will be differences in imitation of the atypical, typical and constant velocity models. If end-state-targets modulate the processing of biological motion, it is expected that imitation of the models will be less accurate, compared to when end-state-targets are absent. Further, eye movements will confirm whether visual attention is directed the cursor or other environmental factors (e.g. end-state-targets) during the observation phase of the imitation task.

1.6.1.4. Chapter Five

Chapter Five will examine whether social context modulates imitation of biological motion kinematics (Lakin & Chartrand, 2003; Wang & Hamilton, 2012). The imitation task will use the same biological and non-biological models as *Chapter Four* (atypical, typical and constant velocity) but remove end-state-targets, and social primes will be examined by showing an image displaying a pro-social, anti-social or neutral eye gaze prime prior to imitation of the model stimuli. The pro-social prime displays a face making direct eye contact; the anti-social prime displays a face looking away from the monitor; and the neutral prime displays a grey background with no person on it. The expectation is that participants will show improved imitation accuracy following the pro-social prime and decreased imitation accuracy following the anti-social prime (Leighton et al., 2010).

1.6.1.5. Chapter Six

The aim of the final chapter is to provide a summary of the above program of work and to integrate these findings with the current understanding in the literature. In addition, conclusions for this body of work will be made and future directions of research will be discussed.

Chapter 2: The Coding of Biological Motion Kinematics During Imitation Learning is a Function of Lower-level Visuomotor Processing

2.1. Abstract

The present chapter investigated the effect of SRC on the representation of atypical biological kinematics during imitation learning. Typical and atypical biological motion models were observed and then imitated with either compatible or incompatible spatial congruency. A compatible trial constituted one where the observed model stimuli moved from left to right and imitation was also executed from left to right. An incompatible trial constituted one where the observed stimuli moved from left to right, but the imitation was executed either right to left, top to bottom or bottom to top. Participants were instructed to observe the model with the intention to later imitate the movement as accurately as possible. Results showed that irrespective of whether the stimulus was observed in a spatially compatible or incompatible orientation, participants imitated both atypical and typical biological motion and imitation was scaled relative to the respective models. Therefore, by demonstrating imitation of novel kinematics during spatially incompatible imitation learning, the current chapter has isolated the processing and representation of atypical biological kinematics to the underlying sensorimotor processes, rather than spatial encoding of peak velocity via processes associated with SRC.

2.2. Introduction

Learning novel movements provides an important means by which humans interact within the world, and with other people. One form of sensorimotor learning is called imitation learning, and requires a person to watch a model with the intention of physically recalling and reproducing the observed action. For example, when observing a hand move between two points at different speeds ('fast' or 'slow'), participants could distinguish between the speeds of the movements and produce an imitation attempt that was scaled to the respective model (Wild et al., 2010). Using TMS to examine the links between perception and action, it was shown that sensorimotor training (Bird, Brindley, Leighton, & Heyes, 2007; Heyes, et al., 2005) could reconfigure the motor system after periods of compatible and incompatible training prior to executing index- and little-finger movements (Catmur et al., 2007). Incompatible training (e.g. observing a little finger movement while executing an index-finger movement) resulted in a countermirror effect, where MEPs were greater in the little-finger when observing the index-finger, and vice versa. These findings show that even though the peripheral motor system is not task-specifically engaged during observation (e.g., a limb is at rest), a sensorimotor representation of the action is developed by engaging a common-coding system linking perception and action (Brass & Heyes, 2005; Jeannerod, 1994; Prinz, 1997).

Direct activation of the sensorimotor system during the observation of actions is said to be underpinned by processes preferentially tuned to biological motion (Press, 2011). As well as facilitating socio-cognitive functioning during interactions between people (Cook, Blakemore, & Press, 2013; Press, Cook, Blakemore, & Kilner, 2011), biological tuning is important for the acquisition of novel motor actions during observational practice (Bird &

Heyes, 2005). Biological tuning has been confirmed across numerous behavioural studies where participants observe different model stimuli that depict typical or atypical human biological kinematics (Hayes, Dutoy, Elliott, Gowen, & Bennett, 2016; Hayes, Elliott, & Bennett, 2010; Hayes, Roberts, Elliott, & Bennett, 2014; Hayes, Timmis, & Bennett, 2009; Roberts, Bennett, Elliott, & Hayes, 2015). Typical kinematics had a movement profile where peak velocity occurred at approximately 50% of the trajectory, which is consistent with goal-directed upper-limb aiming movements (Elliott et al., 2010). Atypical kinematics were novel, and displayed peaks occurring at 18% (Hayes et al., 2016) or 77% (Hayes et al., 2014) of the movement trajectory. From a theoretical perspective, the presentation of atypical kinematics was fundamental for understanding the contribution of low-level visuomotor processes during imitation learning. For example, if a model is presented that has typical kinematics it cannot be ruled out that imitation is based on recognising the movement speed, as opposed to representing the underlying biological motion kinematics. In this case, the feedforward contribution to motor execution would have been associated with rescaling a pre-existing motor representation of a familiar and meaningful movement based on higher-order semantic processes (Rumiati et al., 2005). Whereas, imitation of atypical kinematics cannot be solved by merely recruiting an existing sensorimotor representation, and therefore the sensorimotor system needs to be configured during imitation learning based on representing the kinematics within a mechanism that activates sensorimotor processes.

While an acceptable conclusion, it is relevant that these protocols did not control for the influence of SRC (Heyes et al., 2005). Therefore, it remains a possibility that the spatial position of peak velocity could have been represented through interpretation of the observed visuomotor situation (Hommel & Lippa, 1995). To better isolate processing of biological motion to sensorimotor processes, S-R compatibility can be controlled by arranging the

stimulus and response in an orthogonal (e.g., stimulus hand vertical; responding hand horizontal) orientation (Bertenthal, Longo & Kosobud, 2006; Catmur & Heyes, 2011; Heyes et al., 2005; Press, Bird, Walsh, & Heyes, 2008). Indeed, during automatic imitation, which recruits similar sensorimotor processes as imitation learning (Heyes, 2011), motor responses are facilitated in compatible compared to incompatible trials, thus confirming direct activation of motor representations during action-observation.

Based on the aforementioned methodology, the present chapter investigated S-R compatibility on the reproduction of atypical biological kinematics during imitation learning. Participants observed a model (a single dot) with the intention to reproduce the movement as accurately as possible. During the imitation phase, the model was always imitated moving in a left to right direction on a monitor. During the observation phase, spatially compatible and incompatible trials were randomly interspersed and required observation of the model from one of four origins of movement. In the compatible trials, observation was also in a left to right direction, whereas incompatible trials were observed in a right to left, top to bottom or bottom to top direction. This controlled for both spatially incompatible direction and plane of movement during imitation trials. If the reproduction of atypical biological kinematics is underpinned by direct activation of sensorimotor processes, it is expected that imitation accuracy will be comparable in the spatially incompatible as compatible trials (Catmur et al., 2007; Heyes, 2011). If, however, reproduction is mediated by S-R compatibility associated with spatial orientation, the compatible trials should be more accurate than the incompatible trials (Brass et al., 2000; Sturmer et al., 2000).

2.3. Methods

2.3.1. Volunteers

Twenty participants (aged between 18 and 21 years) volunteered for the study. All participants were right-hand dominant, had normal or corrected-to-normal vision and gave written informed consent. The experiment was designed in accordance with the 1964 Declaration of Helsinki and approved by the research ethics committee of the host University.

2.3.2. Apparatus and Stimuli

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 555 mm. The monitor was connected to a PC (HP Compaq 8000 Elite), which also recorded input of a hand-held stylus on a graphics tablet (Wacom Intuos Pro XL), which displayed a 1:1 ratio between the tablet and screen to reduce any potential learning effects required to complete the imitation task. This ratio was consistent throughout all experiments contained within the current thesis. Experimental stimuli were generated using COGENT toolbox (developed by John Romaya at the Laboratory of Neurobiology at the Wellcome Department of Imaging Neuroscience) and implemented by MATLAB (Mathworks Inc.).

Two non-human agent models were created by a human volunteer performing typical and atypical horizontal movements using a hand-held stylus on a graphics tablet (Figure 2.1A). The stylus movement was represented as a white-dot (diameter = 6 mm) on the computer monitor, and traversed from the left-hand start-position (red-dot, diameter = 12 mm)

to the right-hand end-position located at an amplitude of 200 mm. The total movement duration was exactly 1700 ms. For both models, raw position data were first filtered using a low pass 4th order autoregressive filter with an 8 Hz cut-off. Data were then differentiated using a three-point central difference algorithm to obtain velocity. The typical model reflected an exemplar trial, and thus displayed a typical (Elliott et al., 2010; Flash & Hogan, 1985) bell-shaped velocity profile (dashed trace in Figure 2.1C) with a peak velocity of 0.2 mm/ms that occurred at 44% of the movement duration. For the atypical model (black trace in Figure 2.1C), peak velocity was 0.41 mm/ms and occurred at 18% of the movement duration. Peak velocity and percentage-time-to-peak-velocity were selected as the kinematic dependent variables as they had been used in previous research that had reached publication (Hayes et al., 2014) and were therefore considered appropriate kinematic markers within the literature. Further, as peak velocity and percentage-time-to-peak-velocity were the discrete kinematic markers used to define the model stimuli, it followed that they were also the kinematic markers used to determine imitation accuracy.

The method of using a human volunteer to generate both models was important because it ensured the kinematics were biological and reproducible by participants (Hayes et al., 2016). This did result in movement deviation in the x and y axes, however the latter was minimal (i.e., perpendicular deviation). In addition to conforming with kinematic parameters suggested by previous literature, the typical and atypical biological motion models were also examined through rigorous pilot testing. Although several models were compared, the atypical and typical models used in the current chapter, and indeed much of the thesis, were most frequently identified as being human movements that presented discernibly different kinematic structures when viewed by novice observers. Comprehensive pilot testing of the models and inclusion of a familiarisation phase prior to testing alleviated a requirement to post-experimentally debrief participants on their perception of the stimuli or requirements of the task.

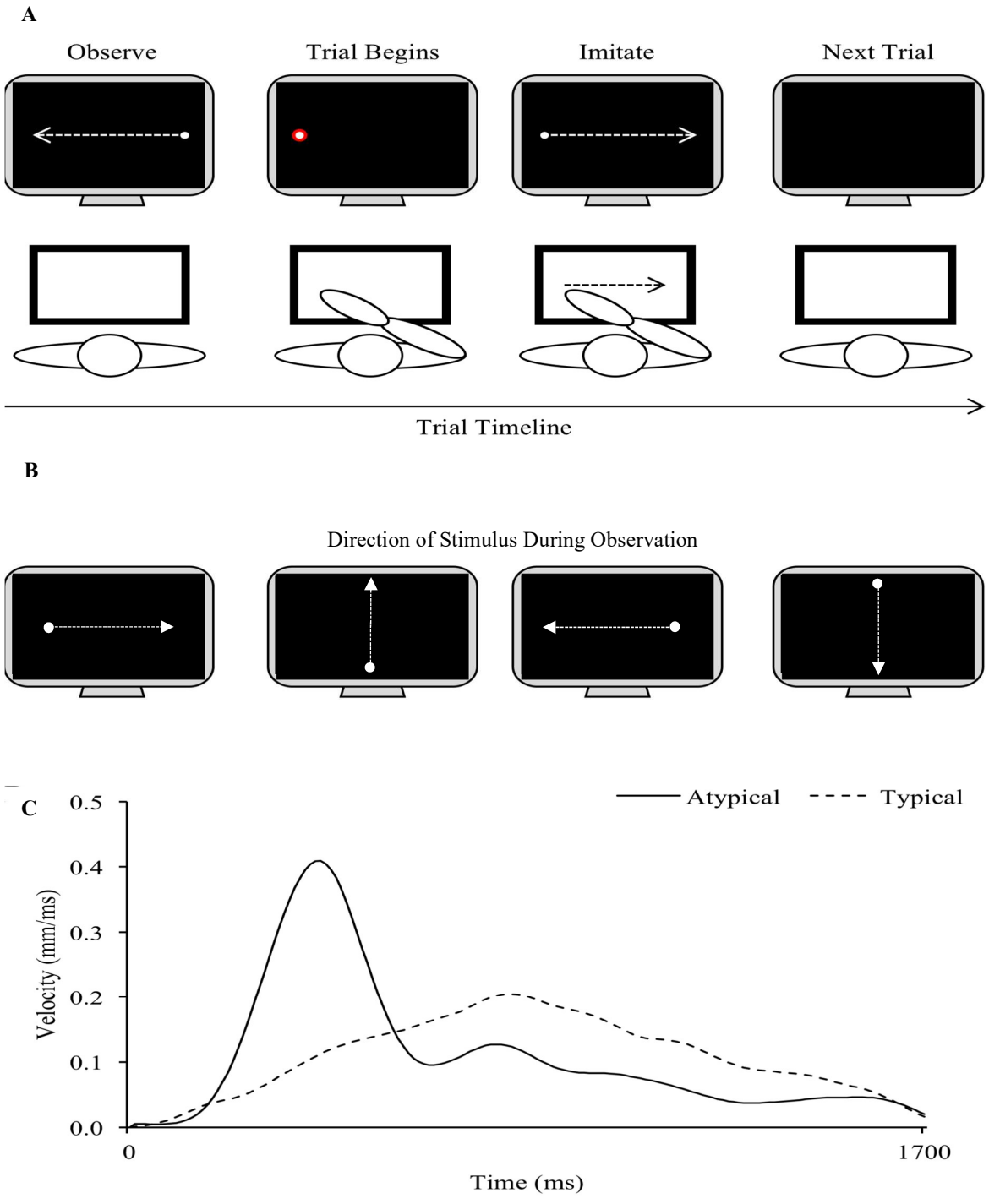


Figure 2.1. (A) A schematic representation of the experimental trials. The black outlined rectangle represents a graphics tablet. The white circle displayed on the CRT monitor represents the model. The single-segment movement is depicted by the arrow (i.e., from the

start-position to the end-position). (B) The directions of the model stimuli during observation, originating from the white circle. (C) Displacement time-series displaying typical (dashed trace) and atypical (black trace) velocity models.

2.3.3. Procedure

The experiment consisted of familiarisation and experimental phases. In the familiarisation phase, participants performed 6 trials with the intention of understanding the imitation protocol. The 6 familiarisation trials had an observation and imitation phase, as in the experimental phase, and consisted of observing a constant velocity model, which displayed the exact movement duration and amplitude of the experimental models, but with a constant velocity in the horizontal x axis of 0.120 mm/ms. Observing the constant velocity model ensured construct validity by preventing participants experiencing biological motion before the imitation trials, although no specific information was provided regarding the nature of model, nor was feedback regarding imitation performance provided. Following an observation, participants were instructed to imitate the model as accurately as possible by using the stylus on the tablet. The familiarisation phase allowed participants to understand the spatiotemporal relationship between the stylus movement on the graphics tablet and cursor movement on the screen, and quantified base-line motor behaviour associated with performing typical goal-directed movements.

The experimental phase consisted of 80 trials that comprised 10 blocks of 8 trials. A block contained 4 typical and 4 atypical biological kinematic models wherein each of the 4 trials had a different point of origin during observation (left to right; right to left; top to bottom; bottom to top; see Figure 2.1B). Every imitation was from left to right, so each block had 1 spatially compatible atypical and typical trial and 3 spatially incompatible atypical and typical trials. Trial order within a block, as well as block order, was randomised across volunteers.

The randomised structure reduced predictability of an upcoming models and promoted imitation on a trial-by-trial basis. This research design was like previous research examining biological motion coding that had reached publication (Hayes et al., 2009; 2014) and thus, was accepted as a balanced and thorough way in which to design the experimental protocol.

2.3.4. Data Reduction

To quantify imitation of movement kinematics, analysis focused on x-axis data. Position data of the start and end of the movement was identified in each imitation trial. The start was identified when the cursor was moved beyond the perimeter of the home-position, while the end was identified when the participant clicked the lower-button on the stylus. From this identification process, the position data was filtered using a low pass 4th order autoregressive filter with an 8 Hz cut-off. The filtered data were then differentiated using a 3-point central difference algorithm to obtain velocity. A MATLAB routine extracted the movement and displayed the velocity curve for each trial. Using a mouse, an experimenter manually selected the start, peak, and end of the movement on the velocity curve. While the clicking of the lower-button during the imitation trial indicated a general point at which the participant considered their imitation trial to be complete, manual picking of the data ensured a consistent end-point based on minimum velocity thresholds, where the MATLAB routine integrated the velocity curve to identify the start of the movement when velocity was > 0.003 mm/ms, and the end when velocity was < 0.003 mm/ms. Peak velocity and percentage-time-to-peak-velocity from each trial was quantified, with percentage-time-to-peak-velocity calculated as (time to peak velocity / movement time) x 100. Intra-participant means were calculated from 10 trials associated with each model and origin of movement (e.g., 10 trials

for the atypical model in the left-to-right origin). 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; 2016; Andrew et al., 2016).

2.3.5. Data Analysis

Intra-participant mean data were submitted to separate 2 Model (atypical; typical) x 4 Origin (left-to-right; right-to-left; top-to-bottom; bottom-to-top) repeated measures ANOVA. Significant main and/or interactions effects involving more than two means were analysed using Tukey HSD post-hoc procedure and alpha was set at $p < 0.05$.

2.4. Results

2.4.1. Peak Velocity

A main effect of model [$F(1, 19) = 39.241$, $p < 0.001$] for peak velocity indicated that magnitude was significantly higher when imitating atypical (M = 0.280 mm/ms; SD = 0.079 mm/ms) compared to typical (M = 0.192 mm/ms; SD = 0.033 mm/ms) biological kinematics. As seen in Figure 2.2A, the ANOVA did not reveal a main effect of origin [$F(3, 57) = 1.707$, $p = 0.176$] or a model x origin interaction [$F(3, 57) = 1.800$, $p = 0.157$].]

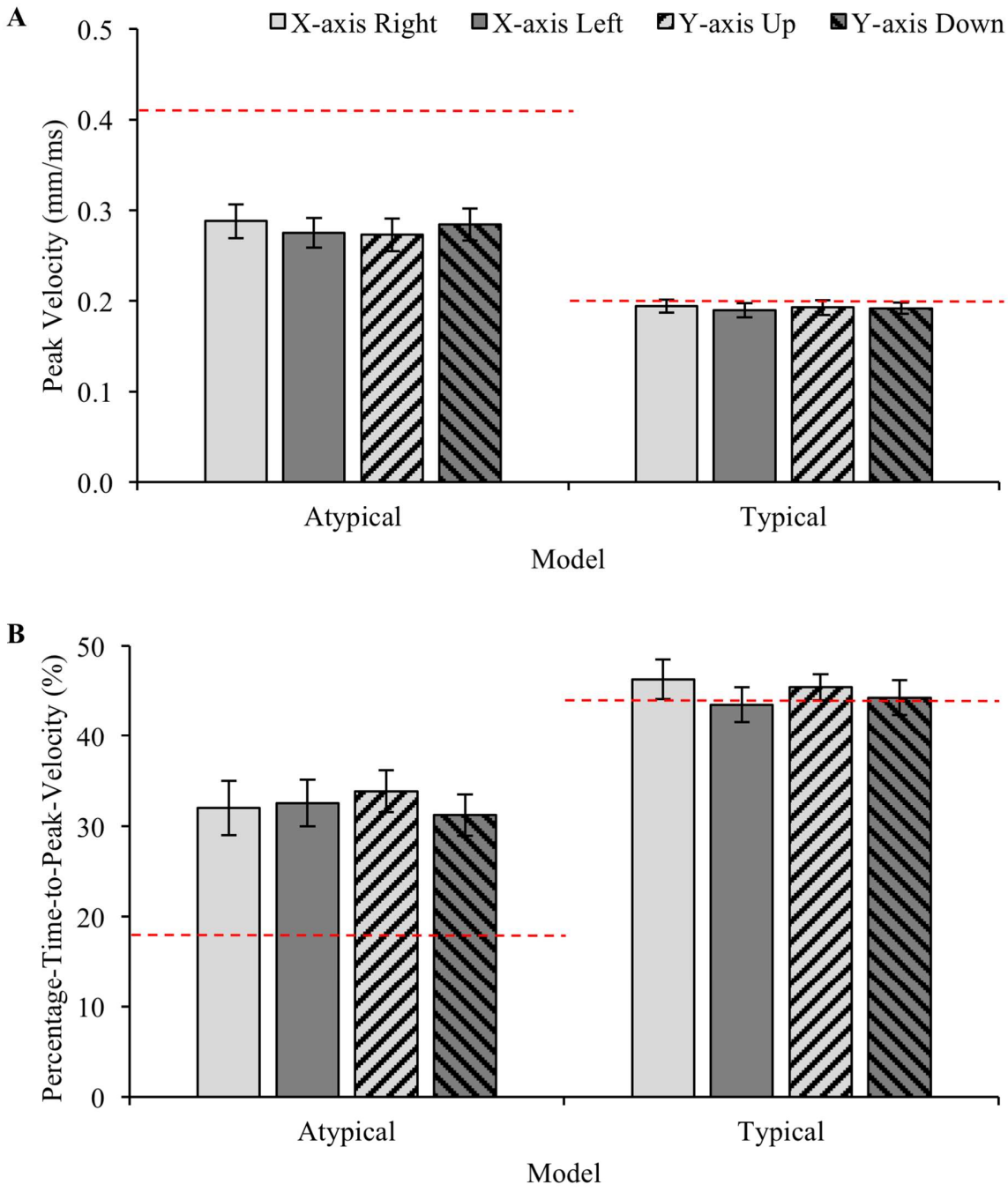


Figure 2.2. (A) Peak velocity of imitation across all four origins of movement (error bars represent standard error of the mean) presented as a function of model. Dashed line represents the model. (B) Percentage-time-to-peak-velocity of imitation across all four origins of movement (error bars represent standard error of the mean) presented as a function of model. Dashed line represents the model.

2.4.2. Percentage-Time-To-Peak-Velocity

A main effect of model [$F(1, 19) = 46.639$, $p < 0.001$] for percentage-time-to-peak-velocity indicated that peak velocity occurred significantly earlier when imitating the atypical ($M = 32$ %; $SD = 11$%) compared to typical ($M = 45$ %; $SD = 8$%) biological kinematics (Figure 2.2B). The ANOVA did not reveal a main effect of origin [$F(3, 57) = 1.161$, $p = 0.332$] or a model x origin interaction [$F(3, 57) = 0.893$, $p = 0.450$].

These effects can be seen in the exemplar velocity traces illustrated in Figure 2.3. When imitating the atypical biological kinematics, peak velocity occurred earlier in the movement across all origins (Figure 2.3A). When imitating the typical biological kinematics, peak velocity occurred toward the midpoint of the movement for all origins (Figure 2.3B).

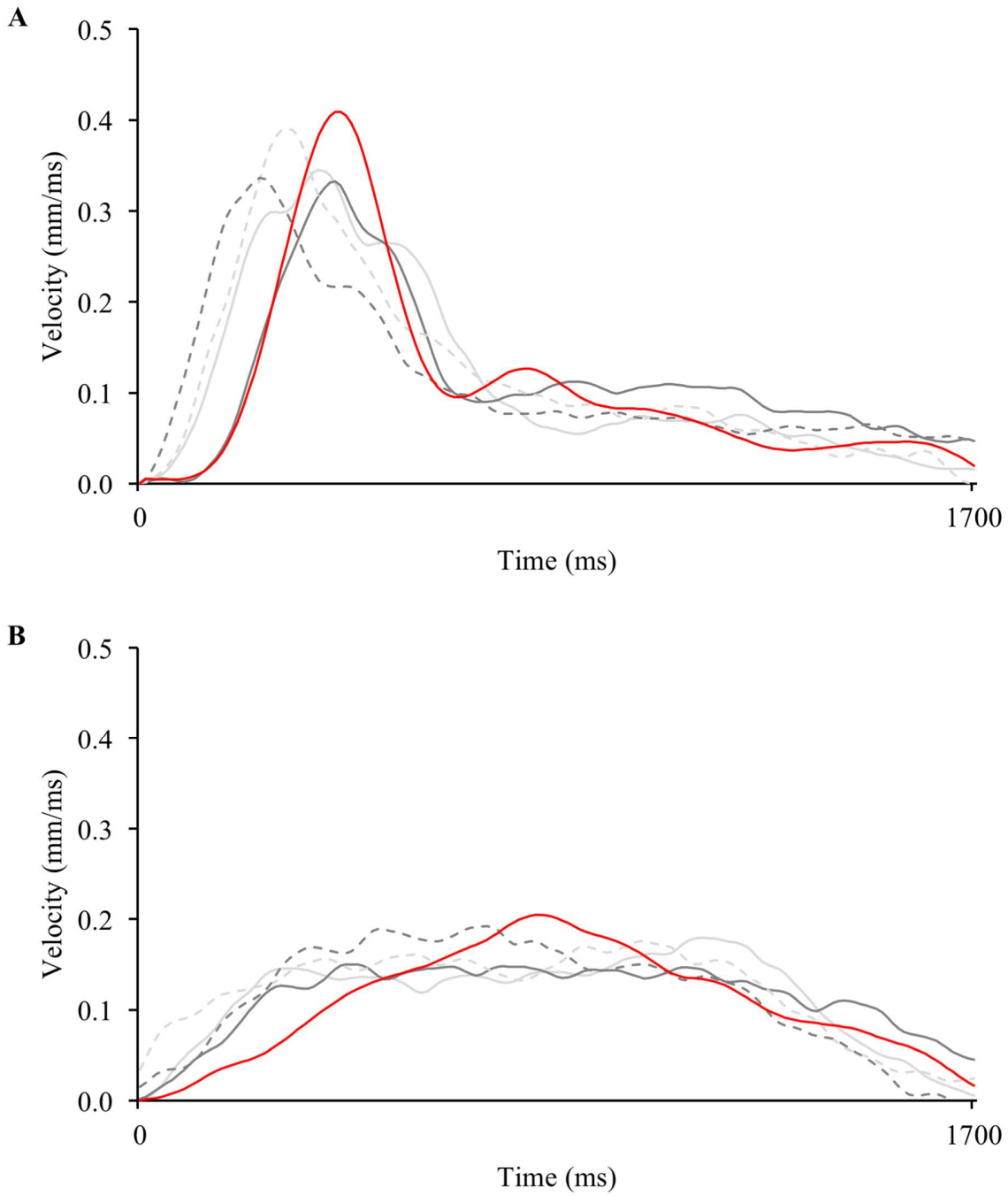


Figure 2.3. (A) Exemplar velocity traces for imitation of the atypical model during compatible (black trace) and incompatible (light-grey, light-grey dashed, and black dashed traces) trials, as well as the model (red trace). (B) Exemplar velocity traces for imitation of the typical model during compatible (black trace) and incompatible (light-grey, light-grey dashed, and black dashed traces) trials, as well as the model (red trace).

2.5. Discussion

The present chapter investigated the influence of S-R compatibility on the reproduction of atypical biological kinematics during imitation learning. Results showed that percentage-time-to-peak-velocity occurred earlier in the movement trajectory, and was scaled to the model, during imitation of the atypical, compared with the typical model. Similarly, the magnitude of peak velocity was greater during imitation of the atypical, compared with the typical model. Following observation of the typical model, peak velocity occurred towards the midpoint of the trajectory and reflected the constraints of the task, as well as a pre-existing motor repertoire. In addition to the imitation of atypical biological motion kinematics, results showed both atypical and typical biological motion kinematics were imitated with a similar degree of accuracy during spatially compatible and incompatible trials.

These findings support previous work (Hayes et al., 2016) that showed atypical kinematics are represented during imitation learning. As before, it is suggested that this occurs within a mechanism that activates sensorimotor processes. However, to control the influence of S-R compatibility (Hommel & Lippa, 1995), the observation and imitation trials were spatially decoupled in both the direction (left to right and right to left) and plane (top to bottom and bottom to top) of the imitated movements. The fact that incompatible trials showed reproduction of the atypical kinematics when physically executing the movement in the opposite (left to right) or orthogonal (top to bottom, bottom to top) direction, strengthens the suggestion that sensorimotor adaptation occurs via lower-level processes linking visual and motor representations (Catmur, Walsh, & Heyes, 2007; Catmur & Heyes, 2011). For example, there is a possibility that participants represented a kinematic landmark during observation,

such as the position that peak velocity occurs (e.g., spatial position relative to the monitor frame), however this is a less likely explanation that would require a spatial translation to reproduce an accurate atypical trajectory in an incompatible reproduction of the stimulus.

In addition to low-level sensorimotor processes underlying the adaptation effects, other higher-order processes may have been involved. Specifically, visual attention and intention could have modulated the lower-level processing of the atypical kinematics following the instructions given to participants to observe the model with the intention to execute a movement during imitation that was as accurate to the observed stimulus as possible (Hayes et al., 2014; 2016). Also, having perceived the atypical model had an explicit acceleration profile that differed from the typical model that was observed, and/or their own pre-existing sensorimotor repertoire, it follows that during the experimental phase inductive processes could have influenced the developing sensorimotor representation (Burke, Tobler, Baddeley, & Schultz, 2010; Turnham, Braun, & Wolpert, 2011). However, the randomised trial order would have minimised the frequency of repeated stimuli and thus the opportunity to directly compare lower-level sensorimotor representation through repeated trials (Tenenbaum, Griffiths, & Kemp, 2006; Turnham, et al., 2011). Therefore, in line with automatic imitation research (Catmur & Heyes, 2011; Cook & Bird, 2011; Grezes et al., 2003) the imitation of atypical biological motion is more likely to reflect the sensorimotor representation of atypical biological motion resonating with the motor system such that a correlated motor response is generated.

The coding of atypical biological motion through lower-level sensorimotor processes supports previous research that has suggested that biological motion kinematics are processed in the parietal and frontal regions of the perception-action system (Casile et al., 2010; Dayan et al., 2007; Higuchi et al., 2012; Press, Catmur, Cook, Widmann, Heyes & Bird, 2012). In

addition to confirming perception-action system activity, parietal and frontal neural activation during lower-level biological motion coding corroborates the EP-M model of imitation (Hamilton, 2008). Within the EP-M model, the M-route suggests that the STS generates a visual description of the observed stimulus, which is coded in IFG based on the kinematic properties and directly mapped as a motor representation containing the underlying kinematic features for subsequent imitation. This direct transformation of visual description to motor representation corroborates the ASL theory (Heyes & Ray, 2000), which suggests vertical associations are made between the sensory and motor representations that are strengthened through correlated sensorimotor experience of the stimuli. The direct association between the visual and motor representations formed through lower-level processing suggest the imitation of atypical biological motion is likely to be a result of reproducing the underlying kinematics, rather than the spatial properties of the movement (Iacoboni et al., 1999; Buccino et al., 2004).

To conclude, the present chapter confirmed that atypical biological kinematics associated with an observed novel action are represented and reproduced during imitation learning. Although this effect has been shown previously (Hayes et al., 2014; Hayes et al., 2016; Andrew, et al 2016), the current data extends theoretical knowledge of the processes underlying imitation learning by implementing a methodology that controls movement direction of a model during action-observation and imitation, and thus spatial compatibility. This method better isolates the representation of atypical kinematics to sensorimotor processes rather than spatial encoding. Moreover, by using discrete kinematic markers to both define the models and measure imitation accuracy, these data represent the most accurate measurement of biological motion coding during imitation learning in the current literature.

Chapter 3: Atypical Biological Motion Kinematics are Represented by Complimentary Lower-level and Top-down Processes During Imitation Learning

3.1. Abstract

Learning a novel movement requires a new set of kinematics to be represented by the sensorimotor system. This is often accomplished through imitation learning where lower-level sensorimotor processes are suggested to represent the biological motion kinematics associated with an observed movement. Top-down factors have the potential to influence this process based on the social context, attention and salience, and the goal of the movement. In order to further examine the potential interaction between lower-level and top-down processes in imitation learning, the aim of this study was to systematically control the mediating effects during an imitation of biological motion protocol. In this protocol, a non-human agent model that displayed different novel atypical biological motion kinematics, as well as a control model that displayed constant velocity. Importantly the three models had the same movement amplitude and movement time. Also, the motion kinematics were displayed in the presence, or absence, of end-state-targets. Kinematic analyses showed atypical biological motion kinematics were imitated, and that this performance was different from the constant velocity control condition. Although the imitation of atypical biological motion kinematics was not modulated by the end-state-targets, movement time was more accurate in the absence, compared to the presence, of an end-state-target. The fact that end-state-targets modulated movement time accuracy, but not biological motion kinematics, indicates imitation learning involves top-down attentional, and lower-level sensorimotor systems, which operate as complementary processes mediated by the environmental context.

3.2. Introduction

Imitation is a powerful mechanism that supports human interaction. In familiar social settings, imitation involves the automatic activation of a motor response triggered by observing a similar motor action (Chartrand & Bargh, 1999; Heyes, 2001, 2011; Heyes et al., 2005). For example, individuals execute faster pre-specified movements (e.g., finger tapping) when observing biologically compatible (finger tapping), compared to incompatible (finger lifting), movements (Brass, Bekkering, & Prinz, 2001; Stürmer, Aschersleben, & Prinz, 2000). The shorter motor reaction times occur independent of task instructions, which suggests involvement of automatic sensorimotor processes linking perception and action (Brass & Heyes, 2005; Prinz, 1997).

To understand if the automatic sensorimotor effects are developed through experience, and linked to a general mechanism incorporating processes associated with perception, action and attention (Leighton, Bird, Orsini & Heyes, 2010), studies have examined automatic imitation following correlated sensorimotor training (Bird, Brindley, Leighton, & Heyes, 2007; Catmur, Mars, Rushworth, & Heyes, 2011; Catmur, Walsh, & Heyes, 2007, 2009; Cavallo, Heyes, Becchio, Bird, & Catmur, 2014; Heyes, et al., 2005). For example, individuals performed a countermirror protocol that required compatible or incompatible sensorimotor training (Catmur, et al., 2007). During compatible training, individuals executed index-finger movements, whilst simultaneously observing index-finger movements. During incompatible training, individuals executed index-finger movements, whilst simultaneously observing little-finger movements. After incompatible training, TMS-induced MEPls 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 mediates (Meltzoff & Moore, 1977) the translation of visual information into a motor action.

Of primary interest to the present chapter is the suggestion that similar sensorimotor processes operate during automatic imitation and imitation learning (Brass & Heyes, 2005; Buccino et al., 2004; Heyes, 2011; Iacoboni, 2009). Like the countermirror principle, imitation learning often requires the sensorimotor system to represent a novel biological motion across consecutive imitation trials. Although there is strong evidence that biological motion is processed during automatic imitation (Brass, Bekkering, Wohlschlaeger, & Prinz, 2000; Heyes, et al., 2005; Press & Heyes, 2008) and interpersonal observation-execution imitation tasks (Kilner, Paulignan, & Blakemore, 2003), support from imitation learning studies has typically been based on protocols that manipulated the speed of the imitated movement (Bisio, Stucchi, Jacono, Fadiga, & Pozzo, 2010; Hayes, Timmis, & Bennett, 2009; Wild, Poliakoff, Jerrison, & Gowen, 2010).

Although participants have been shown to imitate different movement speeds (e.g., slow, medium, and fast upper-limb aiming movements), it is notable that the observed stimulus was representative of typical aiming movements (Wild, et al., 2010). Thus, it remains possible that imitation was limited to recognising differences in movement speed between observations, as opposed to representing the underlying biological motion kinematics. In this case, the feedforward contribution to motor execution could have been associated with an individual recruiting and rescaling a pre-existing motor representation of a familiar and meaningful aiming movement (Hayes, Roberts, Elliott, & Bennett, 2014; Hayes, et al., 2009). This would imply imitation was based on higher-order semantic processes (Rumiati, Papeo,

& Corradi-Dell'Acqua, 2010; Rumiati et al., 2005), as opposed to lower-level sensorimotor processes representing the observed biological kinematics.

The current chapter adopted a novel protocol that enabled direct examination of biological motion processing during imitation learning. In addition to displaying a constant velocity control model, the structure of two experimental models was manipulated so that peak velocity in the aiming movements no longer occurred at the typical mid-point (40-60% of the total time) of the trajectory (Elliott, Helsen, & Chua, 2001). With such stimuli, imitation can be quantified according to timing and magnitude of velocity, which in combination would not reflect the kinematics of typical aiming movements (Hayes, et al., 2014). Following this logic, imitation of two different biological motion models was compared, in which percentage-time-to-peak-velocity occurred at 17% or 26% of the total movement time (henceforth atypical17 and atypical26), and thus earlier than normally expected when aiming to a target. By maintaining equal movement time and amplitude, magnitude of peak velocity also differed between the biological motion models (atypical 17 = 0.37 mm/ms; atypical 26 = 0.24 mm/ms). These specific kinematic features were selected for atypical17 and atypical26 based on rigorous pilot testing and conformity with the guidelines for atypical kinematics within current literature. Peak velocities for both models occurred earlier than the 40-60% associated with a typical velocity profile (Elliott et al., 2001) and importantly, were both identified as being different to each other and faster than the typical model used in the previous chapter during pilot testing.

Finally, given that the lower-level processes that code biological motion kinematics are modulated by various top-down processes (Bekkering, Wohlschlaeger, & Gattis, 2000; Heyes & Bird, 2007; Leighton, et al., 2010; Rumiati, et al., 2005; Southgate & Hamilton, 2008; Wang & Hamilton, 2012), motion stimuli was displayed as a non-human agent (a white

dot) to control social context, and in the presence or absence of end-state-targets. The latter manipulation is important because previous work (Hayes et al., 2007; Wild, et al., 2010) has shown that the imitation of biological motion is attenuated in the presence of an end-state-target. In this context, the end-target provides a salient task-relevant (Leighton, et al., 2010) environmental visual cue that modulates attention so that this feature (target attainment) is prioritized and represented during imitation. The removal of end-state-targets in half of the present experimental trials generated a protocol that examined biological motion kinematics during true imitation (Cook & Bird, 2012; Vivanti & Hamilton, 2014).

With behaviourally realisable but atypical biological motion (i.e., atypical17; atypical26), represented as a non-human agent, it was expected that participants would imitate in accord with the observed biological kinematics (Hayes, et al., 2014) and thus produce movements scaled to both timing and magnitude of peak velocity. Because of the constraints on human movement imposed by the neuro-muscular system (Abend, Bizzi, & Morasso, 1982), participants were not expected to move with constant velocity having observed the constant velocity stimulus, or to execute a kinematic profile that resembled the atypical motion kinematics. Rather, it was anticipated that participants would recruit a pre-existing motor response and thus exhibit time of peak velocity that was similar to typical aiming movements. Finally, it was anticipated that imitation of atypical biological motion would be more accurate in the absence, compared to presence, of end-state-targets. In the absence of end-state-targets, there should be minimal contribution from top-down attentional processes, thus encouraging participants to focus on representing the characteristics of lower-level visual stimuli during imitation learning.

3.3. Methods

3.3.1. Volunteers

Data were recorded from twenty participants (aged range 18 - 21 years) who volunteered for the study. All participants had normal or corrected-to-normal vision and gave written informed consent. The experiment was designed in accordance with the Declaration of Helsinki and was approved by the ethics committee of the host University.

3.3.2 Apparatus and Procedures

The apparatus consisted of a PC (Dell Optiplex GX280), a 21-in CRT computer monitor (IIyama Vision Master 505), and a graphics tablet with a hand-held stylus (WACOM Intuos 3). The CRT monitor operated with a spatial resolution of 1280 x 1024, and a refresh rate of 85 Hz. Visual stimuli were generated via MATLAB (The Mathworks, Inc), using Cogent 2000 toolbox (www.vislab.ucl.ac.uk/cogent.php). Importantly, the apparatus used in the current chapter had the same setup as that used in the previous chapter and were consistent throughout the thesis.

Participants were required to observe and imitate the movement of a model (a white cursor, diameter = 8mm) presented on the 21-inch CRT monitor. The model displayed a single horizontal trajectory that originated from a home-target positioned on the left-hand side of the screen. The amplitude of the movement was 200 mm, with a movement time of 1700 ms, and ended on the right-hand side of the monitor. For the end-state-target condition, two red circles representing home-target and the end-state-target (diameter = 16 mm) were positioned at centre-left (home) and centre-right (end-state) of the monitor (Figure 3.1A). To examine

imitation of biological motion, three models were created: atypical (atypical17; atypical26) or constant velocity (Figure 3.2). The atypical models displayed a velocity profile that was positively skewed so that peak occurred at 17% or 26% of movement time, and with a magnitude of 0.37 mm/ms and 0.24 mm/ms, respectively. The models were created by a human volunteer who practiced the two atypical goal-directed aiming movements using a hand-held stylus on a graphics tablet until a white cursor, which represented the stylus, moved from a left-hand home-target to a right-hand end-state-target in a movement time of 1700 ms. The displacement time-series data recorded from a successful practice trial for each model was selected to create the models. The method of using a human to generate the models was critical because it ensured the kinematics of the movement was biological in origin, and thus the movement was achievable. 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, but with a constant velocity trajectory that had no deviations in the perpendicular axis.

Prior to the experimental trials, all participants completed a familiarization period that replicated the conditions of the imitation task. Participants sat on a chair in front of a CRT monitor and held the stylus in their preferred hand. The participants performed four familiarization trials; 2 trials representing the end-state-target condition (see Figure 3.1A) performed in the imitation task, and 2 trials representing the no-end-state-target condition (see Figure 3.1B) performed in the imitation task. Participants were instructed to passively observe the model stimuli during the observation phase, with the intention of reproducing the model as accurately as possible during the imitation trials. These specific instructions were given to provide as little information about the nature of the task and kinematic structures of the models as possible, thus allowing for the most natural imitation of the respective models. Each trial

commenced with the model being positioned in the centre of the home-target. The participants observed the model display a movement from the home-target to an end-target (end-state-target condition), or end space (no-end-state-target condition), with a constant velocity trajectory and a movement time of 1700 ms. A constant velocity trajectory was used to ensure construct validity by preventing participants from experiencing biological motion before the imitation trials. Participants were not informed about the agency of the model or duration of the movement time. Following observation of the model, participants moved the cursor from the centre of the monitor to the centre of the home-target, and clicked the lower-button on the stylus. In an end-state-target condition, the two targets remained on the screen as the participant imitated the model. In a no-end-state-target condition, the two targets were removed before a participant imitated the model. To finish imitation, participants clicked the lower-button on the stylus a second time once the cursor was located in the end-state-target, or end-space in the no-end-state-target condition. After familiarization, all participants confirmed they understood the model, the end-state-target and no-end-state-target conditions, the instruction to imitate, and the sensorimotor association between the stylus on a graphics tablet, and the corresponding movement of cursor on the monitor.

The imitation task comprised 14 blocks of 6 trials (84 trials). A block contained each of the 6 combinations of target (end-state-target, no-end-state-target) and velocity model (atypical17, atypical26, constant) presented in random order. A trial commenced with an observation phase where the home-target (red) was displayed on the monitor for 1000 ms, before disappearing for 1000 ms, and being replaced by a model positioned in the same location. Depending on the trial type, the model moved to an end-state-target (Figure 3.1A) or end-space in the no-end-state-target (Figure 3.1B) condition, with one of three velocity models. After observing the model, participants imitated the movement as per the instructions

given in the familiarization period. This experimental design ensured consistency between experimental chapters within the current thesis by equally distributing trial types and containing a total number of trials that produced sufficient exposure to each condition, as consistent with previously published research (Hayes et al., 2009; 2014).

3.3.3. Statistical Analysis

To quantify imitation performance, and imitation of atypical biological motion, movement kinematics exhibited by the participants were extracted on each trial. The start of movement was defined as the time the centre of the cursor moved beyond the perimeter of the home-target, and the end was calculated when the participant clicked the lower-button on the stylus. For each imitation attempt, the 2-dimensional displacement data were filtered using a low-pass (8 Hz) autoregressive filter. These data were differentiated using a central difference algorithm to obtain velocity. A MATLAB routine extracted the primary movement occurring in the x-axis and identified the following dependent variables: movement time, peak velocity, and percentage-time-to-peak-velocity. The two velocity variables were chosen for analysis because they most reflected the difference between the two atypical biological motion models. Intra-participant means from the 14 trials per condition were calculated for each dependent variable and submitted to separate Model (atypical17; atypical26; constant velocity) x Target (end-state-target; no-end-state-target) repeated measures ANOVAs. Alpha was set at $p < 0.05$ and follow-up testing used the Tukey post-hoc procedure.

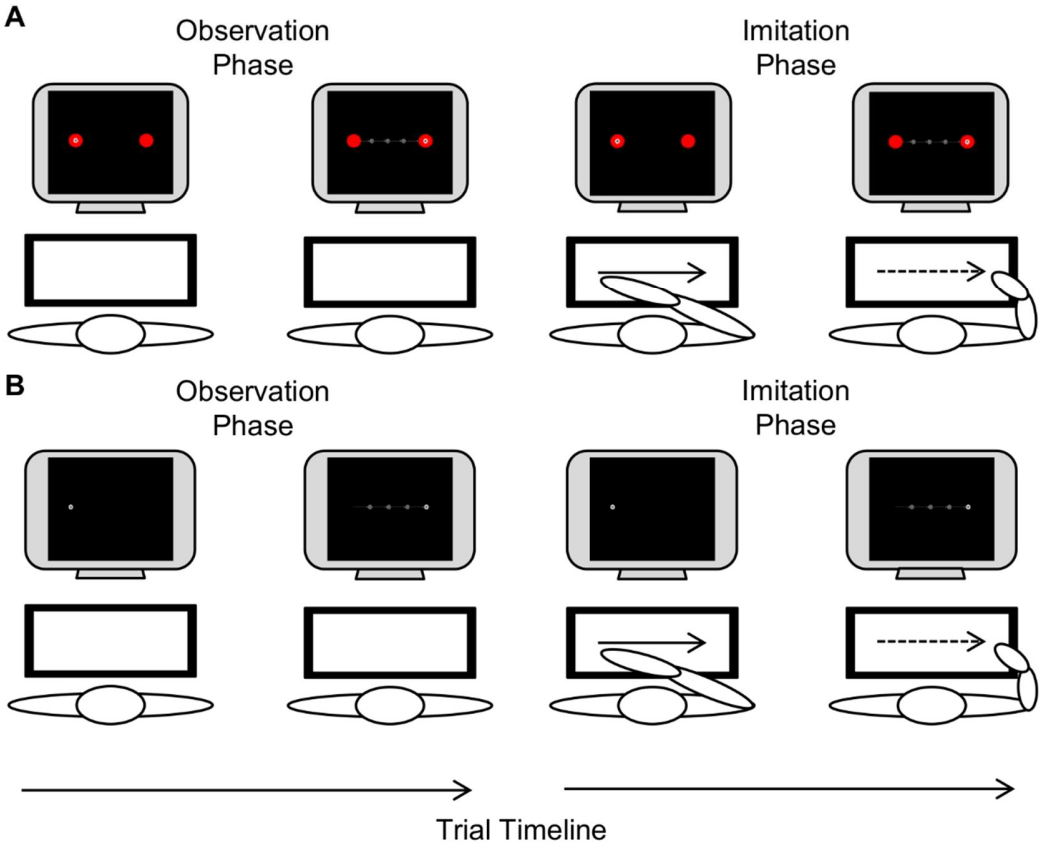


Figure 3.1. A visual representation depicting a single trial in the end-state-target-condition (A) and no-end-state-target condition (B). The apparatus outlined in Panel A and B is a CRT monitor and a graphics tablet. The trial timeline arrows at the bottom of the figure indicate the Observation Phase and Imitation Phase. During the Observation Phase, the non-human agent model is positioned in the left-hand home target (A) and left-hand space (B). The model (atypical17 or atypical26 or constant velocity) displays a horizontal movement of 200 mm from the left-hand home target to an end-state-target (A) or end-space in the no-end-state-target-condition. The model has a movement time of 1700 ms. The Imitation Phase commences with the white cursor positioned in left-hand home target (A) or left-hand space (B). A participant imitates the observed model by controlling a stylus on the graphics tablet.

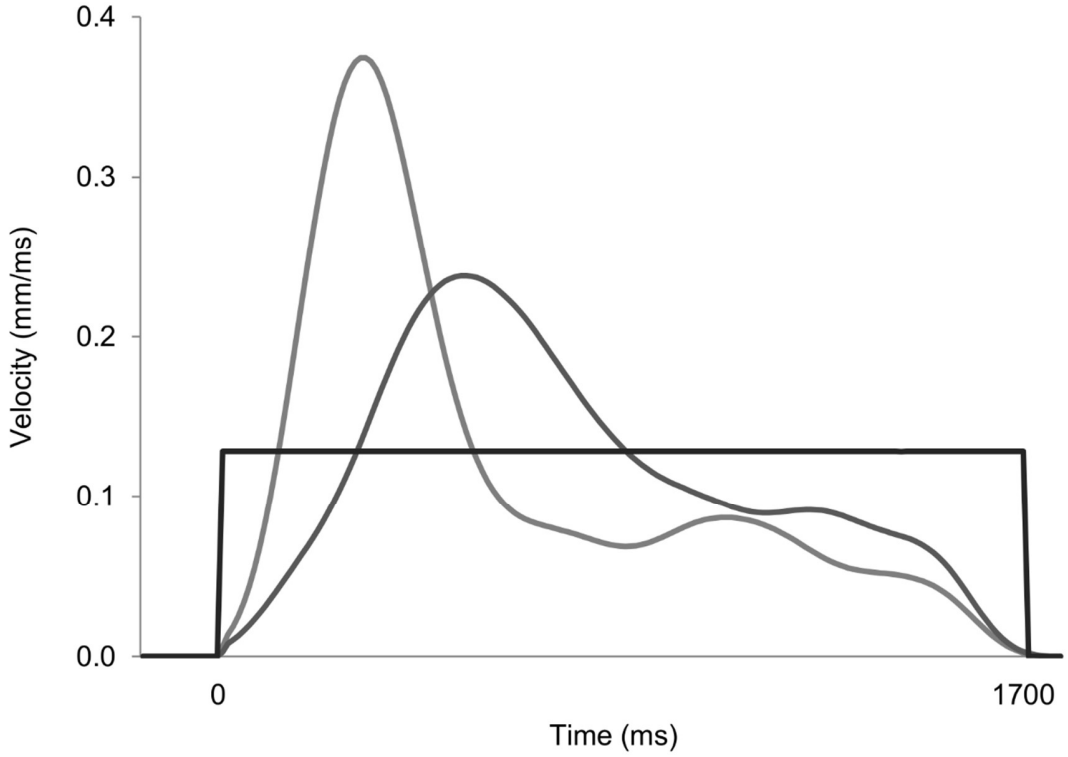


Figure 3.2. The velocity profiles for atypical17 model (light grey trace; peak), atypical26 model (dark grey trace), and constant velocity control model (black trace).

3.4. Results

3.4.1. Movement Time

As illustrated in Figure 3.3, the presence of an end-state-target [$F(1, 19) = 36.61$, $p < 0.05$] modulated movement time, with significantly shorter and more accurate movement times imitated in the absence (M = 2156 ms; SD = 387 ms), compared to the presence (M = 2294 ms; SD = 386 ms), of an end-state-target. Although there was no significant difference in movement times when imitating the atypical17 (M = 2121 ms; SD = 382 ms) and atypical26 (M = 2191 ms; SD = 379 ms) models, the main effect [$F(2, 38) = 17.90$, $p < 0.05$] indicated these two movement times were significantly shorter (*ps* < 0.05) and more accurate than

imitating the constant velocity (M = 2362 ms; SD = 399 ms) model. The interaction concerning model and target [$F(2, 38) = 3.51$, $p < 0.05$] indicated that significantly shorter and more accurate movement times were performed in the no-end-state-target compared to the end-state-target condition ($ps < 0.05$) when viewing atypical17 and atypical26 models. This effect was not significant when imitating constant velocity.

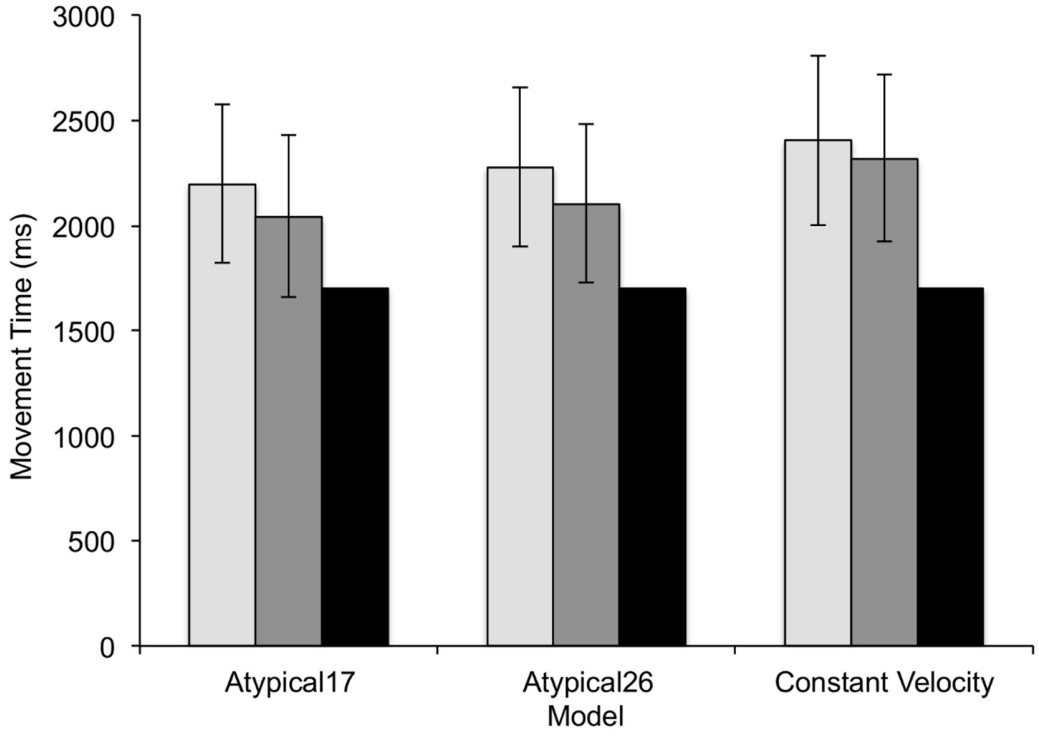


Figure 3.3. Mean movement time data (ms) as a function of model (atypical17, atypical26 and constant velocity) and target condition (light grey = end-state-target; dark grey bar = no-end-state-target). The criterion model data for atypical17 and atypical26 is represented in the black bars. Error bars (±) display the standard error mean.

3.4.2. Peak Velocity

An effect of model [$F(2, 38) = 59.56$, $p < 0.05$] indicated the magnitude of peak velocity was significantly greater when imitating the atypical model (M = 0.24 mm/ms; SD = 0.048 mm/ms) compared to the atypical26 (M = 0.19 mm/ms; SD = 0.036 mm/ms) and constant velocity (M = 0.15 mm/ms; SD = 0.027 mm/ms) models. Moreover, the magnitude of peak velocity was significantly ($p < 0.05$) greater when imitating the atypical26 compared to the constant velocity model. As illustrated in left-hand and centre portions of Figure 3.4, the magnitude of peak velocity executed by the participants in the atypical17 and atypical26 conditions (grey bars) was scaled (i.e., more similar) to peak velocity displayed by the model (black bar). However, peak velocity was not modulated by the presence or absence of an end-state-target [$F(1, 19) = 1.48$, $p > 0.05$], irrespective of how it was combined with the model stimulus [$F(2, 38) = 1.54$, $p > 0.05$].

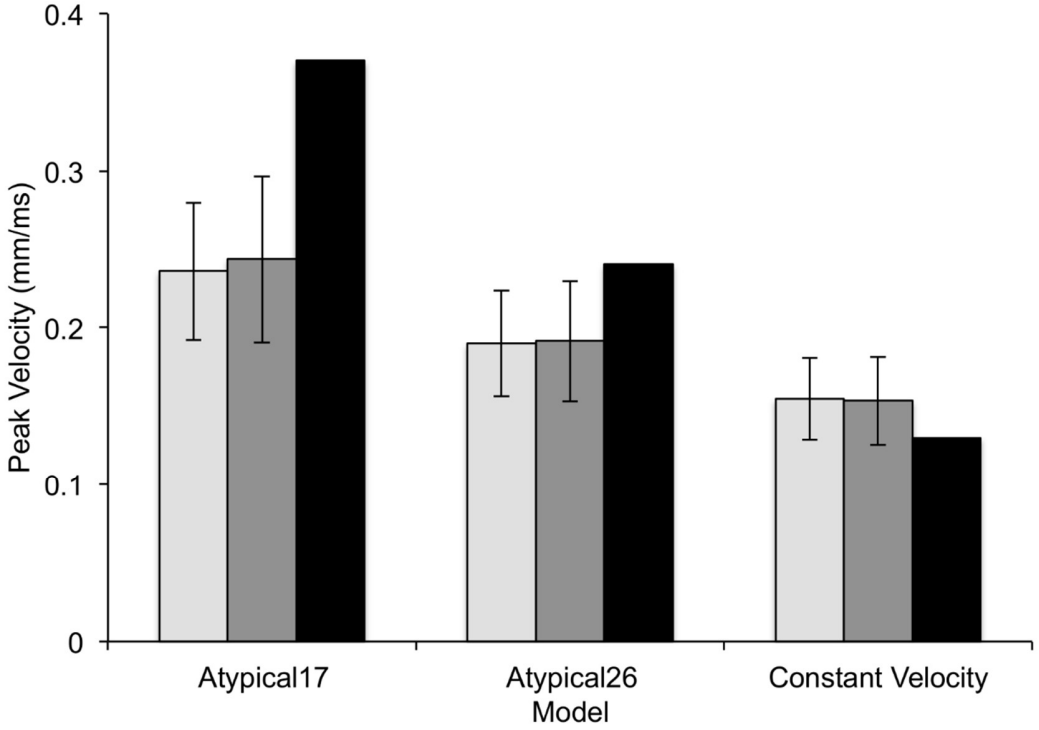


Figure 3.4. Mean peak velocity data (mm/ms) as a function of model and target condition. The target conditions are displayed in the light grey bar (end-state-target) and dark grey bar (no-end-state-target). The criterion model data for atypical17 and atypical26 is represented in the black bars. Error bars (±) display the standard error mean.

3.4.3. Percentage-Time-to-Peak-Velocity

An effect of model [$F(2, 38) = 68.99$, $p < 0.05$] indicated peak velocity occurred significantly earlier in the movement when imitating the atypical17 model (M = 22%; SD = 6%) compared to both the atypical26 (M = 29%; SD = 8%) and constant velocity (M = 38%; 7%) models (*ps* < 0.05). As illustrated in Figure 3.5, the grey bars indicate the temporal occurrence of peak velocity in the atypical17 and atypical26 conditions was scaled to peak velocity displayed by the model (black bar). This effect can also be seen from an exemplar velocity trace in Figure 3.6. When imitating the atypical17 (light grey trace) model, peak velocity occurred significantly earlier in the movement than the atypical26 (dark grey trace) model. When imitating the constant velocity model, peak velocity occurred toward the midpoint of the movement (black trace). Although there was no main effect for target [$F(1, 19) = 1.58$, $p > 0.05$], there was an interaction concerning model and target [$F(2, 38) = 11.40$, $p < 0.05$]. Percentage-time-to-peak-velocity occurred earlier in the movement in the end-state-target condition compared to the no-end-state-target condition when imitating the atypical17 and atypical26 models (*ps* < 0.05). This effect was reversed when imitating constant velocity model.

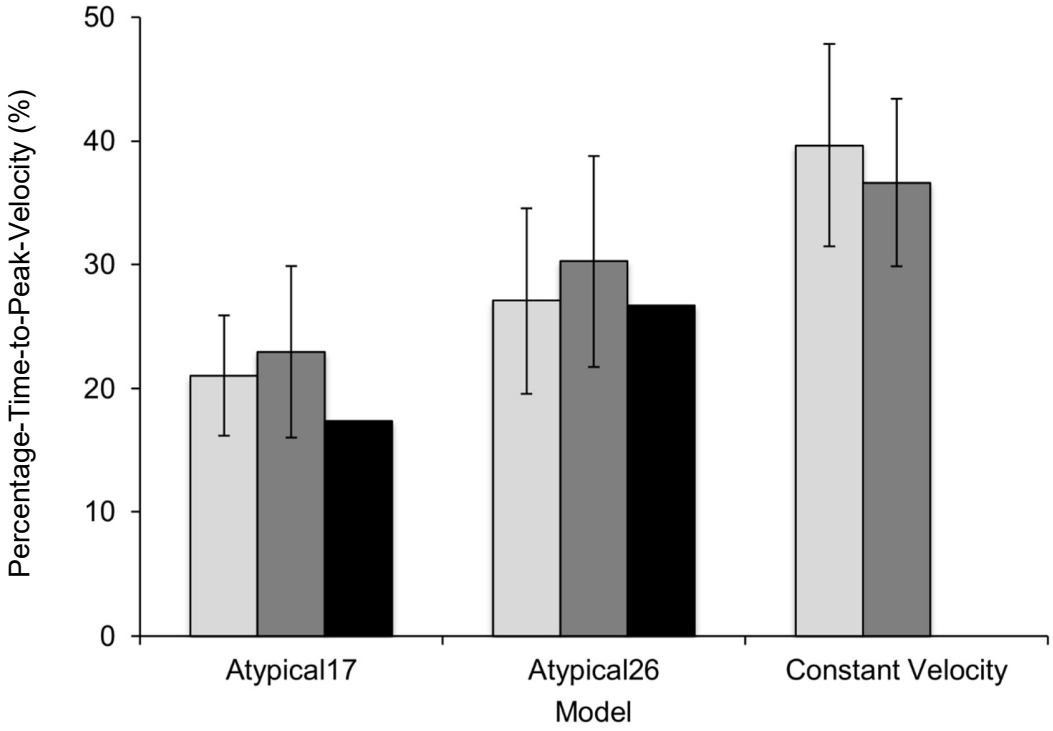


Figure 3.5. Mean percentage-time-to-peak-velocity (%) as a function of model and target condition. The target conditions are displayed in the light grey bar (end-state-target) and dark grey bar (no-end-state-target). The criterion model data for atypical17 and atypical26 is represented in the black bars. Error bars (±) display the standard error mean.

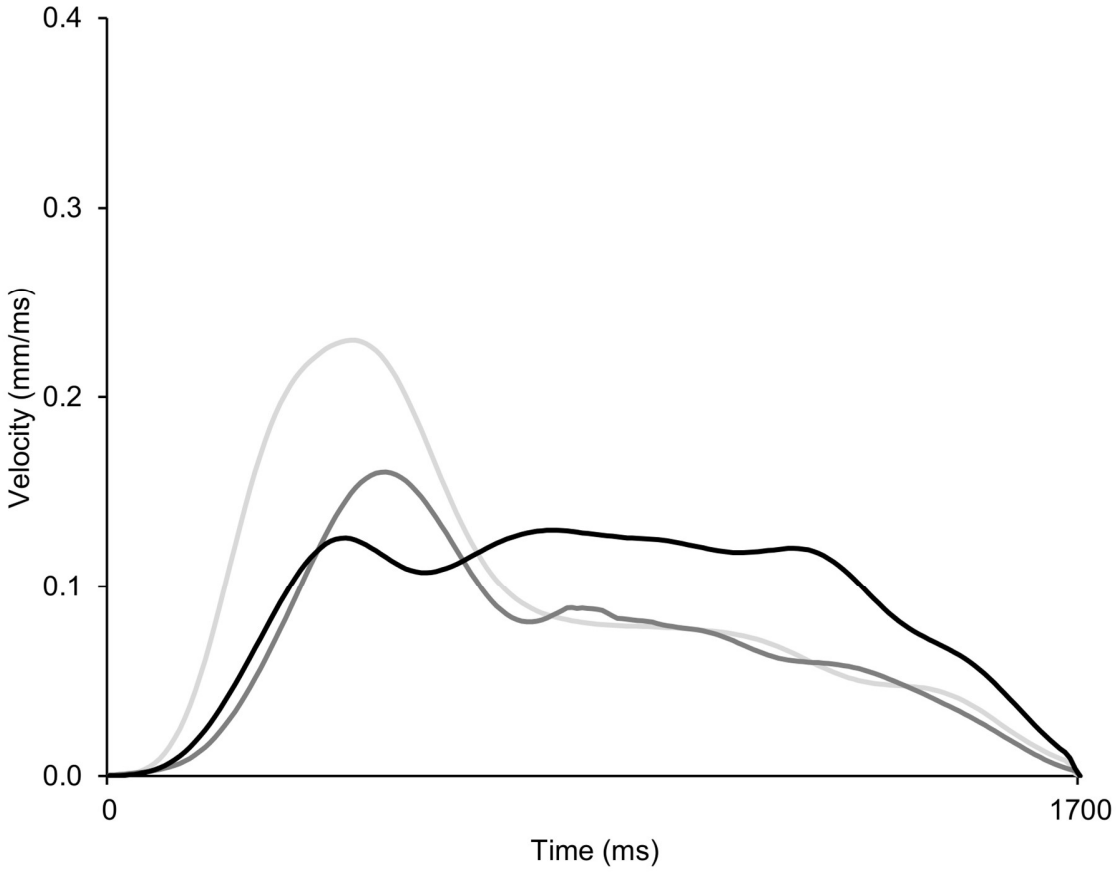


Figure 3.6. Exemplar imitation data of atypical (light grey trace), typical (dark grey trace) and constant (black trace) velocity models, showing peak velocity (mm/ms) and the relative time it occurred (percentage-time-to-peak-velocity) during imitation.

3.5. Discussion

The present chapter examined the representation of biological motion kinematics during imitation learning using a novel protocol that systematically manipulated the structure of a model's kinematic profile. The percentage-time-to-peak-velocity data supported the expectations by indicating peak velocity occurred significantly earlier in the movement after imitating both the atypical17 and atypical26 models. Moreover, while movement time was similar in these conditions, the magnitude of peak velocity also differed in accord with the atypical biological motion models. Imitation of both atypical17 and atypical26 models was confirmed by the data showing participant exhibited peak velocity significantly later in the movement in the constant velocity control condition. Specifically, percentage-of-time-to-peak-velocity occurred closer to the mid-point of the trajectory (38%; see exemplar data displayed in Figure 3.6), which is consistent with what would be expected if participants had imitated a model displaying a typical biological motion profile (Hayes, et al., 2014; Hayes, et al., 2009). Because participants were unable to directly match the constant velocity stimulus due to the anatomical and physiological constraints of the human-motor system (Abend et al. 1982), imitation in the control condition most likely occurred by forming a representation based on the internal (pre-existing motor priors) and external (amplitude and speed of movement) constraints of the task (Elliott, et al., 2001; Roberts, Bennett, Elliott, & Hayes, 2012).

As expected, the findings also showed that imitation learning was modulated by the presence or absence of end-state-targets. Having observed the two atypical biological models in the absence of end-state-targets, participants exhibited shorter movement times, which were more accurate (M = 2156 ms) compared to when end-state-targets were present (M = 2294

ms). As suggested previously (Wild, et al., 2010), this effect was unlikely to be associated with differences in movement amplitude, which was 6 mm shorter when end-state-targets were absent[1]. Neither was it a function of greater average acceleration, which was less in the absence of end-state-targets (i.e., similar peak velocity but achieved later). Although not measured in the present experiment, an explanation for the less accurate imitation of movement time in the presence of end-state-targets is that participants paid more attention (Leighton, et al., 2010) to target attainment and thus were more goal-directed during movement execution. As a consequence, it is likely they focused more on aiming to position the cursor in the end-target, which resulted in proportionately more time after peak velocity in the deceleration phase (Elliott, Hansen, Mendoza, & Tremblay, 2004).

The nature of this top-down attentional effect is important from a theoretical position because the decrease in movement time accuracy when end-state-targets were present did not lead to a concomitant decrease in the imitation of atypical biological motion kinematics. This is consistent with the suggestion that during imitation both lower-level sensorimotor and top-down attentional processes operate in a complementary (Buxbaum & Kalénine, 2010;

Footnote

[1]Additional analyses were conducted to determine if movement time was correlated with movement amplitude. Separate within-participant correlations were run on these two dependent variables for end-state-target and no-end-state-target conditions. For each participant, a correlation was run on movement time and movement amplitude from 42 trials (i.e., 14 trials and 3 velocity models). The logic is that a positive correlation would occur if longer movement times were associated with longer movement amplitudes, and vice versa. The group mean *r* value for the end-state-target condition was 0.27 ± 0.27, and 0.30 ± 0.2 for the no-end-state-target condition. Furthermore, of the 20 participants, 9 had a significant *r* value in the end-state-target condition, and 12 had a significant *r* value no-end-state-target condition. Only 8 of the participants exhibited a significant *r* value in both the end-state-target condition and no-end-state-target condition. In addition, the mean r^2 for the end-state-target condition was 0.14 ± 0.18 and 0.15 ± 0.14 for the no-end-state-target condition, and the coefficient of determination was less than 0.5 for all participants. These analyses indicate no clear trend across participants for a relationship between movement time and amplitude.

de Lange, Spronk, Willems, Toni, & Bekkering, 2008; Heyes, 2011) manner in order to represent biological motion kinematics, as well as other salient factors in the environment (Leighton, et al., 2010).The fact that kinematics and the end-state-target context were coded suggests an equitable contribution of processing which is perhaps less hierarchical than concluded in previous work (Bekkering, et al., 2000; Hamilton, Brindley, & Frith, 2007; Hayes, Hodges, Scott, Horn, & Williams, 2007; Wohlschlager, Gattis, & Bekkering, 2003). The more equitable contribution shown in the present chapter most likely reflected the fact that the to-be-imitated movement kinematics could not be solved by merely recruiting a pre-existing motor pattern.

To minimize the potential modulation of biological motion processing by top-down factors associated with goal coding (Bekkering, et al., 2000), attention/salience (Leighton, et al., 2010), teleological reasoning (Csibra & Gergely, 2007) and social modulation (Wang & Hamilton, 2012), the atypical biological models were observed as non-human agents in the absence of end-state-targets. The finding of temporal correspondence (Gangitano, Mottaghy, & Pascual-Leone, 2001) between observed (atypical17; atypical26) and imitated movement kinematics is therefore consistent with biological motion being processed through lower-level visuomotor processes operating in the human mirror-mechanism (Brass & Heyes, 2005; Casile et al., 2010; Dayan et al., 2007; Press, Cook, Blakemore, & Kilner, 2011). Detection of biological motion is suggested to occur in a neural substrate associated with the pSTS (Allison, Puce, & McCarthy, 2000), while coding the kinematic properties of an observed action (Hamilton, 2008; Iacoboni, 2009) is suggested to occur in the fronto-parietal mirror-system (Di Dio et al., 2013; Press, et al., 2011). Within the fronto-parietal mirror mechanism, the premotor region has been associated with coding the temporal features of visual information through analysis of MEPs during different phases of a grasping action

(Gangitano, et al., 2001). Moreover, evidence that certain phases of movement are reflected in time-synchronized neural activation (e.g., greatest activation during display of maximal grip aperture), has been suggested to indicate online visual processing during observation of biological motion. In line with this reasoning, the data from the current chapter suggest the finding of temporal correspondence between the model and imitation of atypical biological motion was in part based on the online visual processing of such motion during each observation trial. Such findings of continual matching of action-execution with action-observation is consistent with previous work on biological motion coding during observational practice (Hayes, et al., 2014).

In summary, the findings in the present experiment showed atypical biological motion kinematics was represented during imitation learning, both in the presence and absence of end-state-targets. Imitation of biological motion kinematics involves top-down attentional and lower-level visuomotor systems, which operate as complementary processes.

Chapter 4: Eye movements confirm visual attention is stimulus driven during observation of biological motion.

4.1. Abstract

The previous experimental chapters have demonstrated the lower-level coding of biological motion (*Chapter Two*) are influenced by top-down factors related to the goal of the movement, namely end-state-targets (*Chapter Three*). Goal-directed modulation of imitation is related to visual attention becoming more goal-directed during observation, resulting in less visual information from the observed stimulus being coded (Wild et al., 2010). In order to examine eye movement behaviour and strategy, the aim of this study was to record eye movements during observation of the model stimuli, prior to imitation of model stimuli. In this protocol, a modified version of that used in *Chapter Three,* a non-human agent model was used that displayed novel atypical biological motion kinematics, as well as biological and non-biological control models that displayed typical biological motion kinematics and constant velocity respectively. Importantly the three models had the same movement amplitude and movement time. Also, the motion kinematics were displayed in the presence, or absence, of end-state-targets. Kinematic analyses showed atypical biological motion kinematics were imitated, and that this performance was different from the typical and constant velocity control condition. Although the imitation of atypical biological motion kinematics was not modulated by the end-state-targets, movement time was more accurate in the absence, compared to the presence, of an end-state-target. These data replicate the findings from *Chapter Three*. Eye movement analysis showed no difference in visual strategy during observation of model stimuli when end-state-targets were present, compared to when they were absent. Therefore, these data suggest that the coding of biological motion kinematics is a result of tracking the cursor during observation and consequently, associated with lower-level visuomotor processing of the visual stimuli.

4.2. Introduction

Imitation as a learning mechanism is complex, involving the formation and refinement of an internal action model that enables observed biological motion properties to be voluntarily reproduced by the human observer. Imitation learning requires processes that transform and integrate input from observing the visual stimulus, with afferent and efferent inputs that are generated when voluntarily activating the motor system. When the observed biological motion stimulus is novel, these bi-directional processes are said to operate at a lower-level of the CNS, whereby they directly link perception and action (Brass & Heyes, 2005; Heyes, 2011; Iacoboni, 2005). A seminal example of the direct relationship between movement perception and motor execution was reported by Sturmer, Aschersleben and Prinz (2000), who showed response times to opening or closing the hand were shorter when cues were compatible with the response (e.g. open hand stimulus and open hand response) than incompatible (e.g. close hand stimulus and open hand response). Support for sensitivity to coding biological motion during observation of a stimulus was reported in a series of studies by Kilner and colleagues (Kilner, Paulignan & Blakemore, 2003; Kilner, Hamilton & Blakemore, 2007). Both human and robotic arm movements were observed while concurrently executing either congruent or incongruent arm movements. It was predicted that if perception and action shared similar neural circuitry during activation, observing an incongruent movement to that of the executed movement would create interference (e.g. variance from the stimulus). Importantly, significant interference was reported when observing human incongruent movement but not robotic incongruent movement, which suggests the direct link between perception and action is specifically related to the detection of biological motion.

In addition to linking perception and action, the detection of biological motion is also crucial to the ability to learn through observation. Indeed, it has been reported that biological motion perception underpins the ability to differentiate between moving stimuli (i.e., a video clip of a hand moving at slow and fast speeds) and thereby accurate imitation, as evidenced by the participant's movement kinematics relative to the observed model (Wild, Poliakoff, Jerrison & Gowen, 2010). The neural components of this process have been examined using TMS. When observing a human hand complete a reaching-grasping action, MEPs increased in amplitude relative to the amount of observed finger aperture (Gangitano, Mottaghy & Pascual-Leone, 2001). These findings showed that as well as linking action observation and action execution, underlying visuomotor processing in the mirror mechanism also took account of the temporal components of the observed stimuli.

The coding of kinematics has recently been demonstrated by examining biological motion coding during imitation learning (Hayes, Dutoy, Elliott, Gowen & Bennett, 2016). By requiring participants to imitate two slightly different atypical biological motion models (peak velocities that occurred at 17% and 26% respectively), the study sought to determine whether a general representation was formed during observation or if specific representations were developed that reflected the kinematic profile of each model. Imitation of the two atypical models was significantly different from each other and importantly, scaled to the models, thus demonstrating specific kinematic properties of biological motion are coded during imitation learning. Further, and in line with GOADI (Bekkering et al., 2000; Wohlschlager et al., 2003), it was predicted that imitation would be modulated by the presence of end-state-targets such that visual attention and motor output would become goal-directed (Wild et al., 2010). Consequently, when end-state-targets are present it is suggested that goal attainment becomes the primary feature of the action, at the expense of coding stimulus kinematics. Results showed

that end-state-targets modulated the accuracy of movement time, such that it was more accurate when end-state-targets were absent, but not the coding of biological motion kinematics. As the imitation of kinematics was not influenced by the presence of end-state-targets, it was concluded that this process was a function of lower-level visuomotor processing. However, the goal-directed modulation of movement time suggested that top-down attentional systems operated alongside lower-level processing, relative to the environmental context.

It has been suggested that increased imitation effects when end-state-targets are absent could be a function of eye movement strategies during observation (Wild et al., 2010). Due to the less predictable nature of a stimulus moving between undefined start and end points, it follows that more visual attention would be paid to the stimulus, thus leading to subtle differences in movement kinematics being coded through direct visuomotor mapping (Rumiati & Tessari, 2002). Conversely, when end-state-targets are present, it is likely that eye movements would become more predictive and goal-directed, resulting in a larger saccade to the end target and a longer goal-directed fixation (Flanagan & Johansson, 2003). The current chapter was designed to further examine these suggestions. The same novel protocol was used as in previous chapters (*Chapters Two* and *Three*) to examine biological motion coding during imitation learning but now with the addition of eye movement recording during the observation phase of the protocol. Eye movements were recorded to determine eye movement strategies, and thus the location of overt visual attention during observation of the stimulus. The observed stimuli showed either typical (percentage-time-to-peak-velocity = 44%) or atypical (percentage-time-to-peak-velocity = 17%) biological motion models and a constant velocity (non-biological motion) model. The atypical model was replicated from *Chapter Two* whereas the typical model represented a natural aiming movement with a bell-shaped velocity

profile (Elliott, Helsen & Chua, 2001). The constant velocity model was generated based on an equation accounting for time and displacement of the movement. All model stimuli were presented as anon-human agent (white dot) that moved either between two non-defined start and end locations (no-end-state-target condition) or two red targets (end-state-target condition).

As observation and imitation components of the experiment are similar to previous research (Andrew et al., 2016;Hayes et al., 2016), the imitation results are expected to replicate those findings. That is, biological motion kinematics would be imitated such that there would be a difference between the atypical and typical imitation behaviour, with each scaled relative to their respective models. Imitation with end-state-targets is expected to be less accurate than with no-end-state-targets. With respect to eye movements, it is expected that the cursor will be tracked during observation and thus eye movement velocity will closely resemble the velocity profiles of the respective stimuli. This pattern of eye movements would confirm that overt attention was directed to the observed models, thereby providing the opportunity to perceive and code the underlying biological kinematics.

4.3. Methods

4.3.1. Volunteers

Nineteen participants (aged between 18 and 21 years) volunteered for the study. All participants were right-hand dominant, had normal or corrected-to-normal vision and gave written informed consent. The experiment was designed in accordance with the 1964

Declaration of Helsinki and approved by the research ethics committee of the host University.

4.3.2. Apparatus and Stimuli

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 890 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 4.1A). 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.).

Eye movements of both groups were recorded using the EyeLink1000 (SR Research Ltd., Mississauga, Ontario, Canada), which sampled eye gaze locations in the horizontal and vertical axes at 1000 Hz. Data was stored for off-line analysis with routines written in MATLAB. A chin and forehead rest was used to minimise head movement, and to ensure that participant eyes were located 890 mm perpendicular to the centre of the computer monitor. At this distance, the cursor subtended a visual angle of 13°. A nine-point calibration and validation of gaze location accuracy occurred prior to the pre- and post-test.

4.3.3. Procedure

The imitation task required participants to observe and imitate non-human agent models that displayed a single horizontal trajectory that originated from a home-position on the left side of the monitor and terminated at an end-position on the right side of the monitor (Figure 4.1). The movement amplitude of a model was 200 mm and total duration was 1700 ms. To examine imitation of biological motion, two models were created that displayed typical or atypical velocity profiles (Hayes et al., 2015; 2016; Andrew et al., 2016). The typical model was created by a human volunteer and displayed a natural (Flash & Hogan, 1985; Elliott et al., 2010) bell-shaped velocity profile (dark grey trace in Figure 4.2) with a peak velocity of 0.200 mm/ms that occurred at 44 % of the movement duration. The atypical model (solid-black trace in Figure 4.2) was created by the same volunteer and displayed a novel kinematic trajectory, with a peak velocity of 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 were biological. In addition to the biological motion models, a constant velocity model was used to create a non-biological control condition (light grey trace in Figure 4.2). This non-biological model was computer-generated and moved at a uniform velocity of 0.120 mm/ms across the same 200 mm amplitude and 1700 ms duration as the biological motion models. During trials with end-state-targets, two red circles representing home-target and end-state-target (diameter = 16 mm) were positioned at left (home) and right (end) sides of the monitor respectively. During trials with no-end-state-targets, only one red circle was present and represented the home position to encourage a consistent start location for all imitation trials.

Prior to a familiarisation period that replicated the task requirements of the imitation protocol (e.g. operating the stylus, start location of each trial, controlling the cursor, ending

the imitation trial), participants were informed that their task was to “watch the movement of the dot, with the intention to then imitate it as accurately as possible”. Then, during the 6 familiarisation trials, participants observed a non-human agent model move from the home-position with a constant velocity to the end-position. The model displayed the exact movement duration and amplitude of the experimental models, but with a constant velocity in the horizontal x axis of 0.120 mm/ms. There were no deviations in the perpendicular y axis. Using this model ensured construct validity by preventing participants experiencing biological motion before the imitation trials. Participants were not informed about the duration of the movement, or the type of stimuli. After observing the model, participants imitated by moving the stylus on the tablet so that a cursor displayed on the monitor moved from the home-position to the end-position as per the model. Following movement execution, there was a 4000 ms inter-trial delay before the next trials commenced. All participants confirmed they understood the instructions on how to observe and imitate the trajectory of the model, and the sensorimotor association between the stylus on the graphics tablet and the corresponding movement of the cursor on the monitor. Participants then performed the imitation protocol that consisted of 14 blocks of 6 trials. A block contained two typical and two atypical biological motion trials, as well as two non-biological motion trials; of the two trials for each model, end-state-targets were either absent or present. Trial order within a block, as well as block order, was randomised across volunteers. The randomised structure reduced predictability of an upcoming model(s) and end-state-target presence and promoted imitation on a trial-by-trial basis.

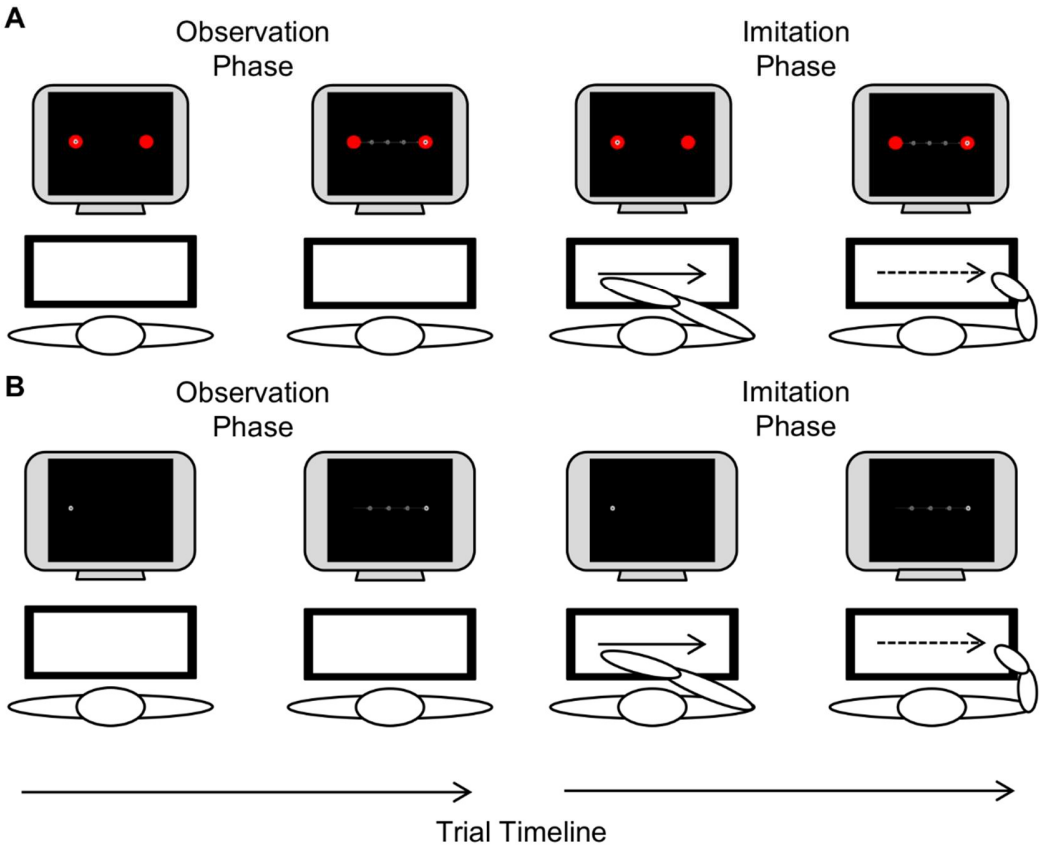


Figure 4.1. A visual representation depicting a single trial in the end-state-target-condition (A) and no-end-state-target condition (B). The apparatus outlined in Panel A and B is a CRT monitor and a graphics tablet. The trial timeline arrows at the bottom of the figure indicate the Observation Phase and Imitation Phase. During the Observation Phase, the non-human agent model is positioned in the left-hand home target (A) and left-hand space (B). The model (atypical, typical or constant velocity) displays a horizontal movement of 200 mm from the left-hand home target to an end-state-target (A) or end-space in the no-end-state-target-condition. The model has a movement time of 1700 ms. The Imitation Phase commences with the white cursor positioned in left-hand home target (A) or left-hand space (B). A participant imitates the observed model by controlling a stylus on the graphics tablet.

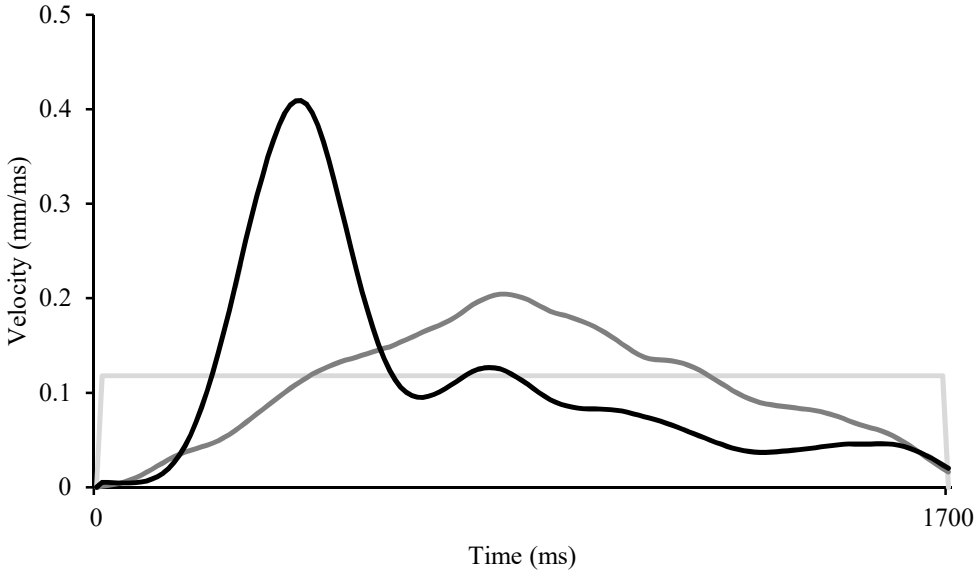


Figure 4.2. The velocity profiles for atypical model (black trace), typical model (dark grey trace), and constant velocity control models (light grey trace).

4.3.4. Data Reduction

4.3.4.1. Imitation

To quantify imitation of movement kinematics, analysis focussed on x-axis data only. Within the x-axes, position data was identified at the start and end of the movement in each imitation trial. The start was identified by the cursor moving beyond the perimeter of the home-position, while the end was identified when the participant clicked the lower-button on the stylus. From this identification process, the position data was filtered using a low pass 4th order autoregressive filter with an 8 Hz cut-off. The filtered data were then differentiated using a 3-point central difference algorithm to obtain velocity. A MATLAB routine extracted the movement and displayed the velocity curve for each trial. Using a mouse, an experimenter manually selected the start, peak, and end of the movement on the velocity curve to identify general regions around which the MATLAB program would then identify the exact locations

based on predetermined velocity criteria.. The MATLAB routine integrated the velocity curve to identify the start of the movement when velocity was > 0.003 mm/ms, and the end when velocity was < 0.003 mm/ms. Movement time, peak velocity and percentage-time-to-peak-velocity from each trial was quantified. Intra-participant means were calculated from 10 trials associated with each model and origin of movement (e.g., 10 trials for the atypical model in the left-to-right origin). 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; Andrew et al., 2016).

4.3.4.2. Eye Movements

Eye gaze locations were low-pass filtered using a zero-phase digital filter (autoregressive; forward and backward filter; cut-off frequency, 35 Hz). Eye velocity and acceleration were then derived from eye position data using a three-point central difference algorithm. Next, saccades were identified and removed from the smooth response using a technique described in previous research (Bennett & Barnes, 2003). Saccades were identified as points in the acceleration trace exceeding a threshold of 750°/s^2. When the threshold criteria were exceeded, the complete saccade trajectory was identified by finding the peak and trough of acceleration. On the rare occasions when the use of the acceleration threshold failed to identify a saccade, these were identified by a second pass in which a maximum velocity criteria of 30°/s was applied. By using these criteria, saccades of 0.3°or more were reliably detected and segregated from blinks and other noise. Saccades were generally of small amplitude and brief duration, so linear interpolation was used as a simple and adequate method

of waveform restoration. To obtain desaccaded smooth eye velocity, data points equivalent to 12ms at the beginning and end of the identified saccade trajectory were excluded to ensure that no saccadic element remained when applying subsequent interpolation. A linear interpolation routine was used to bridge the gaps produced by removal of saccades from the eye velocity trajectory. The desaccaded eye velocity data were then filtered at 35 Hz with a low-pass, zero-phase filter. This extraction process generated a smooth velocity trace of the eye movement recorded during observation of the stimuli. From this trace, peak velocity and percentage-time-to-peak velocity were extracted from each trial.

4.3.5. Data Analysis

Intra-participant mean data for each dependent variable were submitted to separate 3 Model (atypical; typical; constant velocity) x 2 Goal (end-state-target; no-end-state-target) 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 $p < 0.05$.

4.4. Results

4.4.1. Imitation Data

4.4.1.1. Movement Time

The presence of an end-state-target [$F(1, 18) = 15.29$, $p < 0.05$] modulated movement time, with significantly shorter and more accurate movement times imitated in the absence (M = 2663 ms; SD =431), compared to the presence (M = 2815 ms; SD = 420 ms), of an end-

state-target. An effect of model [$F(2,36) = 89.61$, $p < 0.05$] indicated movement time was significantly shorter when imitating the atypical (M = 2502 ms; SD =379 ms) than the typical (M = 2625 ms; SD =403 ms) and constant velocity (M = 3089 ms; SD = 495 ms) models. Moreover, movement time was significantly shorter ($p < 0.05$) when imitating the typical, compared with the constant velocity model. As seen in Figure 4.3, there was no interaction [$F(2, 36) = 1.47$, $p > 0.05$] concerning any combinations of model and target.

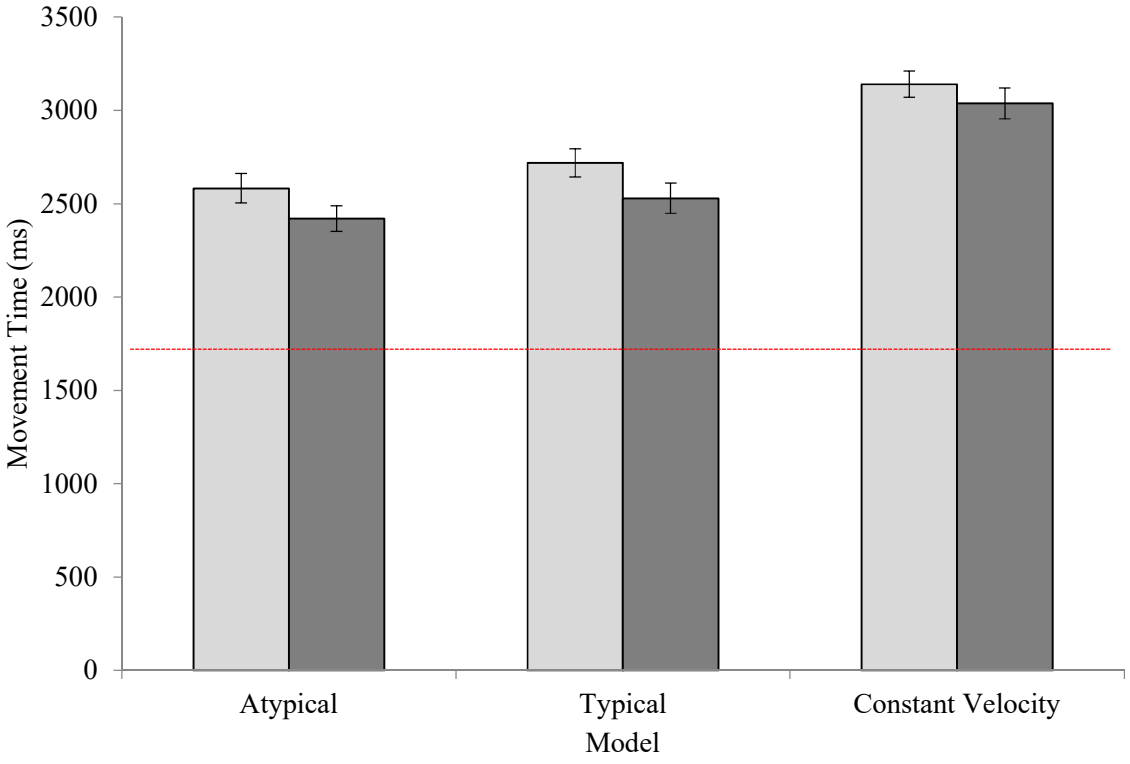


Figure 4.3. Mean movement time data (ms) as a function of model (atypical, typical and constant velocity) and target condition (light grey = end-state-target; dark grey bar = no-end-state-target). Model data is displayed by the dashed red line and error bars (±) display the standard error mean.

4.4.1.2. Peak Velocity

An effect of model [$F(2, 36) = 91.05$, $p < 0.05$] indicated the magnitude of peak velocity was significantly greater when imitating the atypical model (M = 0.17 mm/s; SD = 0.04 mm/ms) compared to the typical (M = 0.12 mm/s; SD = 0.02 mm/ms) and constant velocity (M = 0.1 mm/s; SD = 0.01 mm/ms) models. Additionally, the magnitude of peak velocity was significantly greater ($p < 0.05$) when imitating the typical compared to the constant velocity model. As seen in Figure 4.4, peak velocity was not modulated by the presence of targets [$F(1, 18) = 1.48$, $p > 0.05$] and there was no significant interaction between the presence of targets and the model stimuli [$F(2, 36) = 1.42$, $p > 0.05$].

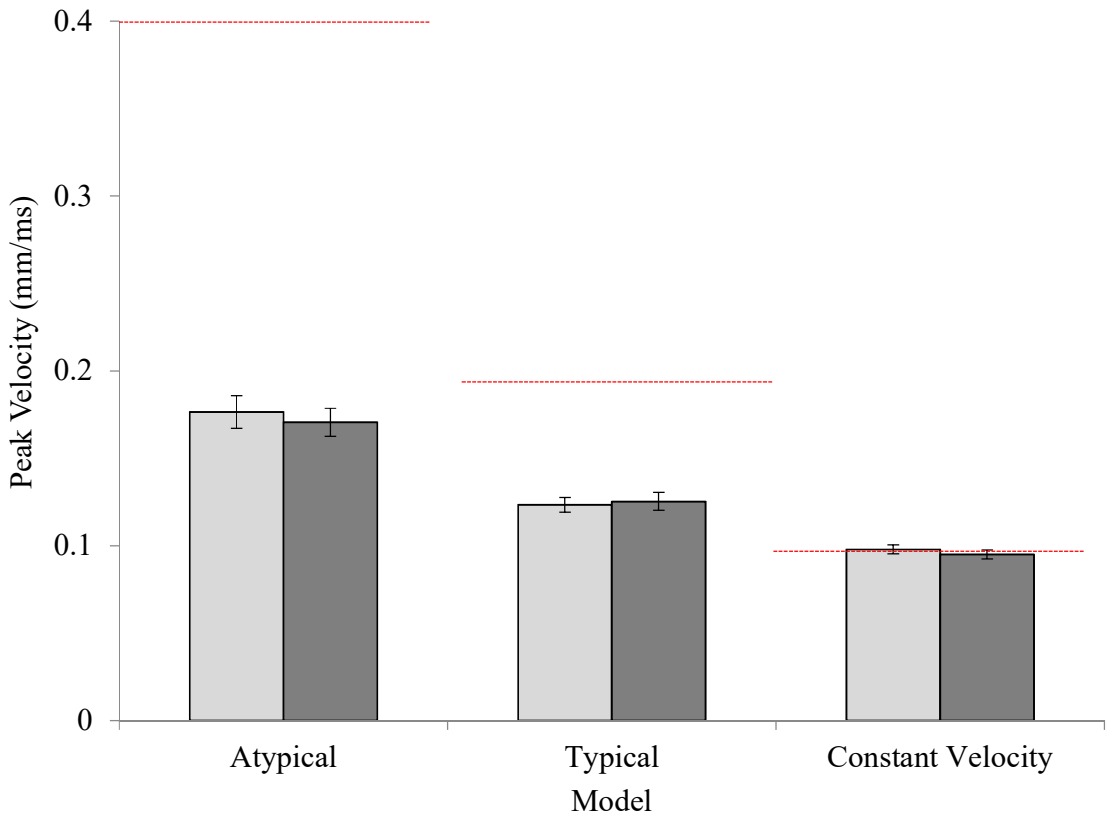


Figure 4.4. Mean peak velocity data (mm/ms) as a function of model (atypical, typical and constant velocity) and target condition (light grey = end-state-target; dark grey bar = no-end-state-target). Model data is displayed by the dashed red line and error bars (±) display the standard error mean.

4.4.1.3. Percentage-Time-to-Peak-Velocity

An effect of model [$F(2, 36) = 88.37$, $p < 0.05$) indicated peak velocity occurred significantly earlier when imitating the atypical model (M = 25%; SD = 11%) compared to the typical (M = 39%; SD =14%) and constant velocity (M = 44%; SD = 17%) models. Peak velocity also occurred significantly earlier ($p < 0.05$) when imitating the typical compared to the constant velocity model. Although there was no main effect for targets ($F_{1, 18} = 0.55$, $p > 0.05$) there was an interaction between model and target ($F_{2, 36} = 4.02$, $p < 0.05$). As seen in Figure 4.5, percentage-time-to-peak-velocity occurred earlier in the movement in the end-state-target condition compared with the no-end-state-target condition when imitating the typical and constant velocity models (ps < 0.05). This effect was reversed when imitating the atypical model.

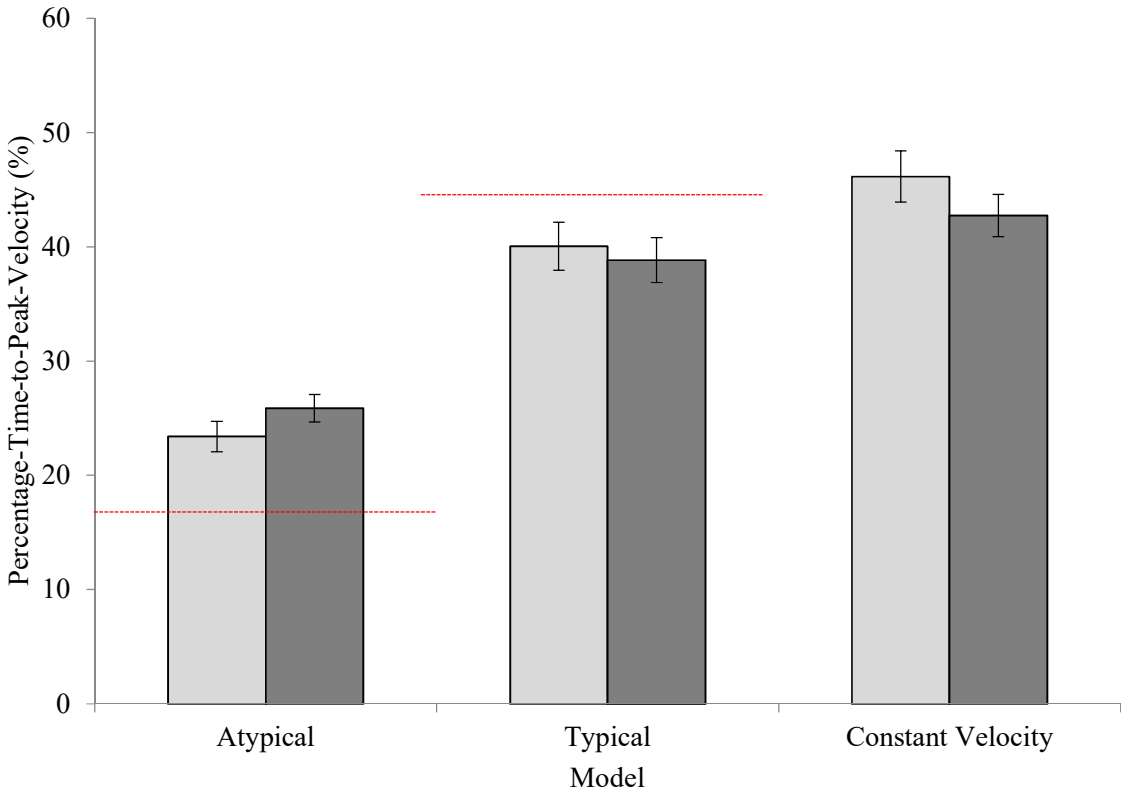


Figure 4.5. Mean percentage-time-to-peak-velocity data (%) as a function of model (atypical, typical and constant velocity) and target condition (light grey = end-state-target; dark grey bar = no-end-state-target). Model data is displayed by the dashed red line and error bars (±) display the standard error mean.

4.4.2. Eye Movement Data

4.4.2.1. Magnitude of Peak Velocity

As seen in Figure 4.6, a main effect of model [$F(2, 22) = 4.26$, $p < 0.05$] indicated that peak eye velocity was significantly greater when observing the atypical model (M = 12.03°/ s; SD = 4.20°/ s) compared to the typical (M = 10.35°/ s; SD = 3.06°/ s) and constant velocity (M = 9.50°/ s; SD = 2.19°/ s) models. However, the presence of targets did not modulate [F(1,

11) = 0.01, $p > 0.05$] the magnitude of peak velocity and there was no significant interaction between targets and the model stimuli [F(2, 22) = 0.92, $p > 0.05$].

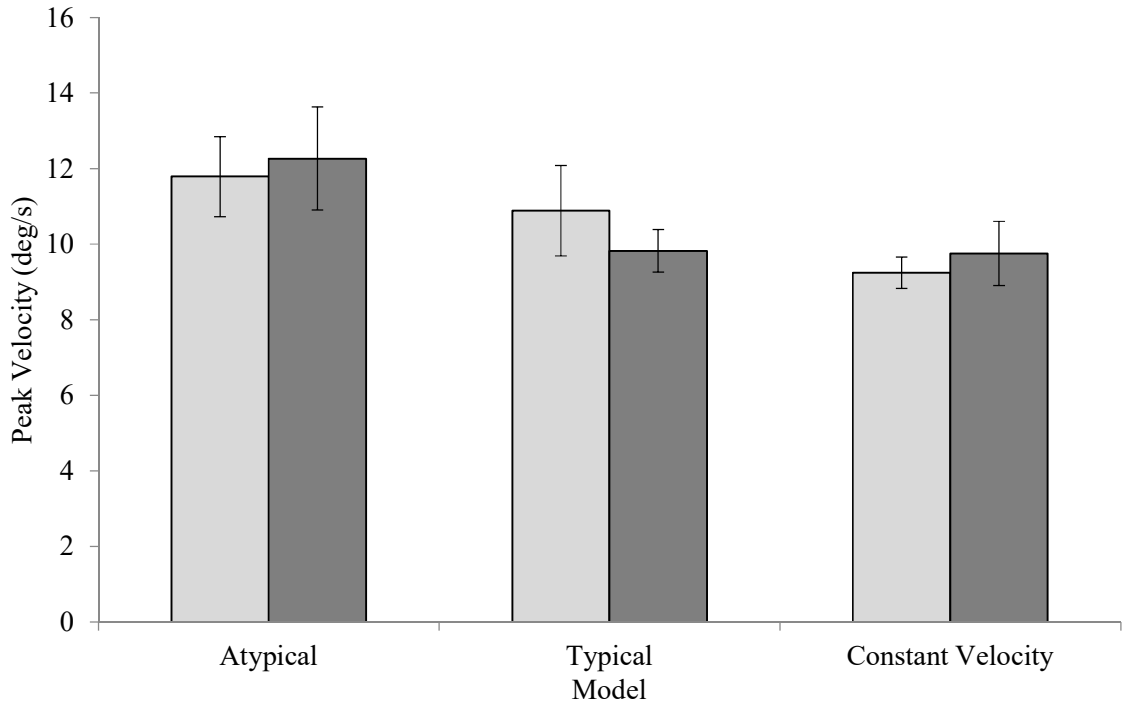


Figure 4.6. Mean peak velocity eye movement data (degrees/s) as a function of model (atypical, typical and constant velocity) and target condition (light grey = end-state-target; dark grey bar = no-end-state-target). Error bars (±) display the standard error mean.

4.4.2.2. Percentage-Time-to-Peak-Velocity

An effect of model [$F(2, 22) = 3.83$, $p < 0.05$] indicated peak eye velocity occurred significantly earlier in the movement when observing the atypical model (M = 40%; SD = 10%) compared to the typical (M = 52%; SD = 13%) and constant velocity (M = 44%; SD = 12%) models. However, as seen in Figure 4.7, the time of peak eye velocity was not modulated by the presence of targets [$F(1, 11) = 1.75$, $p > 0.05$] and there was no significant interaction between targets and the model stimuli ($F(2, 22) = 1.57$, $p > 0.05$].

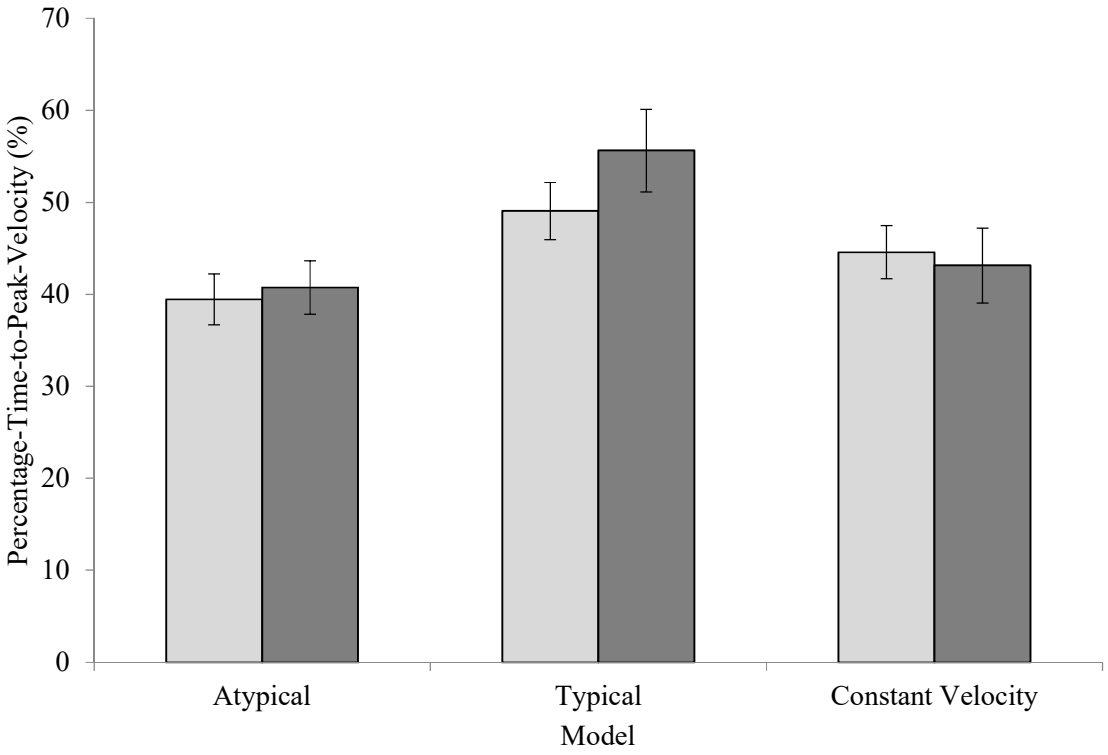


Figure 4.7. Mean percentage-time-to-peak-velocity eye movement data (%) as a function of model (atypical, typical and constant velocity) and target condition (light grey = end-state-target; dark grey bar = no-end-state-target). Error bars (±) display the standard error mean.

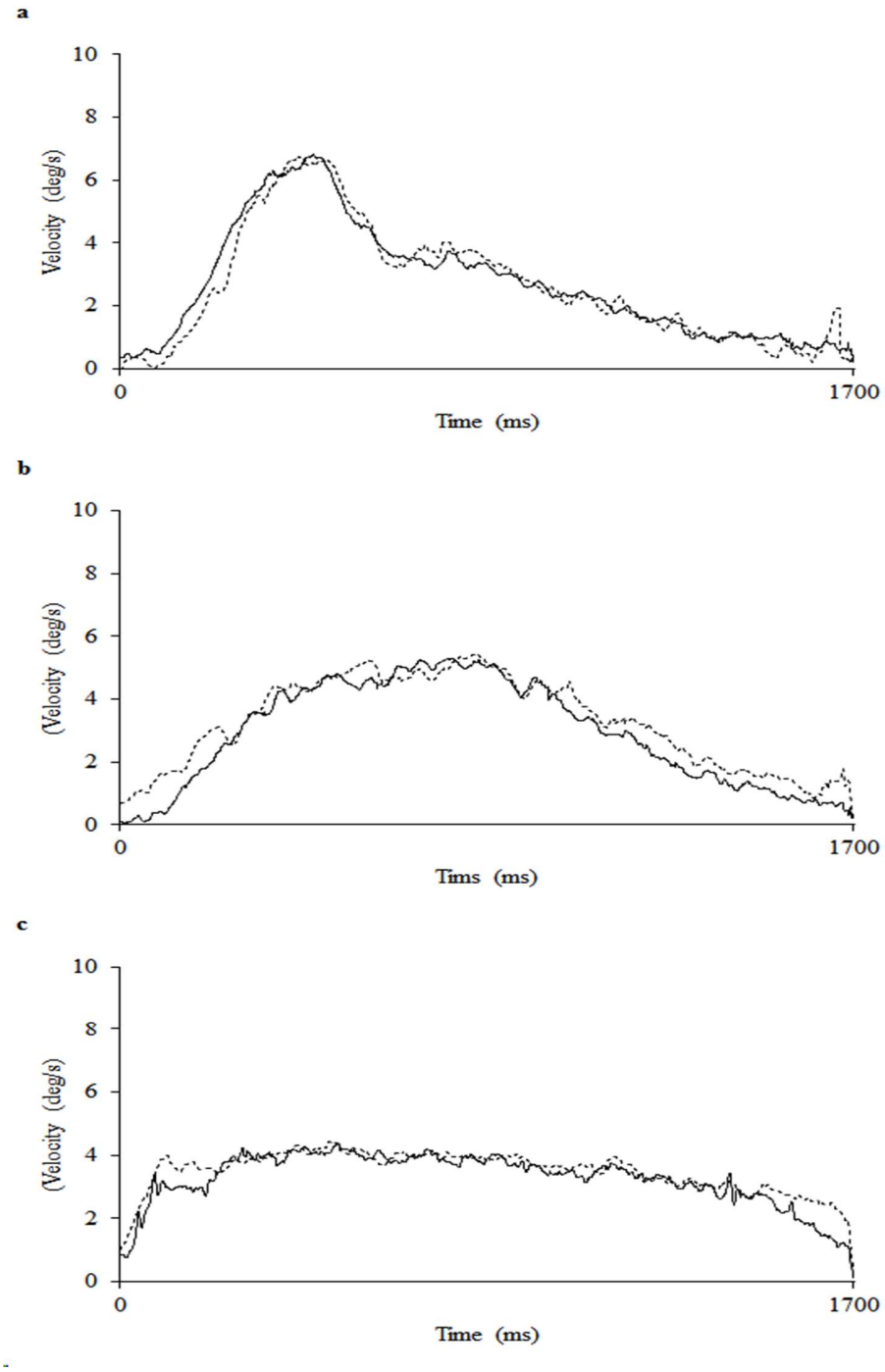


Figure 4.8. Exemplar traces of eye movement velocities (degrees/ s) during observation of the atypical (A), typical (B) and constant velocity (C) models. The dashed black line depicts trials where targets were present, and the solid black line depicts trials were targets were absent.

4.5. Discussion

The aim of the present chapter was to examine the underlying stimulus features that facilitate the coding of atypical biological motion during imitation learning, and whether these influence eye movements, and thereby the locus of overt attention. Consistent with previous work (Hayes et al., 2016; Hayes, Roberts, Elliott & Bennett, 2014) and those of the current thesis, the results demonstrate biological motion coding during imitation learning, such that peak velocity was greater and percentage-time-to-peak-velocity occurred earlier when imitating the atypical than the typical model. In other words, imitation of kinematics did not simply reflect the natural constraints of the task (e.g. bell-shaped velocity profile). These results concur with the suggestion that accurate coding and representation of biological motion during imitation learning occurs through lower-level sensorimotor processes (Brass & Heyes, 2005; Press, Cook, Blakemore & Kilner, 2011). Further, it was found that the presence of end-state-targets modulated imitation similarly to previous research (Hayes et al., 2016). Movement time was more accurate to that of the model when end-state-targets were not present relative to when they were present, yet kinematics were not modulated. It was suggested that this finding was a consequence of increased visual attention to target attainment, and thus greater focus was on moving the cursor to the target after peak velocity had been achieved (Elliott, Hansen, Mendoza & Tremblay, 2004).

The eye movement data of the current chapter provides further insight into the process of imitation, and more specifically where overt visual attention is directed during observation. Results showed that, like the imitation data, eye movements had greater magnitude of peak velocity, which occurred earlier in the observation when watching atypical, compared with

typical and constant velocity models. Further, these effects were consistent regardless of the presence of targets during observation. Thus, the velocity profile of the eyes suggest participants were attempting to track the stimuli. By evidencing tracking of the cursor during observation, these data confirm that during the observation phase of the experiment, participants had the opportunity to extract and code the salient velocity characteristics from the respective models. This finding may have been facilitated by the instructions given prior to the experiment, whereby participants were told to observe the model with the intention of imitating it as accurately as they could (Hayes et al., 2014; 2016). Indeed, by continuing to track the cursor throughout each trial, the direct correlated sensorimotor experience between observation and imitation would have become stronger over time (Heyes, 2011), thereby underpinning accurate imitation of the respective models. Moreover, when visual attention is engaged on a specific feature, for example the trajectory of the model stimuli, it is suggested that information processing is up-regulated during the early stages of skill acquisition (Higuchi, Holle, Roberts, Eickhoff & Vogt, 2012). Therefore, up-regulation could have facilitated the formation of internal representations based on task characteristics such as kinematics (e.g. peak velocity, percentage-time-to-peak-velocity) or temporal features (e.g. movement time).

In addition to general tracking of the model stimuli, eye movement data further demonstrates tracking of model trajectories is consistent both when end-state-targets are absent and present. These findings differ from previous research that showed eye movements become more goal-directed when end-state-targets are present during a task and moreover, that this results in the modulation of biological motion coding (Wild et al., 2010). Consequently, authors suggested that the lower-level processes involved in imitation of kinematics, and top-down modulation associated with visual attention and end-state-targets,

were exclusive processes that operated independently. In addition to not reporting goal-directed eye movements, results from the current chapter also show only movement time, and not kinematics, are modulated by the presence of end-state-targets. These effects corroborate previous research that has shown a general acquisition of kinematics, but modulation of movement time, during a learning task with varying levels of feedback (Andrew et al., 2016). In line with results in the present chapter, these findings demonstrate that various higher-order cognitive systems (e.g. visual attention, end-state-targets, feedback) can influence the processing of temporal components of the stimuli during observation of the models and thus, not only lower-level, but additional top-down processes are involved in the coding of biological motion during imitation learning.

In summary, the findings in the present study showed atypical biological motion kinematics were coded and represented during imitation learning. Further, eye movements confirmed that visual attention was directed to the stimuli during observation, thus providing the opportunity to perceive and code and consequently, confirms previous assumptions that imitation of biological motion kinematics through lower-level visuomotor processing.

Chapter 5: Social Attitudes Modulate Biological Motion Coding During Imitation Learning

5.1. Abstract

Imitation of novel kinematics has been shown to be a function of lower-level processing (*Chapter Two*), which can be modulated by top-down factors related to end-state-targets (*Chapter Three*), attention and social context. In order to further examine the potential interaction between lower-level and top-down processes during imitation learning, the aim of this study was to systematically control the social context during the imitation protocol. In this protocol, a non-human agent model was used that displayed different novel atypical biological motion kinematics, as well as a control model that displayed constant velocity. Importantly the three models had the same movement amplitude and movement time. Prior to observation of the models, a social prime displaying either neutral (no eye gaze), pro- (direct eye gaze), or anti-social (averted eye gaze) eye gaze was presented on the monitor. Kinematic analyses showed atypical biological motion kinematics were imitated, and that this performance was different from the typical and constant velocity control conditions. Although imitation accuracy of atypical biological motion kinematics was not modulated by the social primes, the variability of imitation reduced after viewing pro- and anti-social primes, relative to the neutral prime. The fact that social primes modulated imitation variability, but not imitation accuracy, indicates observation of social primes resonated with the fidelity of the representations formed during observation, such that the corresponding motor representation were produced with greater accuracy relative to the explicit kinematic features of the models.

5.2. Introduction

Imitation of an observed action can accelerate the acquisition attainment of complex, novel motor skills (Hayes, Elliott & Bennett, 2010) and their respective movement kinematics (Wild et al., 2010; Hayes, Dutoy, Elliott, Gowen & Bennett, 2016). Imitation of observed actions engages sensorimotor processes (Heyes, 2001) that occur in the fronto-parietal mirror-mechanism (Fadiga et al., 2000; Rizzolatti & Craighero 2004). This region of the brain is known to link perception and action (Rizzolatti, 2005; Rizzolatti & Foagssi, 2014). In addition to actions, imitation is also an important feature of developing social interaction and communication, as demonstrated in the mirroring of behaviours such as posture, facial expressions, language and understanding (Hurley & Chater, 2005; Chartrand & van Baaren, 2009).

When the observed and executed actions share spatial alignment, the neural connection between perception and action generally enables and facilitates imitation; however; when they do not share spatial alignment, results have shown there to be interference effects. For example, greater movement deviation occurs when observing a stimulus move orthogonally to the concurrent arm movement e.g. observing horizontal arm movements while concurrently making vertical arm movements (Kilner, Paulignan & Blakemore, 2003; Chaminade, et al., 2005). This interference effect was termed ‘motor contagion’ (Blakemore & Frith, 2005) and refers to the co-activation of incompatible internal representations. This process is largely subconscious and consequently, governed by lower-level processes (Rizzolatti, Fogassi & Gallesse, 2001; Iacoboni, 2005).

However, it has also been shown that these lower-level processes associated with automaticity can be modulated by top-down processes associated with social cognition (Cook & Bird, 2011; Wang & Hamilton, 2011). For example, completing a word scramble task modulated the accuracy and speed with which imitation was made in a subsequent stimulus response compatibility (SRC) protocol (Leighton, Bird, Orsini & Heyes, 2010). When the word scramble displayed pro-social words (friend, together, assist), the accuracy of imitation and speed of reaction time improved. Conversely, imitation accuracy and reaction time deteriorated following anti-social word scrambles (independent, apart, single). It was concluded that social primes act as modulatory top-down processes that influence the lower-level processing of visual information. That is, pro-social cues down-regulate, and anti-social cues up-regulate, inhibitory processes operating in the SRC protocol (Cook & Bird, 2011).

The notion of top-down social processes modulating imitation is the foundation of the social top-down response modulation (STORM) model (Lakin & Chartrand, 2003; Wang & Hamilton, 2012). STORM suggests that imitation, or lack thereof, can enhance or inhibit a given social situation. For example, the "Chameleon effect" (Chartrand & Bargh, 1999) suggests that imitating actions or behaviours, such as body language, gestures and postures (Chartrand & van Baaren, 2009), can result in increased rapport and feelings of closeness (van Baaren, Holland, Kawakami & can Knippenberg, 2004) between people. However, any social repercussions are dependent on the imitative behaviours being detected by the partner. Eye gaze is considered a critical social cue that conveys important social knowledge, understanding and confirms visual attention. As such, they have been extensively examined in the context of identifying imitative behaviours (Perrett, Smith, Potter, Mistlin, Head, Milner et al., 1985; Pelphrey, Morris & McCarthy, 2005; Wang, Ramsey & Hamilton, 2011;).

A recent study combined a classic SRC protocol (Heyes et al., 2005) with an eye gaze social prime (Wang & Hamilton, 2011). The imitation task required participants to open or close their hand in response to a hand-opening or hand-closing stimuli. Prior to the imitation task, a spatially aligned video clip showed a head movement that displayed either a direct (pro-social) or averted (anti-social) eye gaze prime. Results showed that observing direct gaze improved reaction time of hand movements compared to observing averted gaze. Similarly, when the hand and the eye gaze primes were spatially decoupled, such that the hand appeared to the side of the face, direct gaze was again the only social prime that enhanced imitation (Wang & Hamilton, 2014). These findings suggest that imitation can be modulated by social engagement cues and that eye gaze could drive the degree to which an action is imitated.

Together these findings contribute to the credible interpretation of social context directly modulating imitation. It is important to recognise, as has been previously highlighted (Roberts, Bennett, Elliott & Hayes, 2015), that the discussion of visuomotor mapping was in relation to observing a human hand and subsequent changes in reaction time. This is somewhat different from the observation of continuous biological motion and analysis of biological motion properties. Therefore, while the research by Wang and Hamilton is well-conducted and informative with regard to the social priming of reaction time, it does not provide any insight into the underlying processes associated with the imitation of biological motion e.g. whether it is a function of lower-level visuomotor processing related to kinematics e.g. being topographically similar (Catmur & Heyes, 2011) or top-down modulation based on the goal or spatial end-point of the movement (e.g., movement compatibility; Brass, Bekkering & Prinz, 2001).

The present chapter examined social modulation of biological motion coding during imitation learning. The same experimental protocol and imitation task as in *Chapters Three*

and *Four* was used, but with target goals removed from all trials to control for any additional top-down goal-related modulatory effects. In addition, a pro-social (direct gaze), anti-social (averted gaze) or neutral (no model) human model prime (Wang & Hamilton, 2011) was displayed prior to observation of the model stimuli. The model stimuli were also consistent with those from the previous chapter; that is, atypical and typical biological motions, as well as non-biological constant velocity models. If biological motion is coded during imitation learning, it is expected that imitation of the atypical model will be different to that of the typical model. Further, if social primes modulate the processing of biological motion, there are two additional expectations. First, the coding of biological motion kinematics will be more accurate following the pro-social primes and less accurate following the anti-social primes (Leighton et al., 2010). Second, in addition to imitation accuracy, social primes have also modulated movement variability, as demonstrated during online imitation of congruent and incongruent arm movements (Roberts et al., 2015). If this effect is applicable to imitation learning, movement variability is likely to be greater following the anti-social primes than the neutral or pro-social primes (Roberts et al., 2015).

5.3. Methods

5.3.1. Volunteers

Nineteen participants (aged 18-21 years) volunteered for the study. All participants were right-hand dominant, had normal or corrected-to-normal vision and gave written informed consent. The experiment was designed in accordance with the 1964 Declaration of Helsinki and approved by the research ethics committee of the host University.

5.3.2. Apparatus and Stimuli

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). 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.). The social prime images [120 mm (h) x 160 mm (w)] were integrated into the experimental procedure and displayed in the centre of the monitor.

5.3.3. Procedure

The imitation task required participants to first view an image depicting one of the three social primes, then observe and imitate non-human agent models that displayed a single horizontal trajectory that originated from a home-position on the left-hand side of the monitor and terminated at an end-position on the right-hand side of the monitor (as in *Chapters Three* and *Four*). The movement amplitude of the models was 200 mm and total duration was 1700 ms. As in *Chapters Two* and *Four*, biological motion was examined by comparing imitation of two models that displayed typical or atypical velocity profiles (Hayes et al., 2016; Andrew et al., 2016). The typical model (dark grey trace in Figure 5.1D) displayed a natural (Elliott et al., 2010) bell-shaped velocity profile with a peak velocity of 0.200 mm/ms that occurred at 44 % of the movement duration and was designed as a control condition when examining

biological motion coding. The atypical model (black trace in Figure 5.1D) was created by the same volunteer and displayed atypical velocity profile with a peak velocity of 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 were biological. In addition to two biological motion models, a non-biological constant velocity model (light grey trace in Figure 5.1D) was included to act as a stimulus control for any potential biological tuning and expectation of the nature of the stimuli. This model also had an amplitude of 200 mm and duration of 1700ms, thus resulting in a constant velocity of 0.120 mm/ms, and no y-axis deviation.

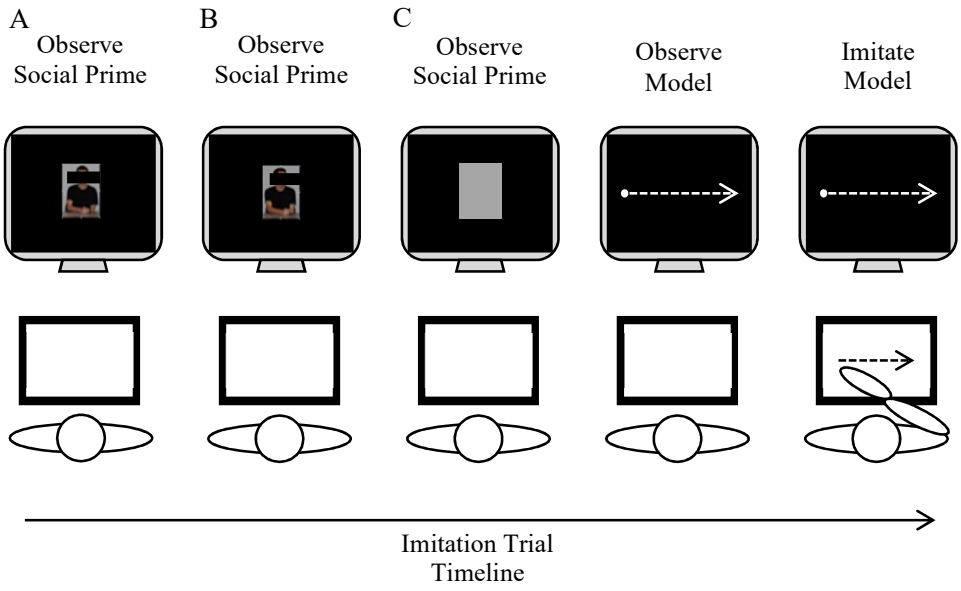
To examine the effect of social primes on imitation learning, an image was displayed in the centre of the monitor prior to the imitation task, which displayed either a pro-social (see Figure 5.1A), anti-social (see Figure 5.1B) or neutral (see Figure 5.1C) prime. The social primes displayed an image of a human head and shoulders in front of a grey background, where the salient information pertained to the type of eye gaze the model was engaging in. In the pro-social prime, the model engaged in 'direct gaze' such that he seemed to be looking straight back at the participant and making eye contact. In the anti-social prime, the model engaged in 'averted gaze' such that the model's head was turned slightly to one side and was looking away from the centre of the image. The neutral prime was designed to control for a general effect of social context and as such, only displayed the grey background used in both social primes and included no human model. The primes used in the current chapter were modified from previous research that had reached publication (Wang et al., 2011a; 2011b; Wang & Hamilton, 2012) and had also been used in pilot testing, where the salient features of the social primes e.g. the gaze direction of the eyes in the image (direct/ averted), were shown

to be obvious and recognisable, without conveying any information regarding what they were attempting to modulate.

Participants first performed 6 familiarisation trials that replicated the task requirements of the imitation protocol. The experiment commenced with participants being instructed to "*observe a model stimulus and then imitate what you see as accurately as possible*". A non-human agent model located in the home-position then moved with a constant velocity to the end-position. The model displayed the exact movement duration and amplitude of the experimental models, but the familiarisation phase contained only constant velocity trials. Further, the familiarisation phase didn't display any of the social primes prior to observing the model stimulus. Using this model ensured construct validity by preventing participants experiencing biological motion or social priming before the imitation trials. Participants were not informed about the duration of the movement, or the type of stimuli.

During the experiment trials, one of the social prime images was displayed for 2000 ms prior to the beginning of the imitation task, after which there was a 1000 ms delay before the onset of the to-be-observed model stimulus. After observing the model, participants imitated by moving the stylus on the tablet so that a cursor displayed on the monitor moved from the home-position to the perceived end-position as per the model. Following movement execution, there was a 4000 ms inter-trial delay before the next social prime and model appeared for action-observation. All participants confirmed they understood that there would be an image to view prior to observing a model, the instruction on how to observe and imitate the trajectory of the model, and the sensorimotor association between the stylus on the graphics tablet and the corresponding movement of the cursor on the monitor. Participants then performed the imitation protocol that consisted of ten blocks of nine trials. A block contained three typical and three atypical biological kinematic models, as well as three

constant velocity models. The three trials of each model stimulus were preceded by of one of each of the social primes – pro-social, anti-social and neutral – such that each social prime was also shown three times in a block of nine trials. Trial order within a block, as well as block order, was randomised across participants. The randomised structure reduced predictability of upcoming social primes and models and promoted imitation on a trial-by-trial basis.



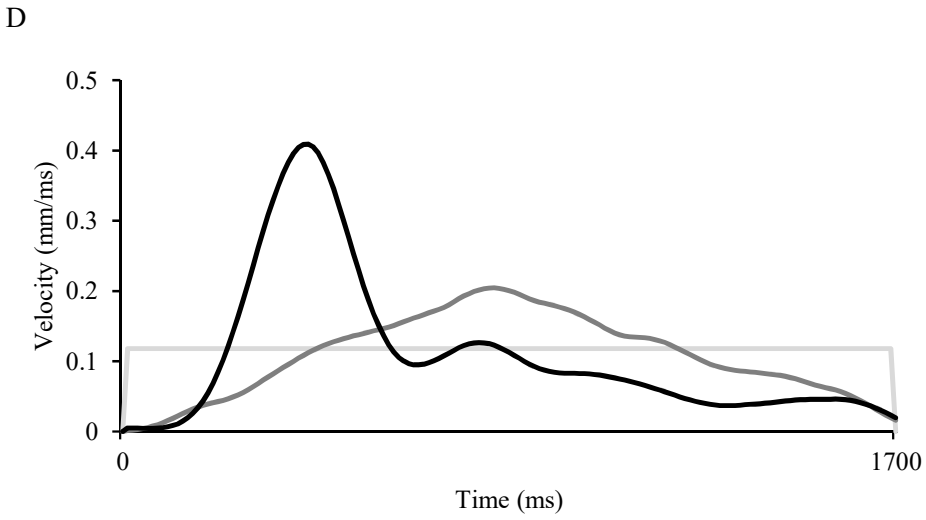


Figure 5.1. A schematic representation of the experimental design. Prior to observation of the stimulus, a social prime was shown depicting pro-social (A), anti-social (B) or neutral prime (C). The black outlined rectangle represents a graphics tablet. The white circle displayed on the CRT monitor represents the model. The single-segment movement is depicted by the arrow (i.e., from the start-position to the end-position). (D) Displacement time-series displaying atypical (black trace), typical (dark grey trace) and constant (light grey) velocity

models. The black boxes shown in the image were added to conceal the identity of the model but were not included in the actual experiment.

5.3.4. Statistical Analysis

To quantify accuracy of biological motion imitation, movement kinematics were extracted from the x-axis movement exhibited by the participants on each trial. Initially, the start was identified as the moment the cursor moved beyond the perimeter of the home-position, while the end was identified when the participant clicked the lower-button on the stylus. The resulting position data was filtered using a low pass 4th order autoregressive filter with an 8 Hz cut-off. The filtered data were then differentiated using a 3-point central difference algorithm to obtain velocity. A MATLAB routine then displayed the velocity profile for each trial, such that an experimenter could manually identify the start, peak, and end of the movement on the velocity profile. Using these points as a guide, the MATLAB routine identified the start of the movement as the moment when velocity was > 0.003 mm/ms, and the end when velocity was < 0.003 mm/ms. Peak velocity and percentage-time-to-peak-velocity from each trial was used to calculate intra-participant means (10 trials per condition) for each independent variable (Model – atypical, typical, constant velocity x Social Prime - direct gaze, averted gaze, neutral). The kinematic variables (percentage-time-to-peak-velocity, peak velocity and movement time) were selected as they most appropriately represent the structural differences contained within the atypical and typical biological motion models and have been used in previously published research, thus acknowledging them as suitable kinematic markers (Hayes et al., 2014; Andrew et al., 2016).

5.4. Results

5.4.1. Imitation

5.4.1.1. Movement Time

A main effect of stimulus [$F(2, 40) = 25.05$, $p < 0.001$] for movement time indicated that imitation was significantly shorter when imitating atypical (M = 2591 ms; SD = 463 ms) compared to the typical (M = 2677 ms; SD = 468 ms) and constant (M = 3050 ms; SD = 445 ms) velocity models (*ps* < 0.05). Movement time was also significantly shorter when imitating the *typical* compared with the constant ($p < 0.05$) velocity model. As seen in Figure 5.2A, there was no main effect of social prime [$F(2, 40) = 0.56$, $p > 0.05$] and there was no significant interaction between the primes and model stimuli [$F(4, 80) = 0.78$, $p > 0.05$].

5.4.1.2. Peak Velocity

As displayed in Figure 5.2B, a main effect of stimulus [$F(2, 40) = 27.76$, $p < 0.001$] for peak velocity showed that magnitude was significantly greater when imitating atypical (M = 0.201 mm/ms; SD = 0.036 mm/ms) compared to typical (M = 0.167 mm/ms; SD = 0.036 mm/ms) and constant (M = 0.14 mm/ms; SD = 0.034 mm/ms) velocity kinematics. A social prime main effect [$F(2, 40) = 4.32$, $p < 0.05$] indicated magnitude of peak velocity was greater after having viewed the anti-social prime (M = 0.172 mm/ms; SD =0.036 mm/ms), compared to the pro-social (M = 0.167 mm/ms; SD = 0.041 mm/ms) and neutral (M = 0.168 mm/ms; SD = 0.029 mm/ms) primes (*ps* < 0.05). There was no interaction between stimulus and social prime [$F(4, 80) = 0.29$, $p > 0.05$,.

5.4.1.3. Percentage-Time-to-Peak-Velocity

As seen in Figure 5.2C, a main effect of stimulus [$F(2, 40) = 22.76$, $p < 0.001$] for percentage-time-to-peak-velocity showed that peak velocity occurred significantly earlier when imitating the atypical (M = 31%; SD = 15%) compared to typical (39%; SD = 14%) and constant (43%; SD = 15%) velocity models (*ps* < 0.05). Peak velocity also occurred significantly earlier when imitating the typical compared to the constant velocity model (*ps* < 0.05). There was no main effect of social prime [$F(2, 40) = 0.79$, $p > 0.05$] and there was no significant interaction between the stimuli and social primes [$F(4, 80) = 1.14$, $p > 0.05$].

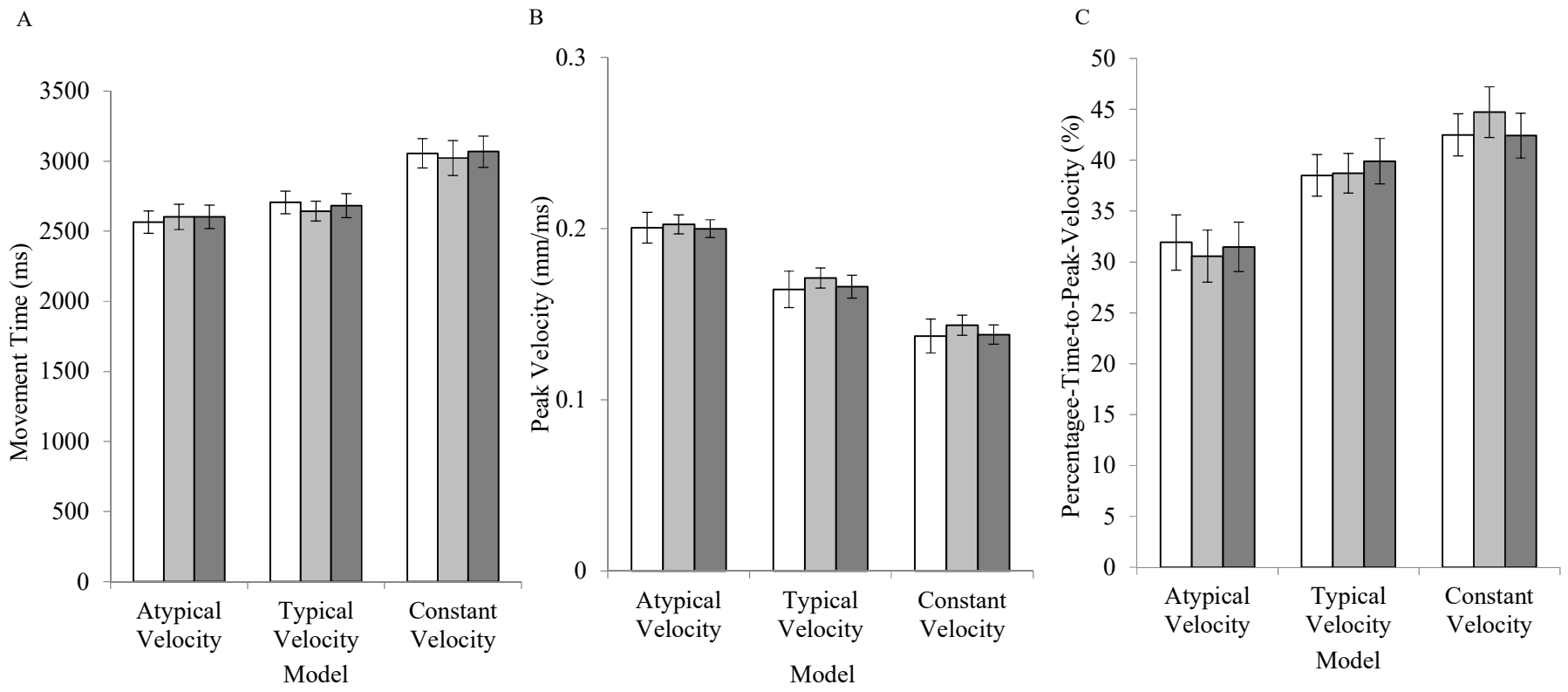


Figure 5.2. Mean imitation data showing movement time (A), peak velocity (B) and percentage-time-to-peak-velocity (C) presented as a function of model and social prime (pro-social = white bar; anti-social = light grey bar; neutral = dark grey bar). Error bars (±) display the standard error mean.

5.4.2. Variability

5.4.2.1. Movement Time

A main effect of social prime [$F(2, 40) = 9.76$, $p < 0.001$] indicated greater variability of movement time when observing the neutral (M = 517 ms) compared with the pro-social (M = 419 ms) and anti-social (M = 441 ms) primes (*ps* < 0.05). As seen in Figure 5.3A, there was no main effect of stimulus [$F(2, 40) = 0.51$, $p > 0.05$] and there was no significant interaction between the primes and model stimuli [$F(4, 80) = 0.93$, $p > 0.05$].

5.4.2.2. Peak Velocity

A main effect of social prime [$F(2, 40) = 5.49$, $p < 0.05$] indicated less variability of peak velocity when observing the neutral (M = 0.029 mm/ms) compared with the pro-social (M = 0.037 mm/ms) and anti-social (M = 0.035 mm/ms) primes (*ps* < 0.05). As seen in Figure 5.3B, there was no main effect of stimulus [$F(2, 40) = 0.67$, $p > 0.05$] and no significant interaction between the primes and the model stimuli [$F(4, 80) = 0.3$, $p > 0.05$].

5.4.2.3. Percentage-Time-to-Peak-Velocity

As seen in Figure 5.3C, there was no main effect of stimulus [$F(2, 40) = 2.27$, $p > 0.05$] and no significant interaction between the primes and the stimuli [$F(4, 80) = 0.32$, $p > 0.05$]. However, a main effect of social prime [$F(2, 40) = 6.75$, $p < 0.05$] indicated greater

variability in percentage-time-to-peak-velocity when observing neutral (17%) compared with the pro-social (12%) and anti-social (15%) primes (*ps* < 0.05).

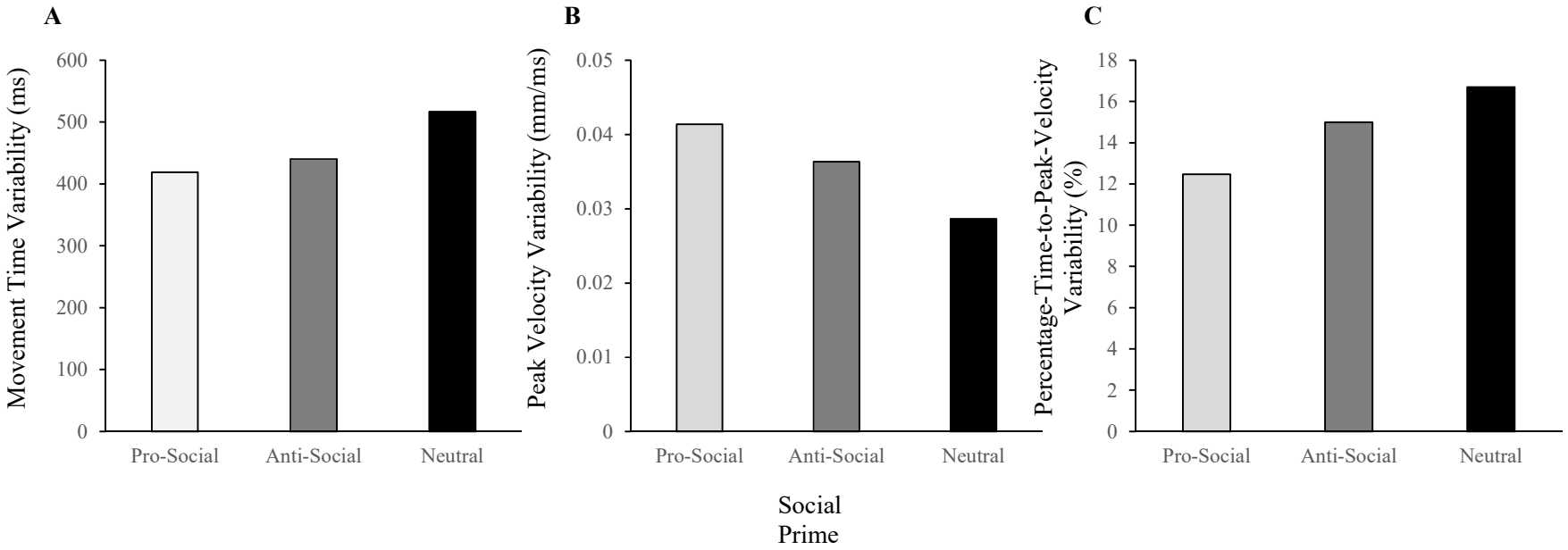


Figure 5.3. Mean variability data showing movement time (A), peak velocity (B) and percentage-time-to-peak-velocity (C) presented as a function social prime (pro-social = white bar; anti-social = light grey bar; neutral = dark grey bar).

5.5. Discussion

The primary aim of the present study was to examine whether and how social primes modulate the coding of biological motion kinematics during imitation learning. By replicating a previously studied imitation learning protocol (Hayes et al., 2014; Hayes et al., 2016), the first expectation was that imitation of the atypical model would be different to that of the typical model. The second expectation was that observing pro-social and anti-social primes prior to the imitation task would both improve (increase accuracy and decrease variability) and reduce (decrease accuracy and increase variability) imitative performance, respectively.

Consistent with previous research (Hayes et al., 2016; 2014; 2010), the results showed that biological motion kinematics were coded during imitation learning. Peak velocity was greater and percentage-time-to-peak-velocity occurred earlier when imitating the atypical, compared with the typical biological motion model (see Figure 5.2). This kinematics are different to those that would be expected given the natural constraints of the task (e.g. typical kinematics) and therefore replicated the imitation findings from previous chapters. Based on the methodologies used in the previous chapters, specifically *Chapter Two*, the coding of atypical biological motion in the present study likely demonstrated lower-level processing of biological motion (Iacoboni, 1999; Catmur & Heyes, 2011). It was also found that the modulatory effect of social primes occurred at two levels: imitation accuracy and imitation variability. In relation to imitation accuracy, peak velocity was more accurate following observation of the anti-social prime; relative to imitation variability, movement time and percentage-time-to-peak-velocity became less variable after observing the pro- and anti-social primes, compared with the neutral prime.

The finding of increased imitation effects following observation of an anti-social prime contrast the social top-down modulation effects typically reported in social imitation studies (Cook & Bird, 2011; Leighton et al., 2010; Wang et al., 2011a). Behavioural research suggests that increased imitation effects often follow pro-social priming, which activates social motives designed to affiliate and assimilate (Lakin & Chartrand, 2003; Wang & Hamilton, 2012; Wang & Hamilton, 2013). As a result, these positive interpersonal behaviours exert top-down control that up-regulates the lower-level visuomotor processing associated with the mirror system to improve imitation (Hogeveen & Obhi, 2012; Wang & Hamilton, 2014). However, there are instances where anti-social up-regulation of imitation have been reported (Wang & Hamilton, 2013; Roberts et al., 2016). These effects corroborate the active-self theory that operates relative to how primes are processed in relation to the 'self'; that is, first-person pro-social and third-person anti-social primes up-regulate and improve imitation, whereas first-person anti-social and third person pro-social down-regulate and reduce imitation effects (Wang & Hamilton, 2013). Therefore, the finding of increased imitation accuracy of peak velocity following anti-social priming in the present study could be interpreted as evidence of the active-self theory, where anti-social third-person priming has up-regulated imitative processes to produce a more accurate representation of peak velocity. Further, this finding demonstrates evidence that the anti-social prime used in the present chapter has acted as a regulator of the underlying processes involved in imitation learning.

The second and perhaps more important social modulation reported in the present chapter concerns decreased movement variability (see Figure 5.3), specifically of movement time and percentage-time-to-peak-velocity, following observation of pro- and anti-social primes. The significance of this findings lies in movement variability not having been previously reported in imitation learning. Variability refers to inherent noise in the motor

system during motor execution and is stochastic within typical movement or imitation thereof (Elliott, Hansen, Grierson, Lyons, Bennett & Hayes, 2010). However, when amplitude remains constant but the force involved in motor execution increases, so in turn does variability (Elliott et al., 2010). As, both biological models displayed greater velocities than the constant velocity model, it could be expected to result in greater movement variability. However, results showed that following the observation of social primes, both models were imitated with less variability, compared with the neutral prime. Therefore, it could be suggested the social primes modulated coding of biological motion such the fidelity of the representations formed during visuomotor processing were more refined, thus resulting in greater control and proficiency during imitation of the models.

As previously discussed, improved imitation following social primes is suggested to be a function of engaging specifically with positive social primes e.g. direct gaze (Wang et al., 2011) or positive word scramble (Leighton et al., 2010). Neurophysiological research has shown that observation or completion of a pro-social prime resonates with and activates regions of the brain associated with mirror neurons (e.g. mPFC) and in essence, 'primes' these regions for the ensuing stimulus (Brass et al., 2001; 2005). With these mirror regions primed, processing of the visual stimulus is upregulated, such that the visual and motor representations are more accurate and thus, imitation improves. Equally, negative social primes have the inverse effect and down-regulate the processing of visual information, such that imitation becomes less accurate. These effects have most commonly been demonstrated in automatic imitation/ mimicry by measuring reaction times (Cook & Bird, 2011; Wang et al., 2011a), and online imitation (Roberts et al., 2015) protocols by measuring imitation accuracy and variability. However, the findings here suggest it is the presence of a social prime per se that modulated imitation, rather than the explicit nature (e.g. pro-social/ anti-social) of the social

prime. These data demonstrate that although the social primes worked in relation to modulating imitation, the interpretation of the primes has resulted in the effects discussed in the current study.

These effects could be associated with the connection between the biological nature of the social primes the observed stimuli. While the atypical and typical models displayed different kinematic structures, they both displayed biological motion kinematics. Similarly, both the pro- and anti-social primes displayed different eye gazes but both were portrayed by a human model. Conversely, the neutral prime did not display any biological features. It has been shown that the neural mechanisms involved in eye gaze priming correspond with regions of the brain associated with detection of biological motion, specifically the STS, IFG and medial prefrontal cortex (mPFC), within the mirror mechanism (Wang et al., 2011b). Moreover, it was suggested that the enhanced connectivity between STS and mPFC following pro-social eye gaze priming implied it was these regions that modulated the sensory input to the mirror mechanism during observation. Therefore, in the context of the present results, it follows that a biological social prime would upregulate the detection and coding of biological motion such that the representations generated were more accurate, as demonstrated by imitation being less variable.

These results appear to add to the current understanding of social modulation during imitation as they are novel in the context of imitation learning. Previous research has largely examined social primes during automatic imitation, a process whereby the imitation is involuntary and relatively independent of intentions (Heyes, 2011). Instead, these results demonstrate that during the intentional acquisition of a novel movement that requires complex processing, social primes still modulate features of biological motion. While imitation learning and automatic imitation engage similar neural circuitry (Iacoboni & Dapretto, 2006),

the data in the current study suggests they may also share the way in which they are influenced by social primes. Moreover, these effects of social modulation corroborate early psychological research examining observational learning, which showed task judgements and work productivity have been shown to improve because of positive social context (Weiss, 1977; Weiss & Shaw, 1979).

In conclusion, the results demonstrated that biological motion kinematics were coded during imitation learning and that social primes involving eye gaze modulated these processes. Based on previous research (Hayes et al., 2016; 2014), biological motion coding was likely a function of lower-level visuomotor processing, where the specific kinematics properties of the biological motion models were represented for motor execution. Moreover, this processing was influenced by social top-down modulation (Wang & Hamilton, 2012), such that the biological nature of the social primes resonated with the biological nature of the model stimuli and resulted in decreased movement variability during motor execution.

Chapter 6: Epilogue

6.1. Aim of the Chapter

The epilogue will present and discuss the key findings from all experimental chapters in the program of work. There will be a critical evaluation in accordance with current literature on imitation and the lower-level processing of biological motion, as well as top-down factors that modulate these processes. Future considerations and translational research will also be discussed with the view to suggesting practical implications.

In short, the present thesis has used a novel protocol that has developed existing methodologies to more accurately examine biological motion coding during imitation learning. Subsequently, the experimental chapters contained within this thesis have valid designs relative to the overall aim of the thesis and therefore extend the current understanding of biological motion coding during imitation learning. To that end, *Chapter Two* is considered the most important chapter of the thesis as it robustly demonstrates that spatial compatibility is not required to imitate biological motion kinematics; rather, the spatially incompatible imitation of atypical biological motion is the first example in imitation learning literature of discrete kinematic markers being imitated through lower-level processes. In knowing that biological motion kinematics are coded through lower-level processes, *Chapters Three* and *Four* extend the current literature by demonstrating how top-down modulations (end-state-targets and visual attention) associated with higher-order cognitive processes operate cooperatively with lower-level processes during the processing of visual information. Finally, *Chapter Five* provides a further insight into top-down modulation associated with social context and imitation, but more importantly is the origin of a broader line of research designed to examine imitation and biological motion coding in people with ASC. The finding of social

modulation discussed in the present thesis extends the current understanding in imitation learning literature, but due to data collection involving neurotypicals only, will also act as a control condition or baseline for future research centred around people with autism.

6.2. Rationale for examining biological motion using an atypical biological motion model

While there is strong evidence of biological motion coding during automatic imitation (Brass et al., 2000; Sturmer et al., 2000; Heyes et al., 2005), online imitation (Kilner et al., 2003; Kilner et al., 2007; Roberts et al., 2015) and the underpinning neuropsychological processes (Iacoboni, 1999; Rizzolatti & Craighero, 2004; Press et al., 2008), it is still unclear whether biological motion is coded during imitation learning. At present, only a small amount of research has investigated biological motion coding during imitation learning, where imitation has been measured in relation to how accurately movement kinematics are reproduced (Hayes et al., 2009; Wild et al., 2010). For example, Wild et al. (2010) demonstrated that observation of "fast" and "slow" hand movements resulted in imitation that was relative to the respective model and thus, the kinematics of the movement were suggested to be coded. Although observation of the "fast" and "slow" models produced differences in the imitation of movement speed, the biological motion kinematics contained within each model were not manipulated directly. For example, while the "fast" and "slow" hand movements used by Wild et al. (2010) displayed different speeds, each of the model kinematics were not manipulated and thus, displayed natural, bell-shaped velocity profiles that were scaled by speed. Therefore, the differences in imitation do not confirm whether the

findings are due to coding of biological motion kinematics or a reproduction of generic differences in the movement speed differences displayed by the models. In this instance, it could be argued that imitation of general movement speed could be a function of strategy (Chong et al., 2009; Eliasmith, Stewart, Choo, Bekolay, DeWolf, Tang et al., 2012; Grezes, Costes & Decety, 1998; Iacoboni et al., 2005; rather than the coding of kinematics (Roberts et al., 2014), where pre-existing motor representations are reconfigured and scaled to produce movements that are similar to the observed visual information (Buccino et al., 2004; Roberts et al., 2014; Vogt et al., 2007). Therefore, in the current thesis a novel protocol was developed to create an imitation task that manipulated the structure of the biological stimuli to directly examine biological motion coding. Within each of the experimental chapters, various combinations of biological and non-biological motion models were examined to establish whether the biological properties of the stimuli are coded and represented during imitation.

Chapters Three, Four and *Five* included a constant velocity model, which was designed to display uniform velocity of 0.1 mm/ms from the onset of movement. These kinematic features make it physically impossible to be imitated through human reproduction because of the constraints on human movement imposed by the neuro-muscular system (Abend et al., 1982; Elliot et al., 2001). As accurate imitation of the constant velocity kinematics was unachievable, it was anticipated that participants would recruit a pre-existing motor response and thus exhibit time to peak velocity that was similar to typical aiming movements (Hayes et al., 2014; Roberts et al., 2014). Therefore, the primary purpose of the constant velocity model in the current thesis was to act as a control condition when comparing imitation of the typical biological and constant velocity models as it allowed a direct comparison between imitation of biological and non-biological motion. Having a control condition for biological motion coding is important as imitation has been shown to be more

accurate following observation of biological, compared to non-biological motion (Brass et al., 2001; Kilner et al., 2003; Press et al., 2005).

Typical biological motion was included in *Chapters Two, Four* and *Five* and displayed human movement that represented a 'natural' trajectory. 'Natural' trajectory refers to the bell-shaped velocity profile that is normally produced during human movement and is dependent on the time, and displacement (e.g., movement amplitude) of a trajectory, peak velocity generally occurs at between 40%-60% of the movement time (Elliott et al., 2001). In the present thesis, a number of models were created, all with a movement time of 1700 ms and displacement of 406 mm criteria. When recording the typical model, start and end targets were visible to ensure trial displacement was consistent and the typical model was performed until the timing goal of 1700 ms was achieved, which meant that the kinematic structure for the typical model reflected a natural profile based on a 406 mm movement and a 1700 ms movement time. As a result, the typical model had a peak velocity of 0.2 mm/ms that, in line with 'natural' kinematics, occurred at 44% of the movement time.

The inclusion of typical biological motion was important to examine any effects of biological tuning; that is, the perception-action system discriminates between biological and non-biological motion, and produces a heightened neural response following detection of biological motion (Tai et al., 2004). In line with this suggestion, the imitation of typical biological motion should be more accurate than copying non-biological constant velocity based on the biological nature of the stimuli. While the perception-action system is suggested to be more sensitive to biological motion (Castiello, Lusher, Mari & Edwards, 2002; Longo & Bertenthal, 2009; Liepelt & Brass, 2010), it has been recognised that the reproduction of typical kinematics could be achieved by rescaling or reproducing existing sensorimotor representations (Hayes et al., 2009; Campione & Gentilucci, 2011) and thus, imitation of the

typical biological motion model does not directly imply biological motion coding. Therefore, all four experimental chapters contained novel, atypical biological motion models to more specifically examine biological motion coding. These atypical models still displayed biological motion, but contained kinematic structures that were not representative of existing sensorimotor representations and deviated from the 'natural' constraints of the task (Roberts, Bennett, Elliott & Hayes, 2012). By not representing existing sensorimotor representations, coding of the underlying movement kinematics contained within the stimuli was required to reproduce the atypical biological motion. *Chapters Two, Four* and *Five* examined the imitation of an atypical biological motion model in comparison primarily with a typical biological motion model to examine whether atypical kinematics were coded during imitation learning. The atypical model in these chapters had a peak velocity of 0.4 mm/ms that occurred at 18% of the movement time. *Chapter Three*, rather than comparing atypical to typical biological motion, examined imitation of two atypical biological motion models that had more similar kinematic structures (peak velocity occurred at 17% and 26% respectively), to further explore the coding of atypical biological motion kinematics. Each atypical model displayed peak velocities that were skewed towards the beginning of the movement and importantly, occurred outside the boundaries of a 'natural' trajectory.

While each of the atypical models contains specific kinematic features that are highly unlikely to be imitated by chance, it could be argued that the discernible kinematic differences between the atypical and typical biological motion models in *Chapters Two, Four* and *Five* mean the issues previously raised with imitation of "fast" and "slow" movements (Wild et al., 2010) may not have been alleviated. It was likely that coding of the model stimuli was localised to the respective kinematics as *Chapter Two* controlled for spatial compatibility, which confirmed that imitation was not a function of reproducing the spatial coordinates of

peak velocity e.g. reproducing peak velocity at the left side of the monitor. However, it was still unclear whether participants were coding the specific kinematic structure underpinning the atypical model (e.g. imitation that was representative of a peak velocity that occurred at 18% of the movement) or just recognising a difference in acceleration, as no post-experimental questionnaire was administered to gauge the response to the imitation task. Therefore, instead of comparing atypical and typical biological motion models, *Chapter Three* compared two atypical models – atypical17 and atypical 26 – that both had a peak velocity skewed towards the beginning of the movement but contained slightly different kinematic profiles. Atypical17 had a peak velocity of 0.37 mm/ms that occurred at 17% of the movement time. In comparison, atypical26 had a peak velocity of 0.24 mm/ms that occurred at 26% of the movement. Here then, imitative differences between these models could not occur without their specific kinematic features embedded within the observed biological motion being coded for motor execution during imitation.

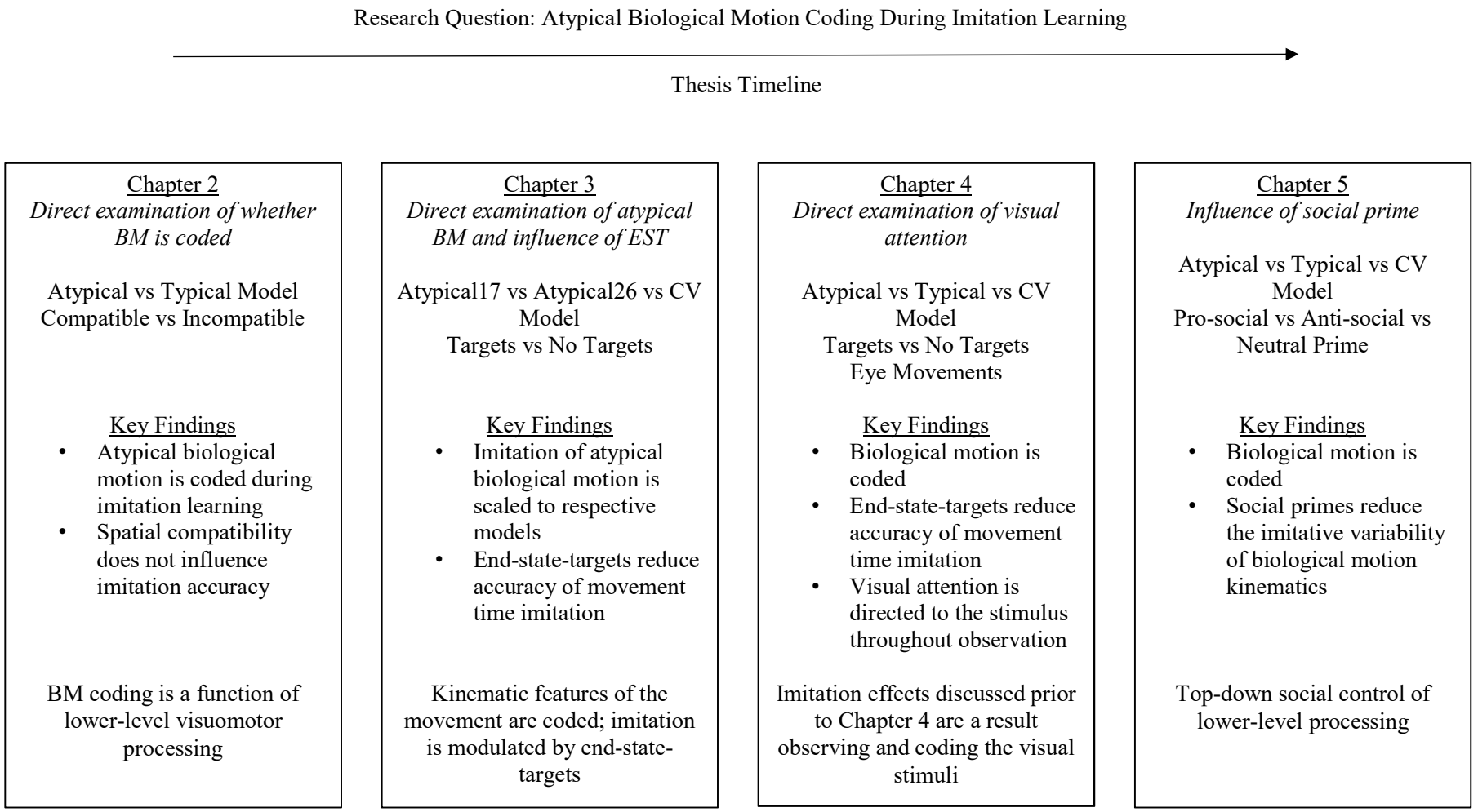


Figure 6.1. A summary of the structure, purpose, flow and key findings from the four experimental chapters (Experiments 1-4).

6.3. Motion Coding

6.3.1. Constant Velocity, Typical, and Atypical Motion

The data reported in the current thesis confirmed the coding of biological motion during imitation learning. Imitation data from *Chapters Two, Four* and *Five* demonstrates imitation of atypical biological motion that was not only different to that of typical biological motion, but was also scaled relative to the respective models. Further, imitation data from *Chapters Four* and *Five* showed imitation of typical biological motion that was different to that of non-biological constant velocity.

In *Chapters Four* and *Five*, where imitation of typical biological motion and non-biological constant velocity were compared, results consistently showed differences in imitation of the two models. For example, results from *Chapter Four* showed when imitating typical biological motion, kinematics was scaled to those of the model, where magnitude of peak velocity was 0.12 mm/ms and occurred at 39% of the movement time. In contrast, imitation of the constant velocity model produced a peak velocity of 0.1 mm/ms, which occurred at 44% of the movement time. While statistically different to the typical model, the percentage-time-to-peak-velocity during reproduction of constant velocity indicates rather than coding the explicit kinematics of the constant velocity model, imitation represented a movement that had been recruited from an existing motor repertoire (e.g. natural kinematics) as peak velocity occurred between the 40-60% window typically associated with a generic bell-shaped velocity profile (Elliott et al., 2001). Further, although the imitation data suggests that the general representation of the typical biological motion model was similar to the underlying kinematics, it is unclear whether imitation reflected coding of the biological

motion kinematics or reproduction of a non-specific movement pattern based on the perceived movement time and displacement during observation of the models. For example, the lacking relative accuracy when imitating the typical biological motion model may have been a function of the number of practice trials; that is, 84 trials may have been sufficient to form a general representation of the typical biological motion model, but insufficient to refine the precise kinematics of the typical biological motion model such that they were representative of the underlying movement features during imitation trials. The differences in imitation of typical biological motion and constant velocity are consistent with previous research that has shown differences in imitation following observation of biological, compared with non-biological motion (Brass et al., 2001; Kilner et al., 2003; Press et al., 2005). In line with these findings, it could be suggested that the differences in imitation between typical and constant velocity models is a result of the biological nature of the typical model inducing biological tuning (Press, 2011) within the perception-action system such that larger visuomotor resonance (Becchio & Castiello, 2012) of the typical biological motion kinematics was produced (Longo et al., 2008), compared to non-biological constant velocity kinematics (Press et al., 2011). Greater visuomotor resonance following observation of human, compared to non-human, movement has been demonstrated during congruent and incongruent online imitation of horizontal and vertical arm movements (Kilner et al., 2003; Roberts et al., 2015). For example, imitation data showed that when making orthogonal arm movements to those observed, interference (or motor contagion; Blakemore & Frith, 2005) was greater when observing a human arm compared with a robotic arm (Kilner et al., 2003). Relative to the current thesis, the difference in imitation of typical biological motion and non-biological constant velocity shown in *Chapters Four* and *Five* extend these findings into imitation learning research, as well as corroborate the suggestion that there is a difference in information

processing based on the biological nature of the observed stimuli (Kilner et al., 2007; Vangeneugden, Pollick & Vogels, 2009).

Biological tuning is supported by neurophysiological research that has shown that observation of biological motion leads to greater neural activation in regions of the brain associated with mirror neurons, compared to observation of non-biological motion (Tai et al., 2004; Costantini, Galati, Ferretti, Caulo, Tartaro, Romani et al.., 2005; Oberman, McCleery, Ramachandran & Pineda, 2007; Engel, Burke, Fiehler, Bien & Rosier, 2008; Miura, Sugiura, Takahashi, Sassa, Miyamoto, Sato et al., 2010). For example, when observing a hand making a grasping action towards a cylindrical object, regions of the left premotor cortex in humans show stronger activations when the grasping action is performed by a human model displaying biological motion, compared to when the action is performed by a robot displaying non-biological motion (Tai et al., 2004). Greater activation of the premotor cortex following observation of biological, compared to non-biological motion hand actions demonstrates biological tuning of the premotor cortex, which extended neuroimaging research that had shown premotor activation during observation of hand and arm movements (Decety, Grezes, Costes, Perani, Jeannerod, Procyk et al., 1997; Grezes, Armony, Rowe & Passingham, 2003; Hamzei, Rijntjes, Dettmers, Glauche, Weiller, & Buchel, 2003), as well as electrophysiological recordings from monkey showing mirror neuron containment within premotor cortex (Di Pellegrino, Fadiga, Fogassi, Gallese & Rizzolatti, 1992; Gallese et al., 1996). Therefore, the biological motion coding demonstrated in the current thesis could be interpreted as the detection of typical biological motion activating the biologically tuned premotor cortex during observation, such that mirror neurons contained within the premotor cortex mapped the visual representation of biological motion formed through observation onto a motor representation that incorporated temporal coding of the movement to be used when

imitating the typical biological motion kinematics (Gangitano et al., 2001). Moreover, the reproduction of existing sensorimotor representations rather than the underlying kinematics following observation of the constant velocity model is consistent with the suggestion that non-biological motion induces less activation of premotor cortex and the mirror neurons contained within it (Tai et al., 2004).

Although a difference between biological and non-biological motion coding had been established, these findings do not examine whether the underlying kinematics of biological motion are coded during imitation learning. Therefore, *Chapters Two, Four* and *Five* included an atypical biological motion model to examine whether observing a model with kinematics different to those of the constraints of the task and not already part of an existing motor repertoire would result in differences in imitation from the typical model. Results across all three chapters consistently showed that imitation of the atypical model produced a greater magnitude of peak velocity that occurred earlier in the movement when compared with imitation of the typical model. For example, *Chapter Two* showed that when imitating the atypical model, peak velocity was 0.28 mm/ms and occurred at 32% of the movement time. Conversely, imitation of the typical model produced peak velocity of 0.19 mm/ms, which occurred at 45% of the movement. This is consistent with previous research that has shown learning and imitation of novel atypical biological motion kinematics following periods of observational practice (Hayes et al., 2014) and although the present thesis has not measured observational practice, it has been suggested that similar processes are recruited during observation practice and imitation learning (Vogt et al., 2007; Heyes, 2011).

More specifically, biological motion kinematics have been examined during imitation learning through observation of hand movements that are either “fast”, “medium” or “slow” (Wild et al., 2010; Stewart, McIntosh & Williams, 2013). Results showed that observation of

faster hand movements elicited faster imitation attempts compared with observation of slower hand movements. These results are corroborated by the imitation data shown in *Chapters Two, Four* and *Five*, which show imitation that is scaled relative to the respective models e.g. observation of the atypical model resulted in a peak velocity that had greater magnitude and occurred earlier in the movement when compared to the typical or constant velocity models. However, the models in the current thesis were manipulated to display explicit kinematic structures (e.g. atypical model had a peak velocity of 0.2 mm/ms that occurred at 18% of the movement), as opposed to non-specific movement speeds (e.g., Wild et al., 2010) and more importantly, those kinematic features were used to examine imitation accuracy. Therefore, by reproducing peak velocity and percentage-time-to-peak-velocity that reflected those of atypical biological motion, it is likely that participants were coding the underlying kinematics contained within the atypical model during observation that formed the representation used for imitation.

To confirm what features of the atypical model were being represented during imitation learning, further examination of the atypical biological motion was required. Therefore, *Chapter Three* included a second atypical biological motion model, which allowed for the direct comparison of two novel, atypical kinematic models – atypical17 and atypical26. By using two different atypical models, neither can be imitated simply by reproducing an existing motor repertoire, as with the typical model, and thus differences in imitation of atypical17 and atypical26 would confirm the coding of the underlying atypical biological motion kinematics. If imitation of atypical biological motion, relative to the typical biological motion, is based on detecting a faster acceleration or different movement speed, it would be expected that imitation of atypical17 and atypical26 produced similar kinematics during imitation. as the differences in acceleration between the models was lower than the 20-25%

required to perceptually discriminate the changes in velocity (Babler & Dannemiller, 1993; Brouwer, Brenner & Smeets, 2002). Results showed that imitation of the atypical17 model produced a peak velocity of 0.24 mm/ms that occurred at 22% of the movement time, whereas imitation of the atypical26 model produced a peak velocity of 0.19 mm/ms that occurred at 29% of the movement time. Here then, rather than showing differences in imitation of two structurally dissimilar models (atypical and typical), the imitation data from *Chapter Three* shows that imitation of two comparable kinematic structures embedded within separate atypical biological motion models are scaled to the respective models. Scaled imitation of both atypical models could not have occurred without the kinematic features of both atypical17 and atypical26 being coded during observation, such that the motor representations formed for imitation featured the peak velocity and percentage-time-to-peak-velocity information of each model respectively. Taken together, these data suggest that biological motion is coded during imitation learning and importantly, biological motion coding is a function of the kinematic features of the observed stimuli being represented for motor execution.

6.3.2. How biological motion is coded?

As discussed above, the primary findings from *Chapters Three, Four* and *Five* suggest that biological motion is coded during imitation learning, but they do not explain how this coding occurs. The process of imitation is a product of reproducing the underlying kinematics of an action through lower-level processes (Iacoboni et al., 2001; Rizzolatti et al., 2001; Buccino et al., 2004), but can often be attributed to the reproduction of the spatial properties of the observed movement, termed spatial compatibility (Brass et al., 2000; Sturmer et al.,

2000; Catmur & Heyes, 2011). When imitation engages implicit lower-level processes, direct associations are formed between the observed action and the to-be-executed action (Iacoboni, 1999; Roberts et al., 2012), such that the visual information provides a visual description that is used for subsequent action coding (Heyes & Ray, 2000). Conversely, when imitation is a product of spatial compatibility, top-down modulatory processes related to spatial and anatomical compatibility are engaged that drive the process of imitation (Heyes, 2011; Roberts et al., 2012). In the context of the current thesis, if imitation was a function of spatial compatibility it would be spatial positioning of the cursor relative to the environment (e.g. the point at which peak velocity occurred on the monitor), rather than the way in which the cursor is moving (e.g. the kinematics of the models). The ability to differentiate them is therefore important to correctly understand the nature of imitation.

Therefore, *Chapter Two* controlled for imitation effects being interpreted as the reproduction of the spatial coordinates at which peak velocity occurred by requiring imitation of both spatially compatible and incompatible model stimuli. This was experimentally controlled by spatially decoupling observation and imitation trials such that spatially incompatible imitation trials required a visual transformation. Reproduction of kinematics that were closer to that of the model stimuli during spatially compatible, compared to incompatible trials, would suggest that imitation was a function of reproducing the spatial properties of the observed movement; however, similar reproduction of kinematics during both spatially compatible and incompatible trials would suggest the kinematics were coded through lower-level visuomotor processes. In addition to showing that observation of the atypical model produced greater magnitude of peak velocity that occurred earlier in the movement compared to imitation of the typical model, the imitation data from *Chapter Two* showed these imitation effects were not modulated by the spatial compatibility between the observed and executed

movements. That is, when the stimuli were observed moving in a different direction (right to left) or orthogonal plane (top to bottom, bottom to top) to the execution requirements (left to right), imitation of both the atypical and typical models were scaled to the respective models. For example, after observing the atypical model move from left to right, spatially compatible imitation produced average peak velocity of 0.29 mm/ms that occurred at 32% of the movement; after observing the atypical model move from top to bottom, spatially incompatible imitation produced peak velocity of 0.29 mm/ms that occurred at 31% of the movement.

By controlling for the spatial compatibility between the observed and executed movements, spatially incompatible imitation is unlikely to be a function of reproducing the spatial coordinates of the visual representation (e.g. movement from right to left where the peak velocity occurs towards the right side of the movement) based on the visuomotor situation (Hommel & Lippa, 1995) and instead, isolates the coding of visual stimuli to lower-level visuomotor processes (Press et al., 2008). Therefore, similar imitation of spatially compatible and incompatible biological motion models shown in *Chapter Two* demonstrates that the coding of atypical biological motion is likely to be a function of lower-level visuomotor processing. The lower-level coding of kinematics suggests the perception-action system maps specific characteristics (e.g. movement kinematics) of the observed stimulus onto a sensorimotor representation that is directly activated during imitation trials (Buxbaum & Kalenine, 2010; Cisek & Kalaska, 2010; Heyes, 2011), which may indicate the visual description produced during observation incorporates temporal coding into the representation (Gangitano et al., 2001). Temporal coding has been demonstrated by measuring MEPs in the finger that were induced by TMS during observation of a reaching-grasping action, where the amplitude of MEPs was modulated by the amount of observed finger aperture; that is, response

facilitation was tuned relative to specific kinematic landmarks contained within the observed movement.

Moreover, in demonstrating similar imitation during spatially compatible and incompatible trials, the findings in *Chapter Two* corroborate previous automatic imitation research that suggests imitation is not a function of spatial compatibility (Brass et al., 2001; Bertenthal et al., 2006; Catmur & Heyes, 2011; Heyes et al., 2005). For example, reaction times were similar when imitating a finger raising or tapping movement that was observed either in the same orientation as required for imitation (spatially compatible) or when the hand was flipped and presented upside-down (spatially incompatible). Flipping the observed finger tapping or raising such that they were observed upside-down decoupled the direction of the movement required for imitation (e.g. finger tapping when flipped had the same spatial compatibility as finger lifting). Likewise, reaction times have been shown to be similar when observing hand grasping and opening movements where the hand was observed in the same (spatially compatible) or orthogonal (spatially incompatible) orientations, where the stimulus hand was observed vertically and imitation hand was horizontal, to that which was required for imitation (Heyes et al., 2005). Observing the hand opening or closing in an orthogonal plane decoupled both the direction of movement and the plane in which the stimulus was observed (e.g. horizontal finger tapping produces a downward movement; orthogonal finger tapping produces a right or left movement), such that the observed and executed movements did not have the same spatial alignment or orientation.

Controlling for spatial compatibility by requiring imitation of observed stimuli that were both spatially compatible (left to right), as well as incompatible in both the direction (right to left) and plane (top to bottom and bottom to top) of the movement, therefore suggests that imitation of the biological motion models is based on the extraction of the kinematic

properties that define each model – for example, magnitude and timing of peak velocity. The extraction of these kinematics could be facilitated by the tracking of the model during observation, as visual attention has been demonstrated to be a crucial feature of biological motion coding through use of point-light displays (Johansson, 1973; Bidet-Ildei, Orliaguet, Sokolov & Pavlova, 2006) and selective attention (Hayes et al., 2014; Longo & Bertenthal, 2009). Therefore, *Chapter Four* controlled for visual attention by recording eye movements during imitation. As seen in Figure 4.8, the eye movement velocity data is similar to the kinematic structures of each of the model stimuli respectively. For example, observing typical biological motion results in eye movement behaviour that produced the expected bell-shaped velocity profile of the typical model, whereas observing the atypical model generated a greater magnitude of peak eye velocity that also occurred towards the beginning of the movement. As the eye velocity data reflects the kinematic structures of the respective models, these data suggest that participants were attempting to track the cursor during the observation phase of the experimental task and moreover, that the visual description supplied to the perception-action system contained all the kinematic data underpinning the models. Therefore, participants had the opportunity to process all underlying kinematic data contained within the model stimuli and thus, project accurate representations of each model onto the motor system that resulted in scaled imitation of the respective models (Costantini, Ambrosini, Cardellicchio & Sinigaglia, 2013; D'Ausilio, Gredeback, Falck-Ytter & Fadgia, 2013).

Having controlled for spatial compatibility (*Chapter Two*) and confirmed eye movements are directed to the stimuli during observation (*Chapter Four*), the imitation of biological motion reported in the current thesis corroborates neurophysiological research on mirror neurons and the perception-action system (Buccino et al., 2004; Iacoboni et al., 1999). Within the perception-action system, it is commonly held that the 'core circuit' for imitation

includes the posterior IFG, vPMC and IPL (Iacoboni & Dapretto, 2006), while frontal regions also show consistent activation during observation and imitation in relation to higher-order control processes (Molenberghs, Cunnington & Mattingley, 2009). It is suggested that observation of biological motion kinematics activated the pSTS, which produced a visual description of the observed stimulus (Allison et al., 2000) that was supplied to the temporal regions of the perception-action system where a visual representation of the observed stimulus was formed for subsequent action coding (Cattaneo et al., 2010; Rizzolatti & Fadiga, 2014). If goals were present during the observation trial (e.g. end-state-targets in *Chapters Three* and *Four*) the visual representation would most likely be processed by IPL, which is associated with processing the top-down components of visual information (Southgate & Hamilton, 2008) such that the goal of the action (e.g. imitating a stimulus that finishes on a red target) would have been incorporated into the visual description to become part of the motor representation used for imitation (Fadiga et al., 1995; Casartelli & Moteni, 2014). If there were no goals present during action-observation (e.g. *Chapter Two*), atypical biological kinematics would have most likely been processed in IFG, which is associated with the lower-level coding of visual information (Rizzolatti & Sinigaglia, 2010) and biological motion (Saygin et al., 2004). IFG facilitates the understanding of the motoric components of an action such that the underlying kinematics of the stimuli would have been represented as a motor representation. This direct connection between the visual description produced by STS and motor representation for imitation is termed visuomotor processing (Bastiaansen, Thioux & Keysers, 2009; Brass, Ruby & Spengler, 2009), which is known to contain neural substrates that facilitate imitation (Iacoboni, 1999; Rizzolatti et al., 2001) and is likely to explain the coding of unmodulated atypical biological motion reported in *Chapter Two*.

In line with the visuomotor processing substrates contained within the perception-action system, atypical biological motion coding could be interpreted as the visual representation of atypical biological motion directly linking with the subsequent reproduction of the same representation, such that subsequent reproduction of the same atypical kinematics formed a link between the observed and executed action that resulted in imitation of atypical biological motion regardless of spatial compatibility (Catmur et al., 2009; Cavallo et al., 2014). The mechanisms underpinning visuomotor processing of biological motion are also at the basis for the associative sequence learning (ASL) theory of imitation (Heyes & Ray, 2000; Heyes, 2001; Brass & Heyes, 2005). ASL suggests that excitatory links or associations connect sensory and motor representations of the same action (Heyes & Ray, 2000), which are formed during learning and development (Schultz & Dickinson, 2000, Catmur & Heyes, 2011). This learning occurs when the observed movement and executed movement are correlated, which engages regions of the brain with mirror properties that enable action understanding (di Pellegrino et al., 1992; Ferrari et al., 2003). This effect has been demonstrated using countermirror protocols, where periods of training have been shown to reconfigure the mirror system (Catmur et al., 2007) and improve automatic imitation effects of non-biological stimuli (Press et al., 2007). For example, in the Catmur et al. (2007) study, participants underwent incompatible training periods where they executed index-finger adductions following observation of little-finger adductions and vice versa, as well as compatible training periods where the observed and executed finger movements were the same. Using TMS to measure MEPs in the musculature of the respective fingers, results showed that incompatible training reversed the mirror effect such that observation of the index-finger adduction primed the musculature in the little finger and vice versa and demonstrates that sensorimotor experience plays a critical role in imitation. The ASL model suggests the proposed visuomotor processing in the current thesis is facilitated by matching

vertical associations connecting sensory and motor representations of the same action during development and learning, which occur when the sensorimotor experience of the observation and execution of an action, in this case observing and imitating atypical biological motion, are correlated (Catmur et al., 2009). As such, sensory representations of atypical biological motion activated by movement observation are more likely to be active during motor representations of atypical biological motion than during motor representations of any other movement (e.g. typical biological motion/ constant velocity).

6.3.3. Modulatory Factors

Although *Chapter Two* demonstrates lower-level processing of atypical biological motion by controlling spatial compatibility and manipulating biological motion, it did so in an unmodulated context; that is, during observation and imitation the only visual information available was that of the cursor containing kinematic information of each of the model stimuli and there were limited environmental factors that could have created top-down modulatory effects e.g. end-state-targets (see "true imitation"; Tomasello, Savage-Rumbaugh & Kruger, 1993). While that is important in understanding whether biological motion is coded and how that process might take place, imitation also recruits general top-down processes that can result in modulation (Bekkering et al., 2000; Chong et al., 2008; Wang & Hamilton, 2012). Therefore, *Chapters Three* and *Five* examined the contribution of end-state-targets (*Chapter Three*) and social primes (*Chapter Five*) to imitation of biological motion during imitation learning.

6.3.3.1. End-state-targets

Chapter Three examined the influence of end-state-targets on imitation of biological motion. End-state-targets have been shown to modulate imitation in both a behavioural (Wohlschlager et al., 2003; Hayes et al., 2008) and neurophysiological (Hamiton, 2008) research. For example, when children observe ipsilateral (cross-hemisphere) arm movements that touch an ear (e.g. right hand touches left ear), they often imitate by making a contralateral (same hemisphere) arm movements (e.g. left hand touches left ear) as they are the most efficient means of achieving the goal (touch the ear). However, when the ipsilateral arm movement is made to a space beside the ear (e.g. right hand moves to space by left ear), children imitate the way in which the arm moves as there is no clear observable end goal (Bekkering et al., 2000). Goal-directed modulation occurs when the observed movement is encoded through hierarchical processing, where goals are prescribed primary importance and achieved at the expense of the underlying movement kinematics. Therefore, having established biological motion is coded during imitation learning in *Chapter Two* (e.g. the means), the purpose of *Chapter Three* was to manipulate end-state-targets to examine whether they modulated the coding of biological motion during imitation learning.

Imitation data from *Chapter Three* showed that in addition to atypical biological motion coding, the presence of end-state-targets modulated imitation such that, relative to the model movement time of 1700 ms, movement time was less accurate when end-state-targets were present (2294 ms) compared to when they were absent (2156 ms). An interpretation of these findings could be that, in line with GOADI, the presence of end-state-targets results in greater importance being placed on goal attainment such that less attention is placed on achieving the movement time goal of the task (Ondobaka & Bekkering, 2012). However, contrary to recent imitation learning research (Wild et al., 2010), the results from *Chapter Three* did not demonstrate the attenuation of kinematics when end-state-targets were present.

Wild et al. (2010) showed that imitation was modulated by the speed of the observed action only when end-state-targets were absent, which resulted in a difference between imitation of the "fast" and "slow" models that was not reported when end-state-targets were present and corroborates both GOADI (Bekkering et al, 2000) and the dual-route model (Rumiati & Tessari, 2002). It was suggested that when end-state-targets were present, they were perceived as the most important feature of the movement (GOADI), which resulted in less visual attention being given to the movement trajectory of the model's limb during observation such that small movement details containing kinematic information were not processed. In the study by Wild et al. (2010) visual attention was quantified by recording eye movements during observation of the stimuli and showed that participants made more saccades towards the end of the movement when end-state-targets were present.

Conversely, when end-state-targets were absent, each trial was without an obvious end-point and as confirmed by the eye movement data, visual attention was orientated to the stimulus such that the movement trajectory was tracked during observation. In tracking the movement trajectory throughout the duration of the observation trial, all the kinematic information contained within the movement trajectory was observed and could be coded. Here then, the results from *Chapter Three* show that imitation of biological motion kinematics occurs regardless of the presence of end-state-targets but movement time is modulated when end-state-targets are present, which contradicts the suggestion than top-down modulatory factors associated with goals are perceived as most important within a hierarchy and instead, requires a different explanation for the findings.

An interpretation for the goal-directed modulation of movement time, but not kinematics, could be related to a mechanism that integrates top-down attentional processes and lower-level visuomotor processes based on the goal of the task (Roberts et al., 2012;

Andrew et al., 2016). A complimentary relationship between lower-level and higher order processes has been shown using motor training that required a 3-segment movement sequence to be acquired under varying levels of feedback (no knowledge of results [KR], high-frequency KR and two reduced-frequency KR) prior to an imitation phase (Andrew et al., 2016). The imitation phase required imitation of models similar to those used in the current thesis (atypical biological motion, typical biological motion and constant velocity) across a single-segment movement that had the same movement time (1700 ms) as the movement learned during motor training but displayed different movement kinematics, thus examining the higher-order processes associated with representing movement time and the lower-level processes associated with kinematic coding. Results showed imitation of biological motion kinematics was similar across all groups, but movement time was more accurate following reduced-frequency knowledge of results (KR), compared to high-frequency KR or no KR groups and suggests that although top-down cognitive processes and lower-level sensorimotor processes are distinct, they must operate together to facilitate both the imitation of biological motion kinematics and modulation of movement time accuracy. Therefore, an appropriate inference of the imitation data from *Chapter Three*, which shows coding of biological motion kinematics and goal-directed modulation of movement time, could be that complimentary lower-level and top-down systems are active during imitation learning that facilitate the coding of biological motion whilst also representing the temporal components of the observed action relative to end-state-targets.

6.3.3.2. Social Primes

Chapter Five examined the influence of social primes on imitation of biological motion. Images displaying either neutral, direct or averted eye gaze primes were shown prior to observation of the model stimuli that were designed to convey neutral, pro- or anti-social context respectively (Wang et al., 2011a). While the effects of social modulation have been examined in the context of automatic imitation (Leighton et al., 2010; Wang et al., 2011a; Wang & Hamilton, 2012), these effects have not yet been examined in the context of imitation learning of biological motion kinematics. From a social psychology perspective, social interaction has been identified as an evolutionary function of humans that defines how imitation occurs, as demonstrated by the chameleon effect (Chartrand & Bargh, 1999), which refers to the nonconscious mimicry of various interactive behaviours (e.g. postures, facial expressions and mannerisms) such that behaviour changes to match that of those within the social environment. Social psychology has also shown social modulation of performance based on subjective features such as attractiveness (van Leeuwen et al., 2009; Kuleza, Szypowska, Jarman & Dolinski, 2014) and social status (Landers & Landers, 1973; McCullagh, 1986). For example, McCullagh (1986) showed that when young girls were cued with women of high (cheerleader) and low (woman in street clothes) social status prior to completing a ladder task, performance of the task was better following observation of the cheerleader.

Imitative behaviour changes can facilitate learning (Bandura, 1977) and/or promote feelings of liking and affiliation amongst those engaged in the imitation (Chartrand & van Baaren, 2009). It is suggested that these feelings of affiliation occur as a result of the perceived positive social consequences (e.g. pro-social priming), which relative to imitation, exert top-down control that modulates visual information processing such that imitation is controlled to

meet the social goal (Hamilton, 2008). Conversely, perceived negative social consequences (e.g. anti-social priming) exert control that down-regulates visual information processing such that imitation becomes worse (Tiedens & Fragale, 2003). These up- and down-regulatory effects have been demonstrated in automatic imitation of hand opening/ closing movements after completing neutral, pro- or anti-social word scrambles (Leighton et al., 2010). Results showed that pro-social priming produced a larger automatic imitation effect (e.g. the difference between compatible and incompatible reaction times was greater) and anti-social priming produced a smaller automatic imitation effect (e.g. the difference between compatible and incompatible reaction times was lesser) when compared to the neutral prime, which suggests a bidirectional relationship between social primes and imitation. Therefore, the purpose of *Chapter Five* was to investigate how pro- and anti-social eye gaze primes modulated the coding of biological motion kinematics in the context of imitation learning.

In addition to confirming biological motion coding, imitation data from *Chapter Five* showed that social primes modulated imitation. In relation to imitation accuracy, following observation of both the atypical and typical biological motion models, imitation of peak velocity was more like the respective model when primed with the anti-social prime, compared to the pro-social and neutral primes. Improved imitation effects following anti-social priming could be interpreted as evidence of the active-self theory (Wheeler, Demarree & Petty, 2007), which suggests the direction of the prime-to-behaviour effect is relative to how the primes are processed in relation to one's self. For example, while priming with the word "smart" is likely to induce an assimilative self-concept (e.g. I am smart) and therefore behaviour (e.g. better performance in an intelligence task), priming with distinct examples of intelligence (e.g. Einstein) induce contrasting self-concepts (e.g. I am not Einstein, therefore I am not smart) and therefore behaviours (e.g. bad performance on an intelligence test

(Dijksterhuis, Spears, Postmes, Stapel, Koomen, van Knippenberg et al., 1998). As the social primes in the *Chapter Five* were observed from a third-person rather than a first-person perspective, they may have prompted a contrasting self-concept, which meant that rather than pro-social primes exerting top-down control that up-regulated information processing (Leighton et al., 2010), the third-person anti-social primes may have induced opposing modulatory effects to those characteristically associated with the pro- and anti-social primes, which resulted in the upregulation of peak velocity during imitation (Roberts et al., 2016). Upregulated imitation following anti-social priming from a third-person perspective has been demonstrated using word scrambles prior to completion of an imitation task (Wang & Hamilton, 2013), where up-regulation and improved imitation was reported when completing pro-social word scrambles in the first person (e.g. "unnatrual am trying to help), as well as anti-social word scrambles in the third person (e.g. "the white sphere is trying to hinder). Conversely, down-regulation and weaker imitation was reported when completing anti-social word scrambles in the first-person (e.g. "I am trying to hinder) and pro-social word scrambles in the third person (e.g. "the white triangle is trying to help).

As well as imitation accuracy being modulated by social priming, variability data indicated imitation performance was significantly more consistent, and less variable, following observation of both pro- and anti-social primes, compared to no prime. For example, percentage-time-to-peak-velocity and movement time were less variable following presentation of the pro- and anti-social primes, compared with presentation of the neutral prime. Percentage-time-to-peak-velocity had a variability of 17% following neutral prime, compared with 15% and 12% following the anti-social and pro-social primes respectively. Similarly, movement time had a variability of 517 ms following the neutral prime, compared with 441 ms and 419 ms following the anti-social and pro-social primes respectively. These

findings corroborate previous research that has shown reduced variability of online imitation following observation of social primes (Roberts et al., 2016). Prior to observing horizontal (control condition) or curvilinear arm movements while executing cyclical horizontal arm movements, participants completed pro-or anti-social word scrambles to influence the social context of the imitation task. Results showed that in addition to greater contagion during observation of curvilinear, compared to horizontal arm movements, completing the anti-social primes increased contagion further during observation of the curvilinear arm movements, relative to the pro-social primes and thus, demonstrates reproduction of a movement more like that which was observed. As only the anti-social prime up-regulated and decreased movement variability, the findings were attributed to the self-active theory as discussed previously (Dijksterhuis et al., 1998; Wang & Hamilton, 2013). However, the variability findings in the current thesis demonstrate that it is the observation of a social prime in general, rather than anti-social specifically, that modulates and reduces movement variability during imitation and therefore, a different interpretation of these results should be considered.

Demonstrating that both pro- and anti-social primes improve imitation by reducing variability suggests rather than the explicit nature of the prime (e.g. anti-social prime up-regulates imitation of peak velocity) or observing no social prime at all (e.g. neutral prime), there is a general priming effect that is related to the observation of any social prime. A general priming effect of imitative variability suggests that social primes are influencing imitation at an intrinsic level (Meltzoff, 1996), such that movement execution is facilitated e.g. imitation is less variable. The general priming effect on variability in *Chapter Five* challenges the classic understanding of social control of imitation, termed STORM (social top-down response modulation; see Wang & Hamilton, 2012), which suggests that during 'successful' pro-social priming, the observer subconsciously forms a positive social affiliation that

enhances and up-regulates the processing of the observed action such that subsequent imitation is improved (van Overwalle & Baetens, 2009). Conversely, if anti-social priming is 'successful', the observer forms negative affiliations that diminish and down-regulate the processing of the observed action such that imitation is impaired (Leighton et al., 2010).

A general effect of social priming could be explained by the neural commonalities that imitation and social modulation share. The active regions during observation of both biological motion and social primes have been explored through the functional connectivity and arrangement of the amPFC, associated with social information processing, and the IFG/vPMC of the perception action system, associated with imitation (Wang & Hamilton, 2011; Spunt and Lieberman, 2012). To ensure participants engaged in observation of the social primes, prior to the experiment they were instructed that observing the images was required during each trial and they were to direct their attention to the images when they were presented on the monitor. Relative to *Chapter Five*, the primes containing eye gazes displayed either direct (pro-social) or averted (anti-social) gaze, which were modified from previous research that has shown both images to activate regions of the brain associated with the STS and the detection of biological motion, as well as the IFG and mPFC that have mirror properties (Wang et al, 2011b). Though the data from *Chapter Five* does not examine neurological activity during the imitation task, it is conceivable that the neurological links between eye gaze priming and imitation of biological motion influenced the way in which the visual stimuli were processed and represented. Based on the neurological associations biological motion and social primes share, it could be suggested that the commonalities between the eye gaze primes and observed stimuli could have created a biological sensitivity (see biological tuning; Blakemore & Frith, 2005), where a biological social prime (pro- and

anti-social) would upregulate the detection and processing of biological motion (atypical model) representations such that imitation was less variable.

6.4. Models of Imitation

The findings discussed throughout the current thesis provide an insight into biological motion coding during imitation learning and corroborate the concept of imitation being a complex process which varies greatly based on the visual information available (Oztop, Kawato & Arbib, 2006; Heyes, 2011, Campbell & Cunnington, 2017). Primarily, all experimental chapters show biological motion is coded during imitation learning, either by demonstrating a difference in imitation between typical and constant velocity models (*Chapters Four* and *Five*), atypical and typical models (*Chapters Two, Four* and *Five*) or atypical17 and atypical26 models (*Chapter Three*). Moreover, by spatially decoupling the observed and imitated trials in *Chapter Two*, scaled imitation of spatially incompatible atypical biological motion isolates the coding of biological motion to lower-level visuomotor processing. As imitation was similar during spatially compatible and incompatible trials, results confirm that biological motion coding is not a function of top-down control based on reproducing the spatial coordinates of kinematic features e.g. peak velocity occurring at the left side of the monitor, but the lower-level processing of the underlying movement kinematics.

In addition to these primary findings, which provide insight into the broader understanding of biological motion coding during imitation learning, *Chapters Three* and *Five* were designed to examine the modulatory influences on biological motion coding. For example, the imitation task in *Chapter Three* included end-state-targets to examine the coding

of biological motion during goal-directed imitation (Bekkering et al., 2000). Imitation data showed that in addition to biological motion coding, the presence of end-state-targets impaired the accuracy of movement time imitation relative that of the models (1700 ms). These results demonstrate that the lower-level processes involved in biological motion coding operate cooperatively with top-down attentional processes associated with end-state-targets to facilitate imitation that incorporates different features (e.g. kinematics, goals) of the observed stimuli (Roberts et al., 2012; Andrew et al., 2016). In addition to end-state-targets modulating imitation, *Chapter Five* shows that social primes modulate the coding of biological motion such that imitation of peak velocity is closer to that of the models following observation of an anti-social prime, relative to a pro-social or neutral prime. Anti-social up-regulation of imitation could be interpreted as evidence of the active-self theory (Wheeler et al., 2007), where the perception of the prime relative to one's self influences how the prime modulates imitation e.g. observing an anti-social prime in the third-person generates an up-regulation of information processing (Wang & Hamilton, 2013).

In addition to the anti-social up-regulation of peak velocity, imitation of movement time and percentage-time-to-peak-velocity was less variable following observation of both pro- and anti-social primes, compared with neutral primes. The reduction of variability following observation of both a pro- and anti-social prime suggests that social modulation is a product of a general priming effect, rather than the explicit nature of the social prime (e.g. pro-social or anti-social), which modulates the efficacy of the representation formed during observation at an implicit level (Meltzoff, 1996).

While there are many individual theories that posit explanations for how imitation occurs (see "theoretical models of imitation", *Chapter One*), the current thesis demonstrates there are many levels of information processing that underpin imitation, which may be better

explained by a combination of theories. Fundamentally, the current thesis shows that biological motion is processed during imitation learning (all chapters) and that this processing can be modulated by the presence of end-state-targets (*Chapter Three*) and social primes (*Chapter Five*). While previous research has suggested that action-goals are prioritised through hierarchical coding (Bekkering et al., 2000; Wohlschlager et al., 2003), *Chapter Three* shows the end-state-target modulation of movement time as well as the coding of movement kinematics, which suggests there may be an embedded system where lower-level coding of biological motion and top-down modulatory control of higher-order cognitive processes operate cooperatively to facilitate successful imitation based on the environmental context and visual information available (Andrew et al., 2016).

The complimentary relationship between lower-level and higher-order systems is consistent with the suggestion that multiple routes within the brain underpin imitation (Bekkering et al., 2000; Di Dio et al., 2013; Hamilton, 2008; 2015; Rumiati et al., 2009; 2014). Relative to *Chapter Three*, the kinematic features of the atypical biological motion model are suggested to be coded by visual areas (temporal gyrus; STS) and IFG (Gallese et al., 2002; Kilner et al., 2009; Molenberghs, Cunnington & Mattingley, 2012; Rizzolatti & Sinigaglia, 2010), whereas the goal of the action (e.g. completing imitation of the atypical biological motion model by reaching an end-state-target) is processed by a parietal route (Chong et al., 2009; Hamilton, 2008). These areas are known to contain mirror neurons and as such, parietal, premotor and frontal regions of the brain are considered to form the 'core circuitry' within the perception-action system and operate together to facilitate the imitation of novel actions such as atypical biological motion (Hamilton, 2015; Iacoboni & Dapretto, 2006). It is suggested that the 'core circuitry' of the perception-action system may function as a network for visual to motor transformations (Hamilton, 2015) that is facilitated by the process of imitation

learning (Heyes, 2011) and may have formed links between the observed atypical biological motion kinematics and an appropriate motor output (Cisek & Kalaska, 2010). This 'core circuitry' is also engaged by non-imitative tasks, as demonstrated through sensorimotor training that required participants to perform a hand movement while observing a foot movement, and perform foot movements while observing a hand movement (Catmur et al., 2008). Results showed that the perception-action system could be reconfigured such that observing a hand movement activated regions of the brain associated with foot movements and vice versa. While the perception-action system can be reconfigured by sensorimotor training, the current thesis suggests that in the context of imitation learning, the perception-action system is also configured to represent the underlying features (e.g. kinematics) of novel actions (atypical biological motion).

Chapter Two provided further support to the suggestion that atypical biological motion coding is a function of lower-level processing by decoupling the spatial compatibility between the observed and executed movements. Results showed imitation of atypical biological motion was similar when the atypical model was observed in spatially incompatible and compatible orientations. Given the spatially compatible trials involved the stimulus moving from left to right, the spatially incompatible trials controlled for movements in opposite direction but same horizontal orientation e.g. observing the stimulus move right to left (Brass et al., 2001), as well as orthogonal orientation e.g. top to bottom or bottom to top (Heyes et al., 2005). Reproducing topologically similar atypical biological motion kinematics during spatially incompatible imitation isolates the coding of biological motion to lower-level visuomotor processes as it cannot be a function of reproducing the spatial coordinates through higher-order cognitive processes (Heyes, 2011). Coding biological motion kinematics through lower-level processes supports the associative sequence learning (ASL) theory of imitation (Heyes

& Ray, 2000; Heyes, 2001; Brass & Heyes, 2005), which suggests that excitatory links or associations connect sensory and motor representations of the same action (Heyes & Ray, 2000), which are formed during learning and development (Schultz & Dickinson, 2000, Catmur & Heyes, 2011). In line with ASL theory, the coding of spatially incompatible atypical biological motion could be a function of the observed and executed movements being correlated e.g. observing atypical biological motion and imitating atypical biological motion, which is suggested to engage regions of the brain with mirror properties that enable action understanding (di Pellegrino et al., 1992; Ferrari et al., 2003).

The general finding of biological motion coding shown throughout all chapters of the current thesis supports the widely-held view that biological motion is coded and represented during observation of a stimulus (Cross et al., 2013; Cutting & Kozlowski, 1977; Grossman, Donnelly, Price, Pickens, Morgan, Neighbor et al., 2006; Johannson, 1973; Kilner et al., 2003; Saygin, Wilson, Hagler, Bates & Sereno, 2004). Further, the coding of all atypical biological motion models (atypical model – *Chapters Two, Four* and *Five*; atypical17 and atypical26 models – *Chapter Three*) is consistent with research that has shown imitation of velocity kinematics following observation of biological motion (Bisio et al., 2010; Wild et al., 2010). The adaption from a pre-existing movement pattern (typical biological motion) to a novel movement pattern (atypical biological motion) suggests that the lower-level visuomotor processes associated with direct coding of movement kinematics (Hayes et al., 2009; 2010) are engaged during the imitation learning protocol used in the current thesis.

This visuomotor processing of atypical biological motion supports neurophysiological research on the perception-action system, which suggests that the biological motion contained within the atypical model is likely to have been detected by the STS (Perrett et al., 1998; Jellema et al., 2000; Allison, Puce & McCarthy, 2000). The STS produces a visual description

of the model is then sent to the IFG and IPL where visual representation of the observed stimulus is formed for subsequent action coding (Cattaneo et al., 2010; Rizzolatti & Fadiga, 2014). As such, the visuomotor processing of atypical biological motion demonstrated in the current thesis provides support to the EP-M model of imitation (Hamilton, 2008), which suggests the perception-action system constitutes an indirect, parietal route for goal emulation and planning (EP) and a direct, frontal route for mimicry (M). Unmodulated imitation of atypical biological motion (*Chapter Two*) is likely to have engaged the direct M-route, which suggests the MTG forms a visual representation of the kinematic features of the atypical model that is sent to the IFG and mapped onto a motor representations containing the underlying atypical kinematic profile (see also, ASL model; Heyes, 2001). In contrast, the goal-directed modulation of movement time (*Chapter Three*) is likely to have engaged the EP route, which as it incorporates higher-order cognitive processes into the visual representation of the observed stimulus. The E-route connects the MTG and IPL and allows for emulation and understanding of the goal of an action, which in this case was imitating the goal of reaching the end-state-target. The P-route connects the IPL and IFG, which facilitates action planning and calculates the best way to achieve the goal e.g. reproduce the kinematics to reach the target, or get there as efficiently as possible (Bekkering et al., 2000). Importantly, the kinematics of the observed stimuli are included in this motor planning, which corroborates the suggestion from the current thesis that lower-level and higher-order cognitive processes are complimentary and embedded within the same general system to facilitate imitation of complex movements.

In addition to end-state-targets, the current thesis also demonstrates the top-down modulation of lower-level biological motion processing through social context in *Chapter Five*. Firstly, results showed that observing an anti-social eye gaze prime produced an up-

regulation of peak velocity such that it became more similar to that of the model, relative to observing a pro-social or neutral prime. By demonstrating that social context can impart control on information processing such that it either inhibits or enhances imitation, these data support the STORM model (Wang & Hamilton, 2012). In line with STORM, it is likely that the social control produced by engaging in the social prime activated the mPFC, which in addition to eye gaze primes (Wang et al., 2011b), has been shown to respond to a wide range of social cues (Kampe et al., 2003; Zink et al., 2008; Teufal et al., 2010). For example, fMRI results showed that during a hand-opening/closing SRC task with eye gaze priming, performing the task activated regions of the brain associated with mirror neurons (STS and IFG) and the pro- and anti-social eye gaze primes engaged mPFC. In accord with neurophysiological research on the perception action system (Buccino et al., 2004; Iacoboni et al., 2001) and the EP-M route (Hamilton, 2008), it is suggested that observation of the anti-social eye gaze prime up-regulated the mPFC prior to observation of the model stimuli such that it was primed for the visual representation of the atypical and typical biological motion models that were generated in the STS and IFG.

Importantly, while social context modulated imitation of peak velocity, it did not modulate the relative coding of the atypical and typical biological motion models. That is, imitation of atypical and typical biological motion kinematics were still scaled relative to the respective models following observation of the social primes. The lower-level processing of biological motion shown that activates both STS and IFG (Iacoboni, 1999; Di Dio et al., 2013), and higher-order cognitive processes associated with top-down modulation controlled by the frontal regions of the perception-action system (Hamilton, 2015), have also been confirmed during social modulation of imitation through dynamic causal modelling (DCM). DCM examines the information processing strength between neural substrates and has confirmed

connectivity between STS and IFG during observation of biological motion (Wang et al., 2011b). Moreover, DCM in the paper by Wang et al. (2011) confirmed the mPFC as the region that exerts top-down control on the mirror neuron system by demonstrating strong connectivity between mPFC and STS and IFG respectively.

6.5. Concluding Remarks and Considerations for Future Research

The experiments conducted within this thesis provides evidence as to the way in which biological motion is coded during imitation learning, and examines some of the attentional factors that modulate the process of imitation. As with most research, the answers that experiments produce often result in new questions being asked. For example, it would be interesting to translate the principles underpinning the imitation task used in the current thesis to more natural environments. All four experimental chapters were lab-based and computational, and were designed to minimise environmental context that may influence imitation. While the current thesis provides several examples of biological motion coding during imitation learning within these controlled contexts, the application of biological motion coding during imitation learning may be different in a real-world environment. For example, sports training in children requires periods of learning that can often involve observation of skills prior to physically practising them. It would be interesting to examine whether the environmental context of skill-based learning (e.g. observing an effector with biological properties, rather than a non-biological white dot) modulated the attention of the observer during acquisition of functional skills (Hayes et al., 2007). For example, eye movements may be more directed to an effector (Bach et al., 2007) when a skill is located to an area of the

body (e.g. hand/ foot) that contains social context, rather than observing the same movement performed by a non-contextualised geometric shape (e.g. white dot). Similarly, instructions may modulate what features of skills are acquired relative to what children believe they are learning (Stanley, Gowen & Miall, 2007), or what features of the movement those instructions direct attention towards (Posner, 1980; Safford et al., 2010).

In addition to sports training in children, the coding of biological motion during imitation learning could be examined in children and adults with ASC. Deficits in imitative behaviour in children and adults with autism have been acknowledged for a long time (Ritvo & Provence, 1953) and more recently, it has been suggested that this deficit stems from reduced neural activity within the perception-action system ('broken mirror hypothesis'; Ramachandran & Oberman, 2006; Williams, Whiten, Suddendorf & Perrett, 2001). While there is neurological (Oberman, Hubbard, McCleery, Altschuler, Ramachandran & Pineda, 2005; Pelphrey, Adolphs & Morris, 2004) and behavioural (Williams, Whiten & Singh, 2004) support of the 'broken mirror hypothesis', there is also evidence that the neurological processes underpinning imitation of biological motion are intact within autistic people (Bird, Leighton, Press & Heyes, 2007; Grecucci, Brambilla, Siugzdaite, Londero, Fabbro, et al., 2013; Sowden, Koehne, Catmur, Dziobek & Bird, 2016). Automatic imitation research suggests the underlying processing within the perception action system may be intact, which is corroborated by imitation learning research that has demonstrated the coding of movement time following observation of a stimulus and thus, engagement in the self-regulation of sensorimotor adaptations (Hayes, Andrew, Elliott, Gowen & Bennett, 2016). However, the imitation of biological motion kinematics has not yet been demonstrated in children or adults with autism (Hayes et al., 2016; Stewart et al., 2013; Wild, Poliakoff, Jerrison & Gowen, 2012), which could suggest that attention is important during the processing of visual

information for people with autism. *Chapter Five* is integral to this potential area of research as it is the first example of discrete biological motion kinematics being modulated by social priming during imitation learning. Moreover, the imitation task used in the current thesis has consistently been shown to be a valid measurement of biological motion coding. By combining a robust protocol with social priming effects in participants who were exclusively neurotypicals, there is a strong foundation on which to develop research into people with ASC and compare imitative responses. By continuing to compare the ways in which neurotypicals and people with ASC process visual information, any differences that are discovered have the potential to highlight deficiencies or provide alternative solutions to facilitate learning by observing and imitating for people with ASC.

Data from *Chapters Two, Three* and *Four* provide evidence that not only is biological motion coded during imitation learning, but that the coding is a function of lower-level visuomotor processing. In *Chapter Two*, it was found that when observation and imitation are spatially incompatible, atypical biological motion kinematics are coded and imitated accurately, and relative to the model. Imitation accuracy is also consistent when trials are spatially compatible. For imitation to remain accurate in spatially incompatible trials, the underlying kinematics, rather than the spatial properties, of the observed stimulus must be coded directly such that they can be transformed onto a spatially incongruent motor output. Importantly, the visual information provided on the monitor during the imitation task was designed to create the most natural imitation responses possible and thus, examine the fundamental nature of biological motion processing e.g. non-biological motion cursor to display the model kinematics, black background. With the evidence that biological motion kinematics is coded through lower-level processing, the protocol could be modified such that it reflected more relatable, real-world environments. For example, rather than observing a

non-biological dot, the study by Wild et al. (2010) examined imitation of movement kinematic by observing a video clip of a human hand moving at different speeds ('fast' or 'slow'). In both the current thesis, and the study by Wild et al., imitation required motor execution using the arm and hand; however, by replicating the effector in both observation and imitation, Wild et al. may have elicited motor priming (Bach & Tipper, 2007; Berger & Hadley, 1975) and consequently influenced imitation. Early neurophysiological research suggested non-biological effectors (e.g. tools) do not activate areas of the brain associated with mirror neurons compared with biological effectors (Gallesse et al., 1996; Perrett et al., 1990) and thus, could influence the processes underpinning imitation. Conversely, more recent data has found it is the nature of the stimuli (e.g. biological/ non-biological motion), not the effector that displays it, that primarily influences imitation (Di Dio et al., 2013). These data suggest that while the protocol in the current thesis provides a more explicit means of examining the imitation of biological motion kinematics, observing a human hand that displays the underlying kinematics of the atypical biological motion model and then physically replicating the movement may influence the way in which the visual stimulus is processed and consequently, imitation.

In addition to restricting the perceived environmental context, the protocol used in the current thesis allowed for explicit top-down modulation to be controlled. For example, *Chapter Two* displayed only the model stimuli to examine coding of biological motion at its most fundamental level. However, *Chapters Three, Four* and *Five* were modified such that end-state-targets (*Chapters Three* and *Four*) and social primes (*Chapter Five*) were displayed to examine the top-down influences on the lower-level processing on imitation of biological motion. The most interesting of these findings was that social primes modulated the variability, but not accuracy, of imitation. Whilst social primes have previously been shown

to decrease variability (Roberts et al., 2016), the data from *Chapter Five* demonstrates that social primes modulate biological motion coding during imitation learning differently to automatic imitation (Brass et al., 2001; Lakin et al., 2003; Wang, et al., 2011a; Bisio et al., 2010) and it is important to understand the context in which the social primes have induced modulatory effects e.g. by influencing the efficacy of the representation formed during observation; by up/ down-regulating the regions of the brain associated with coding visual stimuli. It was suggested in *Chapter Five* that this may be related to the more complex processes underpinning imitation learning, compared with automatic imitation (Heyes, 2011) but the social primes (e.g. eye gaze, word scrambles) also modulate imitation differently. This may be due to eye gaze primes providing a subtler social context compared to word scrambles, where the words contain explicit connotations that suggest their intent (Heyes, 2011). For example, while direct- and averted-gaze were used as the pro- and anti-social primes in the current thesis, it is unknown as to whether they were observed as 'positive' and 'negative' primes. Therefore, a means of confirming social primes have the desired effect would be to issue short questionnaires upon the completion of the imitation task. This would provide quantifiable evidence that the primes did, or did not, influence imitation in line with the purpose of the experiment.

6.6. Summary

To conclude, the current thesis used a novel behavioural protocol to examine the coding of biological motion during imitation learning. The protocol required participants to observe, then imitate a combination of biological and non-biological motion models that

displayed both novel and existing movement patterns to examine whether biological motion was coded, what underlying processes were involved in the coding, and whether the underlying processes could be modulated during imitation learning.

Primarily, the current thesis demonstrated that biological motion is coded during imitation learning by showing differences in imitation between the typical and constant velocity models (*Chapters Four* and *Five*), atypical and typical models (*Chapters Two, Four* and *Five*) and atypical17 and atypical26 models (*Chapter Three*). Key within these findings was the coding of atypical biological motion, which displayed a novel movement pattern that was not already part of the motor repertoire and could not be imitated through recruitment of existing motor patterns (e.g. typical model) or chance. Scaled imitation of atypical17 and atypical26 models confirmed that the coding of biological motion was not a function of strategy based on detecting faster movements that occurred towards the start of the movement, but the explicit kinematic structures of the respective models (e.g. different magnitudes of peak velocity that occurred at 17% and 26% of the movement respectively).

In addition to imitating the respective model kinematics, *Chapter Two* confirmed that the coding of biological motion kinematics was isolated to lower-level visuomotor processes by spatially decoupling the observed and imitated movements. If imitation was governed by higher-order cognitive processes associated with spatial compatibility, imitation would have been more accurate during spatially compatible, than incompatible trials. That imitation of the atypical biological motion kinematics was similar during spatially incompatible and compatible trials demonstrated the processing of the visual stimuli was not related to the spatial positioning of salient movement features (e.g. peak velocity occurred on the right-side of the monitor), but the underlying kinematics contained within the models. As such, the representations formed during observation of the stimuli could be visually transformed to

produce scaled imitation of atypical biological motion kinematics during spatially incompatible trials.

As well as demonstrating the lower-level processes involved in biological motion coding, *Chapters Three* and *Five* suggested that higher-order cognitive processes operate alongside lower-level processes to modulate features of imitation. *Chapter Three* included end-state-targets to examine whether goal-directed imitation would influence the underlying processes involved in biological motion coding. Results showed that while the coding of kinematics was not attenuated, movement time became less accurate when end-state-targets were present during observation and imitation of the model stimuli. As movement time was modulated, but not at the expense of coding the biological motion kinematics, the imitation data from *Chapter Three* demonstrated a complimentary relationship between lower-level visuomotor processing and higher-order cognitive control. Similarly, *Chapter Five* demonstrated that observing social primes prior to observation of the stimuli modulated the processing of the visual stimuli. Observation of the anti-social prime generated up-regulatory effects where imitation of peak velocity was closer to that of the models and corroborated the active-self theory, which suggests the perspective from which a prime is observed relative to one's self influences the way in which the prime interacts with the information processing. In addition, the observation of both pro- and anti-social primes reduced the variability of imitation such that the percentage-time-to-peak-velocity and movement time became more like the respective models, which suggests that a general priming effect, rather than the specific nature of the prime, could modulate the efficacy of the representation formed during observation of biological motion.

When taken together, the results in the present thesis contribute to an extension of the current literature in several ways. Firstly, the protocol used in all four experimental chapters

provided a more applicable measurement of biological motion coding by incorporating discrete kinematic markers into the design of the models that were also used in the analysis of imitation accuracy. Second, results showed that unmodulated biological motion kinematics is coded through lower-level visuomotor processes, which suggests that the way in which movements are performed are incorporated into visual representations that are mapped directly onto motor outputs for imitation, thus improving the understanding of how visual information is processed. This improved scientific understanding can inform anyone who implements learning by observing (e.g. coaches, teachers) in their daily life. Similarly, demonstrating top-down modulation of lower-level processing both extends the current imitation learning literature by demonstrating a complimentary relationship between the two and has the potential to inform learning techniques that involve goals or social context. In addition to extending the current literature and informing practical learning, the social priming study also represents the conception of a body of research examining biological motion coding in people with ASC, which has the potential to be highly impactful.

7. References

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8. Appendix

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Atypical biological motion kinematics are represented by complementary lower-level and top-down processes during imitation learning.

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1. Introduction

Imitation is a powerful mechanism that supports human interaction. In familiar social settings, imitation involves the automatic activation of a motor response triggered by observing a similar motor action (Chartrand & Bargh, 1999; Heyes, 2001, 2011; Heyes et al., 2005). For example, individuals execute faster pre-specified movements (e.g., finger tapping) when observing biologically compatible (finger tapping), compared to incompatible (finger lifting), movements (Brass, Bekkering, & Prinz, 2001; Stürmer, Aschersleben, & Prinz, 2000). The shorter motor reaction times occur independent of task instructions, which suggests involvement of automatic sensorimotor processes linking perception and action (Brass & Heyes, 2005; Prinz, 1997).

To understand if the automatic sensorimotor effects are developed through experience, and linked to a general mechanism incorporating processes associated with perception, action and

attention (Leighton, Bird, & Heyes, 2010), studies have examined automatic imitation following correlated sensorimotor training (Bird, Brindley, Leighton, & Heyes, 2007; Catmur, Mars, Rushworth, & Heyes, 2011; Catmur, Walsh, & Heyes, 2007, 2009; Cavallo, Heyes, Becchio, Bird, & Catmur, 2013; Heyes et al., 2005). For example, individuals performed a countermirror protocol that required compatible or incompatible sensorimotor training (Catmur et al., 2007). During compatible training, individuals executed index-finger movements, whilst simultaneously observing index-finger movements. During incompatible training, individuals 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 specialized mechanism that mediates (Meltzoff & Moore, 1997) the translation of visual information into a motor action.

Of primary interest to the present study is the suggestion that similar sensorimotor processes operate during automatic imitation and imitation learning (Brass & Heyes, 2005; Buccino et al., 2004; Heyes, 2011; Iacoboni, 2009). Like the countermirror principle, imitation learning often requires the sensorimotor system to represent a novel biological motion across consecutive imitation trials. Although there is strong evidence that biological motion is processed during automatic imitation (Brass, Bekkering, Wohlschlaeger, & Prinz, 2000; Heyes et al., 2005; Press & Heyes, 2008) and interpersonal observation–execution imitation tasks (Kilner, Paulignan, & Blakemore, 2003), support from imitation learning studies has typically been based on protocols that manipulated the speed of the imitated movement (Bisio,

Stucchi, Jacono, Fadiga, & Pozzo, 2010; Hayes, Timmis, & Bennett, 2009; Wild, Poliakoff, Jerrison, & Gowen, 2010).

Although participants have been shown (Wild et al., 2010) to imitate different movement speeds (e.g., slow, medium, and fast upper-limb aiming movements), it is notable that the observed stimulus was representative of typical aiming movements. Thus, it remains possible that imitation was limited to recognizing differences in movement speed between observations, as opposed to representing the underlying biological motion kinematics. In this case, the feedforward contribution to motor execution could have been associated with an individual recruiting and rescaling a preexisting motor representation of a familiar and meaningful aiming movement (Hayes, Roberts, Elliott, & Bennett, 2014; Hayes et al., 2009). This would imply imitation was based on higher-order semantic processes (Rumiati, Papeo, & Corradi-Dell'Acqua, 2010; Rumiati et al., 2005), as opposed to lower-level sensorimotor processes representing the observed biological kinematics.

In the current study, we adopted a novel protocol that enabled us to directly examine biological motion processing during imitation learning. In addition to displaying a constant velocity control model, we manipulated the structure of two experimental models so that peak velocity in the aiming movements no longer occurred at the typical mid-point (40–60% of the total time) of the trajectory (Elliott, Helsen, & Chua, 2001). With such stimuli, imitation can be quantified according to timing and magnitude of velocity, which in combination would not reflect the kinematics of typical aiming movements (Hayes et al., 2014). Imitation in this context is not solved by merely recruiting an existing sensorimotor representation associated with a typical upper-limb aiming movement and rescaling (Schmidt, 1975) the representation to meet the goal movement time of 1700 ms. Instead, because the novel atypical biological motion profiles are unlikely to be represented in the sensorimotor repertoire of the participants

(Hayes et al., 2014), imitation requires the specific velocity profile to be represented. Following this logic, we compared imitation learning of two different biological motion models, in which percentage-time-to-peak-velocity occurred at 17% or 26% of the total movement time (henceforth atypical17 and atypical26), and thus earlier than normally expected when aiming to a target. By maintaining equal movement time and amplitude, magnitude of peak velocity also differed between the biological motion models (atypical17 = 0.37 mm/ms; atypical26 = 0.24 mm/ms). Finally, given that the lower-level processes that code biological motion kinematics are modulated by various top-down processes (Bekkering, Wohlschlaeger, & Gattis, 2000; Heyes & Bird, 2007; Leighton et al., 2010; Rumiati et al., 2005; Southgate & Hamilton, 2008; Wang & Hamilton, 2012), we displayed motion stimuli as a non-human agent (a white dot) to control social context, and in the presence or absence of end-state-targets. The latter manipulation is important because previous work (Hayes, Hodges, Huys, & Williams, 2007; Wild et al., 2010) has shown that the imitation of biological motion is attenuated in the presence of an end-state-target. In this context, the end-target provides a salient task-relevant (Leighton et al., 2010) environmental visual cue that modulates attention so that this feature (target attainment) is prioritized and represented during imitation. The removal of end-state-targets in half of the present experimental trials enabled us to develop a protocol that examined biological motion kinematics during true imitation (Cook & Bird, 2012; Vivanti & Hamilton, 2014).

With a behaviorally realizable but atypical biological motion (i.e., atypical17; atypical26), represented as a non-human agent, it was expected that participants would imitate in accord with the observed biological kinematics (Hayes et al., 2014) and thus produce movements scaled to both timing and magnitude of peak velocity. Because of the constraints on human movement imposed by the neuro-muscular system (Abend, Bizzi, & Morasso, 1982), we did

not expect participants to move with constant velocity having observed the constant velocity stimulus, or to execute a kinematic profile that resembled the atypical motion kinematics. Rather, we anticipated participants would recruit a pre-existing motor response and thus exhibit time of peak velocity that was similar to typical aiming movements. Finally, it was anticipated that imitation of atypical biological motion would be more accurate in the absence, compared to presence, of end-state-targets. In the absence of end-state-targets, there should be minimal contribution from top-down attentional processes, thus encouraging participants to focus on representing the characteristics of lower-level visual stimuli during imitation learning.

2. Materials and methods

2.1. Participants

Data were recorded from twenty participants (age range 18–21 years) who volunteered for the study. All participants had normal or corrected-to-normal vision and gave written informed consent. The experiment was designed in accordance with the Declaration of Helsinki and was approved by the ethics committee of the host University.

2.2. Apparatus and procedures

The apparatus consisted of a PC (Dell Optiplex GX280), a 21-in CRT computer monitor (IIyama Vision Master 505), and a graphics tablet with a hand-held stylus (WACOM Intuos 3). The CRT monitor operated with a spatial resolution of 1280 × 1024, and a refresh rate of

85 Hz. Visual stimuli were generated via MATLAB (The Mathworks, Inc), using Cogent 2000 toolbox (www.vislab.ucl.ac.uk/cogent.php).

Participants were required to observe and imitate the movement of a model (a white cursor, diameter = 8 mm) presented on the 21-in CRT monitor. The model displayed a single horizontal trajectory that originated from a home-target positioned on the left-hand side of the screen. The amplitude of the movement was 200 mm, with a movement time of 1700 ms, and ended on the right-hand side of the monitor. For the end-state-target condition, two red circles representing home-target and the end-state-target (diameter = 16 mm) were positioned at center-left (home) and center-right (end-state) of the monitor (Fig. 1A). To examine imitation of biological motion, three models were created: atypical (atypical17; atypical26) or constant velocity (Fig. 2). The atypical models displayed a velocity profile that was positively skewed so that peak occurred at 17% or 26% of movement time, and with a magnitude of 0.37 mm/ms and 0.24 mm/ms, respectively. The models were created by a human volunteer who practiced the two atypical goal-directed aiming movements using a hand-held stylus on a graphics tablet until a white cursor, which represented the stylus, moved from a left-hand home-target to a right-hand end-state-target in a movement time of 1700 ms. The displacement time-series data recorded from a successful practice trial for each model was selected to create the models. The method of using a human to generate the models was critical because it ensured the kinematics of the movement was biological in origin, and thus the movement was achievable. 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, but with a constant velocity trajectory that had no deviations in the perpendicular axis.

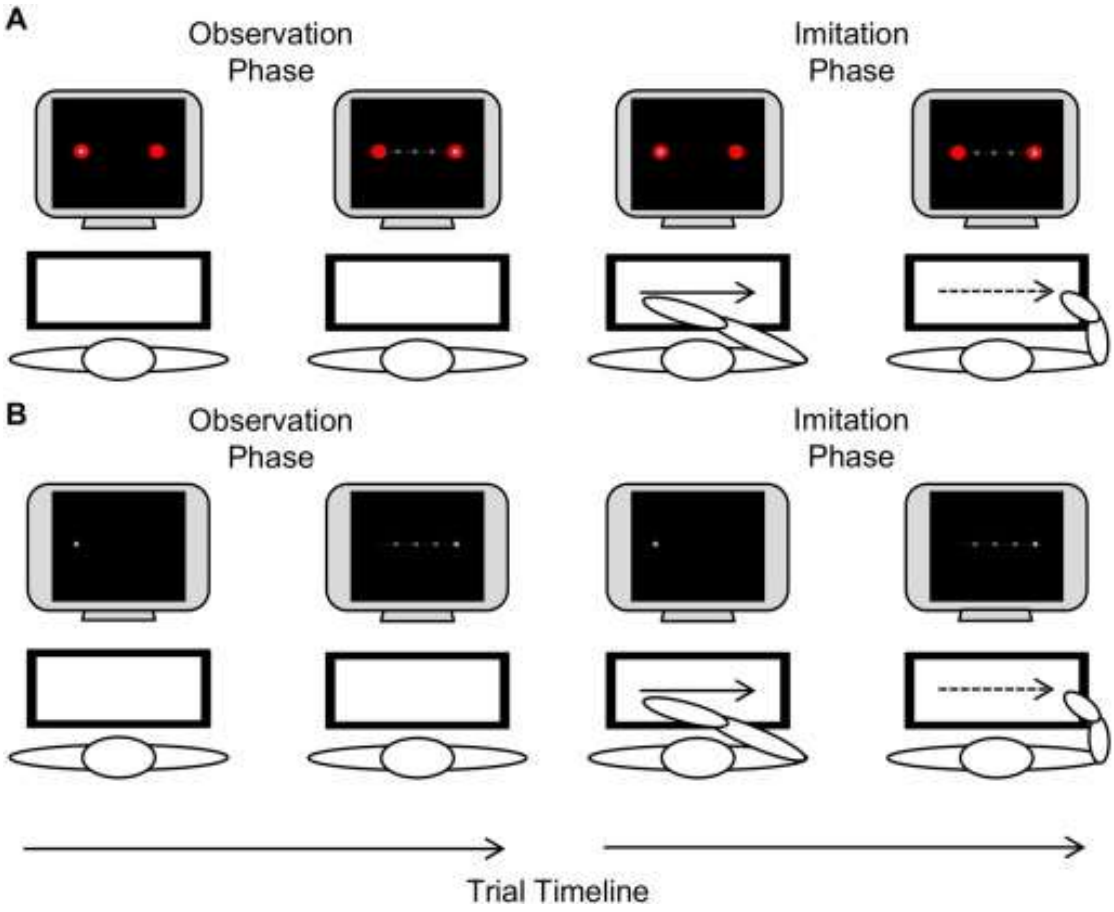

Fig. 1. A visual representation depicting a single trial in the end-state-target-condition (A) and no-end-state-target condition (B). The apparatus outlined in Panels A and B is a CRT monitor and a graphics tablet. The trial timeline arrows at the bottom of the figure indicate the Observation Phase and Imitation Phase. During the Observation Phase, the non-human agent model is positioned in the left-hand home target (A) and left-hand space (B). The model (atypical17 or atypical26 or constant velocity) displays a horizontal movement of 200 mm from the left-hand home target to an end-state-target (A) or end-space in the no-end-state-target-condition. The model has a movement time of 1700 ms. The Imitation Phase commences with the white cursor positioned in left-hand home target (A) or left-hand space (B). A participant imitates the observed model by controlling a stylus on the graphics tablet.

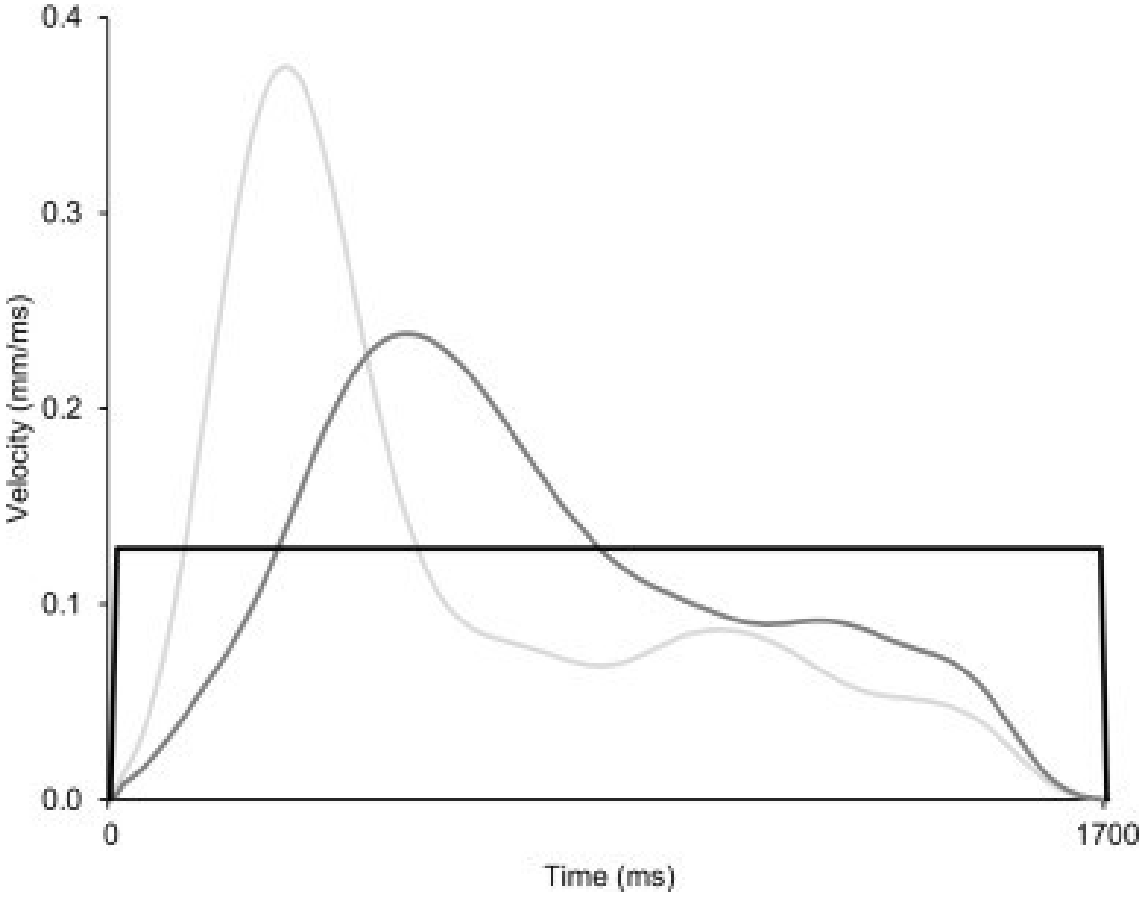

Fig. 2. The velocity profiles for atypical17 model (light gray trace; peak), atypical26 model (dark gray trace), and constant velocity control model (black trace).

Prior to the experimental trials, all participants completed a familiarization period that replicated the conditions of the imitation task. Participants sat on a chair in front of the CRT monitor and held the stylus in their preferred hand. The participants performed four familiarization trials; 2 trials representing the end-state-target condition (see Fig. 1A) performed in the imitation task, and 2 trials representing the no-end-state-target condition (see Fig. 1B) performed in the imitation task. Each trial commenced with the model being positioned in the center of the home-target. The participants observed the model display a movement from the home-target to an end-target (end-state-target condition), or end space

(no-end-state-target condition), with a constant velocity trajectory and a movement time of 1700 ms. A constant velocity trajectory was used to ensure construct validity by preventing participants from experiencing biological motion before the imitation trials. Participants were not informed about the agency of the model or duration of the movement time. Following observation of the model, participants moved the cursor from the center of the monitor to the center of the home-target, and clicked the lower-button on the stylus. In an end-state-target condition, the two targets remained on the screen as the participant imitated the model. In a no-end-state-target condition, the two targets were removed before a participant imitated the model. To finish imitation, participants clicked the lower-button on the stylus a second time once the cursor was located in the end-state-target, or end-space in the no-end-state-target condition. After familiarization, all participants confirmed they understood the model, the end-state-target and no-end-state-target conditions, the instruction to imitate, and the sensorimotor association between the stylus on a graphics tablet, and the corresponding movement of cursor on the monitor.

The imitation task comprised 14 blocks of 6 trials (84 trials). A block contained each of the 6 combinations of target (end-state-target, no-end-state-target) and velocity model (atypical17, atypical26, constant) presented in random order. A trial commenced with an observation phase where the home-target (red) was displayed on the monitor for 1000 ms, before disappearing for 1000 ms, and being replaced by a model positioned in the same location. Depending on the trial type, the model moved to an end-state-target (Fig. 1A) or end-space in the no-end-state-target (Fig. 1B) condition, with one of three velocity models. After observing the model, participants imitated the movement as per the instructions given in the familiarization period.

2.3. Statistical analysis

To quantify imitation performance, and imitation of atypical biological motion, we extracted movement kinematics exhibited by the participants on each trial. The start of movement was defined as the time that the center of the cursor moved beyond the perimeter of the home-target, and the end was calculated when the participant clicked the lower-button on the stylus. For each imitation attempt, the 2-dimensional displacement data were filtered using a low-pass (8 Hz) autoregressive filter. These data were differentiated using a central difference algorithm to obtain velocity. A MATLAB routine extracted the primary movement occurring in the x-axis and identified the following dependent variables: movement time, peak velocity, and percentage-time-to-peak-velocity (i.e., time to peak velocity / movement time) × 100). The two velocity variables were chosen for analysis because they most reflected the difference between the two atypical biological motion models. Intra-participant means from the 14 trials per condition were calculated for each dependent variable and submitted to separate Model (atypical17; atypical26; constant velocity) x Target (end-state-target; no-end-state-target) repeated measures ANOVAs. Alpha was set at $p < 0.05$, follow-up testing used the Tukey post-hoc procedure, and partial eta squared (ηp2) expressed the size of the effect.

3. Results

3.1. Movement time

As illustrated in Fig. 3, the presence of an end-state-target [$F(1, 19) = 36.61$, $p < 0.05$, ηp2 = 0.49] modulated movement time, with significantly shorter and more accurate movement times imitated in the absence (M = 2156 ms), compared to the presence (M = 2294 ms), of an end-state-target. Although there was no significant difference in

movement times when imitating the atypical17 (M = 2121 ms) and atypical26 (M = 2191 ms) models, the main effect [$F(2, 38) = 17.90$, $p < 0.05$, $\eta p2 = 0.66$] indicated these two movement times were significantly shorter ($ps < 0.05$) and more accurate than imitating the constant velocity (M = 2362 ms) model. The interaction concerning model and target [$F(2, 38) = 3.51$, $p < 0.05$, $\eta p2 = 0.16$] indicated that significantly shorter and more accurate movement times were performed in the no-end-state-target compared to the end-state-target condition ($ps < 0.05$) when viewing atypical17 and atypical26 models. This effect was not significant when imitating constant velocity.

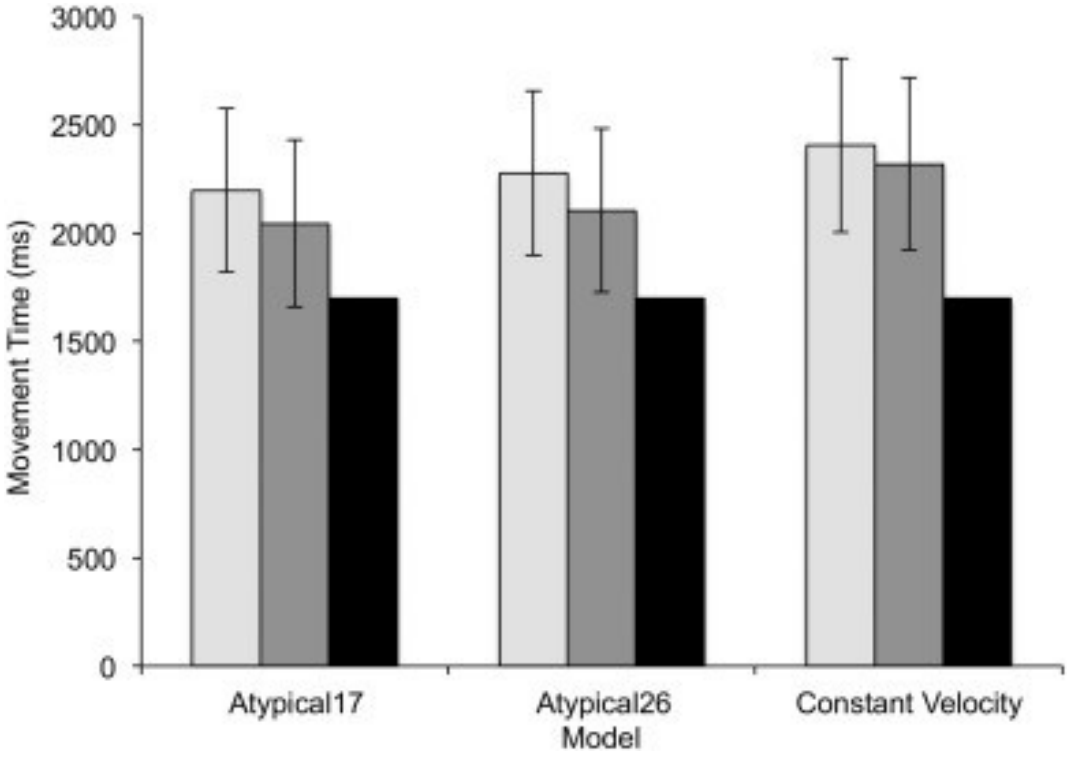

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Fig. 3. Mean movement time data (ms) as a function of model (atypical17, atypical26 and constant velocity) and target condition (light gray = end-state-target; dark gray bar = no-end-state-target). The criterion model data for atypical17 and atypical26 is represented in the black bars. Error bars (±) display the standard error mean.

3.2. Peak velocity

An effect of Model [$F(2, 38) = 59.56$, $p < 0.05$,$\eta p2 = 0.76$] indicated the magnitude of peak velocity was significantly greater when imitating the atypical17 model (M = 0.24 mm/ms) compared to the atypical26 (M = 0.19 mm/ms) and constant velocity (M = 0.15 mm/ms) models. Moreover, the magnitude of peak velocity was significantly ($p < 0.05$) greater when imitating the atypical26 compared to the constant velocity model. As illustrated in the left-hand and center portions of Fig. 4, the magnitude of peak velocity executed by the participants in the atypical17 and atypical26 conditions (gray bars) was scaled (i.e., more similar) to peak velocity displayed by the model (black bar). However, peak velocity was not modulated by the presence or absence of an end-state-target [$F(1, 19) = 1.48$, $p > 0.05$, $\eta p2 = 0.07$], irrespective of how it was combined with the model stimulus [$F(2, 38) = 1.54$, $p > 0.05$, $\eta p2 = 0.17$].

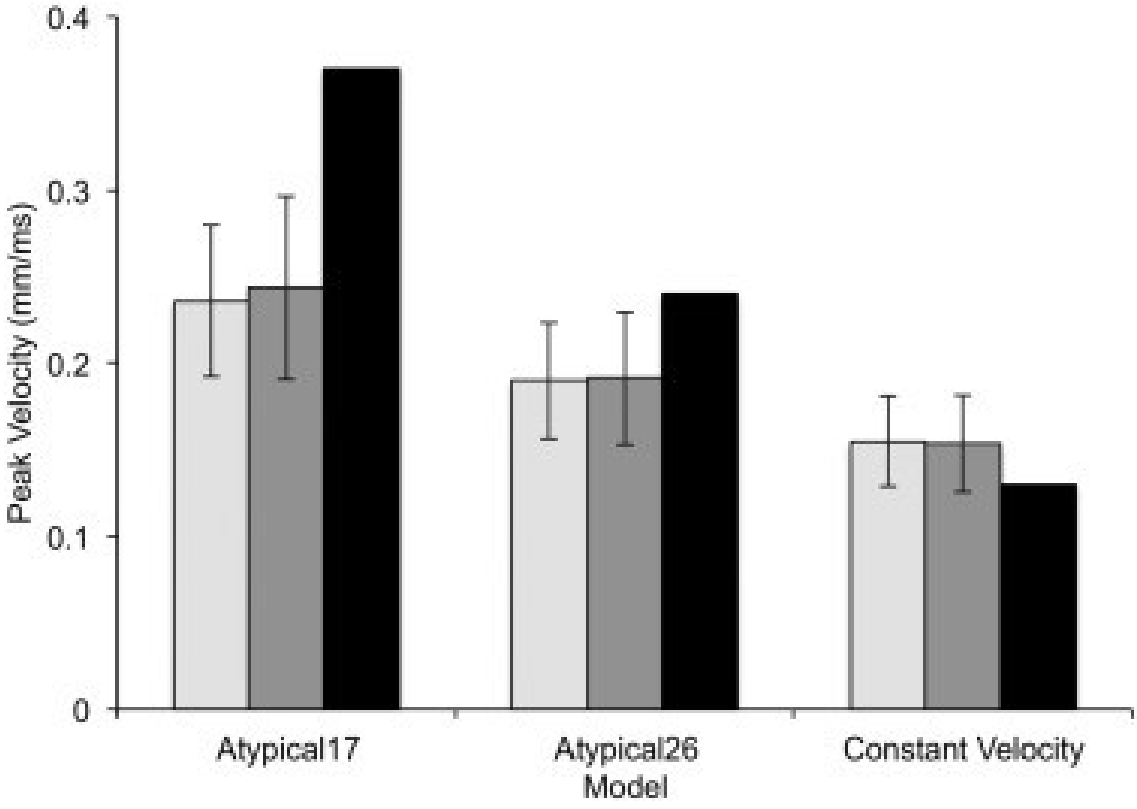

Fig. 4. Mean peak velocity data (mm/ms) as a function of model and target condition. The target conditions are displayed in the light gray bar (end-state-target) and dark gray bar (no-end-state-target). The criterion model data for atypical17 and atypical26 is represented in the black bars. Error bars (±) display the standard error mean.

3.3. Percentage-of-time-to-peak-velocity

An effect of Model [$F(2, 38) = 68.99$, $p < 0.05$, $\eta p2 = 0.78$] indicated peak velocity occurred significantly earlier in the movement when imitating the atypical17 model (M = 22%) compared to both the atypical26 (M = 29%) and constant velocity (M = 38%) models ($ps < 0.05$). As illustrated in Fig. 5, the gray bars indicate the temporal occurrence of peak velocity in the atypical17 and atypical26 conditions was scaled to peak velocity displayed by the model (black bar). This effect can also be seen from an exemplar velocity trace in Fig. 6. When imitating the atypical17 (dark gray trace) model, peak velocity occurred significantly earlier in the movement than the atypical26 (light gray trace) model. When imitating the constant velocity model, peak velocity occurred toward the midpoint of the movement (black trace). Although there was no main effect for Target [$F(1, 19) = 1.58$, $p > 0.05$, $\eta p2 = 0.08$], there was an interaction concerning Model and Target [$F(2, 38) = 11.40$, $p < 0.05$, $\eta p2 = 0.35$]. Percentage-of-time-to-peak-velocity occurred earlier in the movement in the end-state-target condition compared to the no-end-state-target condition when imitating the atypical17 and atypical26 models ($ps < 0.05$). This effect was reversed when imitating constant velocity model.

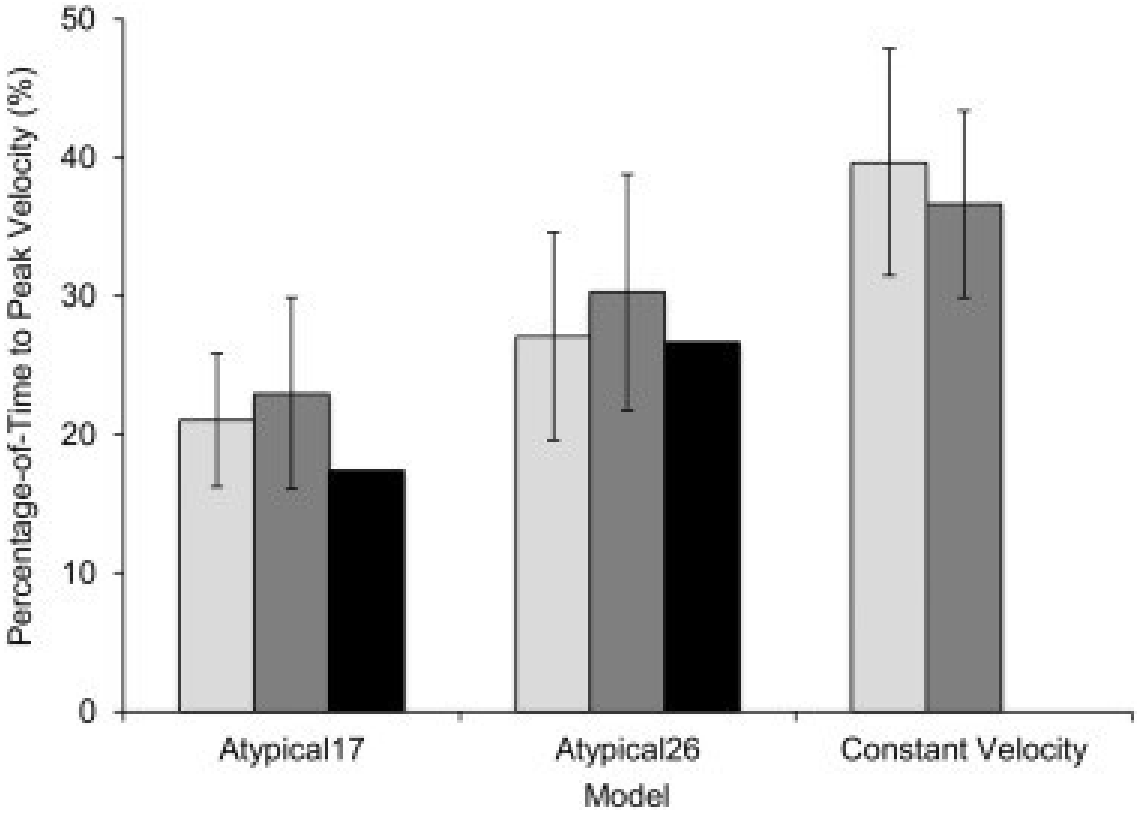

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Fig. 5. Mean percentage-time-to-peak-velocity (%) as a function of model and target condition. The target conditions are displayed in the light gray bar (end-state-target) and dark gray bar (no-end-state-target). The criterion model data for atypical17 and atypical26 is represented in the black bars. Error bars (±) display the standard error mean.

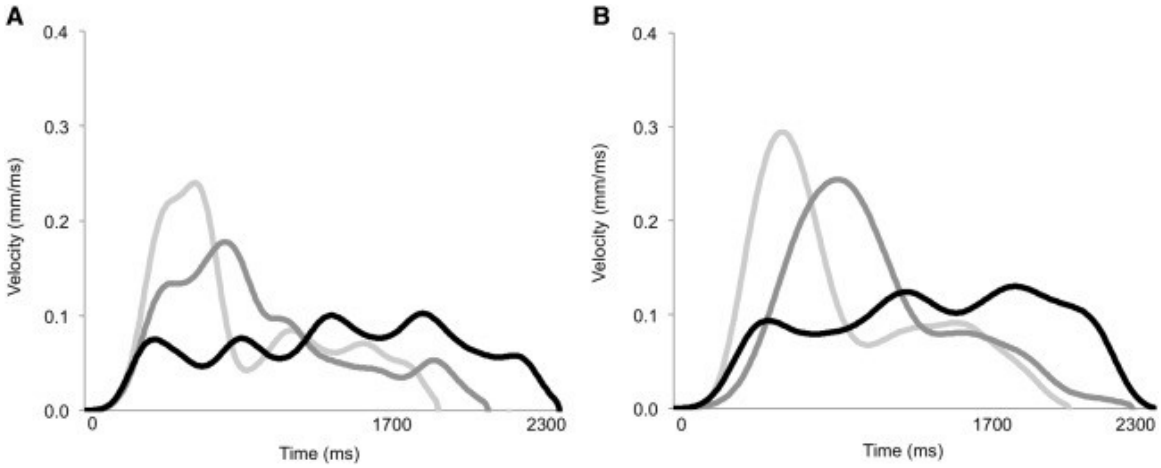

Fig. 6. The velocity profiles are exemplar data from a representative participant imitating atypical17 model (light gray trace; peak), atypical26 model (dark gray trace), and the constant velocity control model (black trace) in the no-end-state-target (A) and end-state-target (B) conditions. The 1700 ms marker displayed on the x axis indicates the total movement time displayed by the three models.

4. Discussion

We examined the representation of biological motion kinematics during imitation learning using a novel protocol that systematically manipulated the structure of a model's kinematic profile. The percentage-time-to-peak-velocity data supported our expectations by indicating peak velocity occurred significantly earlier in the movement after imitating both the atypical17 and atypical26 models. Moreover, while movement time was similar in these conditions, the magnitude of peak velocity also differed in accord with the atypical biological motion models. Imitation of both atypical17 and atypical26 models was confirmed by the data showing participants exhibited peak velocity significantly later (38%) in the movement in the

constant velocity control condition. Moreover, and as displayed in Fig. 6 (black traces in A and B), the exemplar velocity profile(s) illustrates a relatively flat, and stable, trajectory that contains a number of discontinuities. The fact the velocity profile was not bell-shaped suggests participants attempted to imitate the constant velocity model, rather than recruiting a movement trajectory based on internal (pre-existing motor priors) and external (amplitude and speed of movement) constraints of the task. Moreover, the low peak, and discontinuities could be the result of error minimization using visual feedback (Elliott et al., 2001), and/or sensorimotor noise associated with anatomical and physiological constraints of the motor system (Abend et al., 1982).

As expected, the findings also showed that imitation learning was modulated by the presence or absence of end-state-targets. Having observed the two atypical biological models in the absence of end-state-targets, participants exhibited shorter movement times, which were more accurate (M = 2156 ms) compared to when end-state-targets were present (M = 2294 ms). As suggested previously (Wild et al., 2010), this effect was unlikely to be associated with differences in movement amplitude, which was 6 mm shorter when end-state-targets were absent.1 Neither was it a function of greater average acceleration, which was less in the absence of end-state-targets (i.e., similar peak velocity but achieved later). Although not measured in the present experiment, an explanation for the less accurate imitation of movement time in the presence of end-state-targets is that participants paid more attention (Leighton et al., 2010) to target attainment and thus were more goal-directed during movement execution. As a consequence, it is likely they focused more on aiming to position the cursor in the end-target, which resulted in proportionately more time after peak velocity in the deceleration phase (Elliott, Hansen, Mendoza, & Tremblay, 2004).

The specificity of the aforementioned goal-directed imitation effect is important from a

theoretical position because the decrease in movement time accuracy in the end-state-target condition did not lead to a concomitant decrease in the imitation of atypical biological motion kinematics. Also, there was an interaction between the biological nature of observed stimulus (biological motion versus constant velocity) and end-state-target condition. For instance, participants exhibited more accurate movement time in the absence of end-state-targets when observing biological motion but not constant velocity. This effect is somewhat consistent with the suggestion that multiple goals (kinematics; end-state-target-goal), as well as other salient factors in the environment (Leighton et al., 2010), are represented when imitating different movements (Bekkering et al., 2000; Hamilton, 2008). Unlike previous work that typically demonstrated an action-goal (to grasp an ear) was prioritized (hierarchal goal representation) at the expense of biological kinematics (Bekkering et al., 2000; Hamilton, Brindley, & Frith, 2007; Hayes, Hodges, Scott, Horn, & Williams, 2007; Wohlschlager, Gattis, & Bekkering, 2003), we showed the attainment of an end-state-target goal did not affect the representation of biological kinematics. Our findings build upon the aforementioned effects by indicating that top-down and lower-level processes operate within an embedded system that is less hierarchal, and perhaps more complementary (Buxbaum & Kalénine, 2010; de Lange, Spronk, Willems, Toni, & Bekkering, 2008; Heyes, 2011), with the contribution of these processes modulated by the nature of task context. When the biological movement kinematics are novel, as per our atypical biological motion, both processes operate to represent movement kinematics and the end-state-target goal.

To minimize the potential modulation of biological motion processing by top-down factors associated with goal coding (Bekkering et al., 2000), attention/salience (Leighton et al., 2010), teleological reasoning (Csibra & Gergely, 2007) and social modulation (Wang & Hamilton, 2012), the atypical biological models were observed as non-human agents in the absence of

end-state-targets. The finding of temporal correspondence (Gangitano, Mottaghy, & Pascual-Leone, 2001) between observed (atypical17; atypical26) and imitated movement kinematics is therefore consistent with biological motion being processed through lower-level visuomotor processes operating in the human mirror-mechanism (Brass & Heyes, 2005; Casile et al., 2010; Dayan et al., 2007; Press, Cook, Blakemore, & Kilner, 2011). Detection of biological motion is suggested to occur in a neural substrate associated with the posterior STS (Allison, Puce, & McCarthy, 2000), while coding the kinematic properties of an observed action (Hamilton, 2008; Iacoboni, 2009) is suggested to occur in the fronto-parietal mirror-system (Di Dio et al., 2013; Press et al., 2011). Within the fronto-parietal mirror mechanism, the premotor region has been associated with coding the temporal features of visual information through analysis of MEPs during different phases of a grasping action (Gangitano et al., 2001). Moreover, evidence that certain phases of movement are reflected in time-synchronized neural activation (e.g., greatest activation during display of maximal grip aperture), has been suggested to indicate online visual processing during observation of biological motion. We concur with this reasoning and suggest the finding of temporal correspondence between the model and imitation of atypical biological motion was in part based on the online visual processing of such motion during each observation trial. Such findings of continual matching of action-execution with action-observation are consistent with our previous work on biological motion coding during observational practice (Hayes et al., 2014).

In summary, the findings in the present experiment showed atypical biological motion kinematics was represented during imitation learning, both in the presence and absence of end-state-targets. Imitation of biological motion kinematics involves top-down attentional and lower-level visuomotor systems, which operate as complementary processes.