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 Low Fidelity Imitation of Atypical Biological Kinematics in Autism Spectrum Disorders Is Modulated by Self-Generated Selective Attention.

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1   Low fidelity imitation of atypical biological kinematics in autism spectrum disorders is

2   modulated by self-generated selective attention

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1   **Abstract**

2   We examined whether adults with autism had difficulty imitating atypical biological

3   kinematics. To reduce the impact that higher-order processes have on imitation we used a

4   non-human agent model to control social attention, and removed end-state target goals in half

5   of the trials to minimise goal-directed attention. Findings showed that only neurotypical

6   adults imitated atypical biological kinematics. Adults with autism did, however, become

7   significantly more accurate at imitating movement time. This confirmed they engaged in the

8   task, and that sensorimotor adaptation was self-regulated. The attentional bias to movement

9   time suggests the attenuation in imitating kinematics might be a compensatory strategy due to

10   deficits in lower-level visuomotor processes associated with self-other mapping, or selective

11   attention modulated the processes that represent biological kinematics.

12

13   **Key words:** autism spectrum disorders, imitation, biological motion kinematics, attention

14

1   **Introduction**

2

3   Imitation is a powerful mechanism for learning new sensorimotor behaviours (e.g. throwing a

4   Frisbee) as well as for developing socio-cognitive skills such as rapport (Chartrand & Bargh,

5   1999) and affiliation (Lakin & Chartrand, 2003). One way humans acquire these behaviours

6   is by copying a novel movement displayed by another person. This process is defined as 'true

7   imitation' because an observer is required to copy the properties of human movement

8   (biological motion) after observing a model, rather than being able to merely reproduce the

9   movement using an already learned movement pattern based on previous experience (Byrne

10   & Russon, 1998). In the context of human movement, biological motion is the visual-sensory

11   information contained in a movement that describes a particular action (Johansson, 1973;

12   Kozlowski & Cutting, 1977). For example, a person can be judged to be walking based on

13   how the arms and legs move in relation to each other. Therefore, during 'true imitation'

14   (henceforth imitation) attention is directed to the biological motion kinematics (joint

15   configurations; limb velocity) of the observed person/model. Over repeated observations and

16   physical attempts at imitating the model, a new sensorimotor pattern is represented and

17   refined based on the available afferent and efferent sensorimotor feedback.

18   The mechanism underpinning imitation combines higher-order cognitive/attention and

19   lower-level visuomotor processes (Bandura, 1977; Byrne & Russon, 1998; Heyes, 2001)

20   embedded within a system linking perception with action (Prinz, 1997). Although not fully

21   understood, individuals with autism spectrum disorders (henceforth autism) exhibit different

22   neuropsychological processes and behaviour during imitation compared to typically

23   developed individuals (Edwards, 2014; Hamilton, 2013; Vivanti & Hamilton, 2014;

24   Williams, Whiten, & Singh, 2004). That is, people with autism often imitate the end-state

25   goal (to reach a target) of an action (Bird, Leighton, Press, & Heyes, 2007; Hamilton,

1   Brindley, & Frith, 2007; Wild, Poliakoff, Jerrison, & Gowen, 2012), but show difficulties

2   imitating the style (i.e. a gentle or harsh hand action) in which the movement goal is achieved

3   (Hobson & Lee, 1999; Perra et al., 2008; Rogers, 1999; Rogers, Bennetto, McEvoy, &

4   Pennington, 1996; Rogers, Hepburn, Stackhouse, & Wehner, 2003; Rogers & Pennington,

5   1991; Salowitz et al., 2013; Smith & Bryson, 1994).

6   Extending upon original work that used descriptive measures (Bernier, Dawson,

7   Webb, & Murias, 2007; Rogers, et al., 1996; Vivanti, Nadig, Ozonoff, & Rogers, 2008),

8   kinematic analysis has been used to determine what, if any, aspects of movement form (e.g.,

9   velocity; timing of peak velocity) are imitated (Stewart, McIntosh, & Williams, 2013; Wild,

10   et al., 2012; Williams, Casey, Braadbaart, Culmer, & Mon-Williams, 2014). Specifically,

11   participants in the study of Wild et al. (2012) observed a human model performing an upper-

12   limb pointing movement that differed in speed, while context was manipulated so the model

13   aimed to targets (dots on a table), or to end space (dots removed). The notion is that, when

14   targets are removed from the environment, the imitator focuses their attention on imitating

15   the model's movement (kinematics; velocity) as opposed to merely reaching the target (dot)

16   goal. The imitation of the model's movement is thought to occur via direct lower-level

17   visuomotor mapping (Heyes, 2001; Southgate & Hamilton, 2008) and is suggested to be

18   compromised in autism (Edwards, 2014; Stewart, et al., 2013; Williams, et al., 2004). When

19   targets are present, the goal is to aim at a target (an action goal), which occurs via goal-

20   directed processes, and are less affected in autism (Hamilton, et al., 2007). The results from

21   Wild et al. (2012) showed only control participants imitated the different speeds when targets

22   were removed. The lack of scaling of movement speed exhibited in participants with autism

23   was accompanied by less time spent smoothly pursuing the hand (with the eyes) and thus

24   more shifts of gaze between the targets. It was suggested the shift in gaze, and thus attention

25   away from the hand, may have modulated the amount of action-based biological motion

1   information extracted from the model (Vivanti, et al., 2008), which thereby influenced the

2   imitation of movement speed.

3   Notwithstanding an attentional contribution, reduced imitation of kinematics in

4   individuals with autism has been linked to lower-level visuomotor processes (Stewart, et al.,

5   2013). For instance, imitation in a neurotypical control group was similar after observing a

6   human or non-human model, thus indicating that top-down processes associated with social

7   modulation (Cook & Bird, 2012; Spengler, Bird, & Brass, 2010; Wang & Hamilton, 2012)

8   did not exert an influence on behaviour. However, the autism group exhibited greater path

9   length error and action duration in both observation conditions, which was attributed to

10   impaired lower-level visuomotor processes that compromised self-other mapping in the

11   mirror system (Bernier, et al., 2007; Nishitani, Avikainen, & Hari, 2004; Williams et al.,

12   2006; Williams, et al., 2004; Williams, Whiten, Suddendorf, & Perrett, 2001). These lower-

13   level processes link action-observation to action-execution, and sub-serve imitation by

14   mapping observed biological motion onto the motor system (Buccino et al., 2004; Di Dio et

15   al., 2013; Iacoboni et al., 2001; Iacoboni et al., 1999).

16   Although previous work has provided novel contributions to understanding specific

17   imitation deficits in autism by isolating the contribution of lower-level processes (Stewart, et

18   al., 2013; Wild, et al., 2012), the examination of biological motion kinematics was

19   undertaken by manipulating only the speed or amplitude of the modelled movement and by

20   evaluating performance based on data from the whole imitation session. In terms of

21   biological kinematics, the aforementioned context requires an imitator to scale an existing

22   motor pattern (upper-limb movement) to meet new task demands (e.g., faster movement), but

23   does not isolate whether the deficit is attributable to imitating specific lower-level properties

24   (e.g., velocity) of biological motion kinematics. Furthermore, because 'true imitation' is an

25   active process whereby a novel representation is developed and refined over repeated

1   observations, it might be the case that important information about imitation adaptation is

2   masked by collapsing the analysis over all trials. An alternative approach that can reveal

3   more about adaptation is to evaluate performance in the early and late stages of imitation ,

4   which is a typical in observation learning studies (Bird & Heyes, 2005; Byrne & Russon,

5   1998; Hayes, Ashford, & Bennett, 2008). To this end, we further examined imitation of

6   biological motion in individuals with and without autism by employing a novel protocol that

7   required participants to imitate movements that had distinctly different, but still biologically

8   plausible, movement kinematics (Hayes, Roberts, Elliott, & Bennett, 2014). The experimental

9   models displayed movement that had exactly the same spatial and temporal outcomes as a

10   control model, but with a velocity profile of either typical or atypical kinematics. The atypical

11   model ensured that an observer had to configure the sensorimotor system to represent the

12   novel movement kinematics, as opposed to the typical model that could be achieved by

13   rescaling an existing representation of a typical upper-limb aiming movement (Hayes,

14   Timmis, & Bennett, 2009; Vivanti, et al., 2008). We controlled top-down influences by using

15   a protocol that minimized social attention (Cook & Bird, 2012; Wang & Hamilton, 2012) by

16   presenting a non-human agent model (white-dot) with limited social context. To control for

17   visual attention towards end-state target goal-directed features of the task environment

18   (Vivanti, et al., 2008; Wild, et al., 2012), we only displayed target goals in half of the

19   imitation trials in order to encourage attention towards the trajectory of the model. Finally, to

20   examine adaption we examined performance at the early and late phases of imitation.

21

22   **Method**

23

24   *Volunteers*

1   Fifteen typical control participants (14 male; 1 female) and 15 participants with autism (14

2   male; 1 female) volunteered for the study. The volunteers with autism were recruited from an

3   autistic society in North West England, and the University of Manchester, UK. The

4   volunteers were provided with a participant information sheet and selected if they consented

5   to be part of the study. The typical control participants were recruited from Liverpool John

6   Moores University, UK. All participants had normal or corrected-to-normal vision and were

7   screened via self-report for the following exclusion criteria: dyspraxia, dyslexia, epilepsy and

8   other neurological or psychiatric conditions. The participants with autism had a diagnosis of

9   autism, Asperger's syndrome or autism spectrum disorder by an independent clinician.

10   Diagnosis was confirmed by a researcher trained (with research-reliability status) in the

11   administration of module 4 of the Autism Diagnostic Observation Schedule 2 (ADOS-2)

12   (Lord et al., 2000). All participants with autism met the threshold for a diagnosis of autism

13   spectrum disorder on the ADOS-2 total classification score, and communication and

14   reciprocal social interaction subscales. Moreover, groups were equated for age, and using the

15   Wechsler Abbreviated Scale of Intelligence (WASI-II) (Wechsler, 1999), matched for full-

16   scale IQ, and the verbal and performance subscales. Sample characteristics for these

17   aforementioned control variables are presented in Table 1. The experiment was designed and

18   conducted in accordance with the 1964 Declaration of Helsinki and approved by the research

19   ethics committee of Liverpool John Moores University, UK.

20

21   *Procedure*

22   Participants were sat so their eyes were located approximately 555 mm from the centre of a

23   21-inch CRT monitor (Iiyama Vision Master 505), operating with a resolution of 1280 x

24   1024 pixels and a refresh rate of 85 Hz. Connected to the monitor was a desktop PC (Dell

25   Optiplex GX280), graphics tablet and a hand-held stylus (Wacom Intuos Pro XL) (Fig. 1a).

1   In-house routines programmed in MATLAB (The Mathworks, Inc.) controlled the

2   experiment, and the visual stimulus, which was generated using the Cogent 2000 toolbox

3   (www.vislabucl.ac.uk/cogent.php).

4   Participants were provided with general instructions to "watch and then copy the

5   movement displayed by a white dot on the computer monitor". The model (i.e., a non-human

6   agent model) displayed a single horizontal trajectory that originated from a home-target

7   (diameter = 12.50 mm) positioned on the left-hand side of the screen and ended at a right-

8   hand end-target (diameter = 12.50 mm), or right-hand end-space (i.e., no target; see below).

9   The amplitude of the movement was 200 mm, and the total movement time was 1700 ms. To

10 examine the imitation of biological motion, three non-human agent models were created that

11 displayed typical, atypical or constant velocity profiles. The kinematics of the typical model

12 trajectory were of biological origin as they were created by a human volunteer. To do so, the

13 volunteer practised the task by performing typical goal-directed aiming movements using a

14 hand-held stylus on a graphics tablet until a white dot/cursor (diameter = 6.25 mm), which

15 represented the stylus, moved from the left-hand home-target to the right-hand end-target in

16 1700 ms. The model displayed a typical (Flash & Hogan, 1985) bell-shaped velocity profile

17 (displacement time-series is displayed as the dark grey trace in Fig. 1b) that had a magnitude

18 of peak velocity that was 0.20 mm/ms and a peak that occurred at 44% of the movement

19 time. The atypical model (black trace in Fig. 1b) was created by the same volunteer, and thus

20 ensured the kinematics of the movement were both biological and achievable by human

21 participants. The volunteer practised performing an atypical movement until the 200 mm

22 amplitude was completed in 1700 ms. The atypical model had a magnitude of peak velocity

23 that was 0.41 mm/ms that occurred at 18% of the movement time. The model displaying

24 constant velocity was created according to the amplitude (200 mm) and time (1700 ms)

25 constraints associated with the task. The model displayed the exact movement time and

1   moved at constant velocity in the horizontal axis (0.118 mm/ms), with no deviations in the

2   perpendicular axis (see Fig. 1b).

3   The imitation task comprised 14 blocks of 6 trials (84 trials). A block contained the

4   typical, atypical and constant velocity models, each performed in the target and no-target

5   conditions. Trial order within a block, as well as block order, was fully randomised across

6   participants. The randomised structure reduced predictability of an upcoming model(s) and

7   thus promoted imitation on a trial-by-trial basis. Prior to the experimental trials, all

8   participants completed a familiarisation period that replicated the conditions of the imitation

9   task. Participants performed four familiarisation trials, 2 trials representing a target condition,

10   and 2 trials representing a no-target condition. Each trial commenced with the model being

11   positioned in the centre of the home target at the left-hand side of the display after which it

12   moved to the end-target (target condition), or end-space (no-target condition), with a constant

13   velocity in a time of 1700 ms. A constant velocity trajectory was used to ensure construct

14   validity by preventing participants from experiencing biological motion before the actual

15   imitation trials. Participants were not informed about the time duration of the movement, the

16   different types of stimulus, or the end-state target manipulation. Therefore, after observing a

17   model, participants were only provided with a general instruction to copy the model (not a

18   specific instruction to copy a certain aspect of the model; e.g., the kinematics) by moving the

19   stylus on the tablet so that the cursor moved to the end-target, or end-space, as per the

20   movement of the model. All participants confirmed they understood the model, the target and

21   no-target conditions, the instruction to imitate a model, and the sensorimotor association

22   between the stylus on a graphics tablet and the corresponding movement of cursor on the

23   monitor.

24

25   Insert Fig. 1 here

1

2   *Data Reduction and Analysis*

3   To quantify imitation of motor timing, we extracted movement time from each trial and

4   calculated an accuracy score (*motor timing accuracy*), which reflected the signed (+ or -)

5   difference between a participant's movement time and that of the model (e.g., 1900 ms –

6   1700 ms = 200 ms). To examine motor timing consistency, we calculated a variability score

7   (*motor timing variability*) that represented the within-participant distribution of movement

8   time within a condition (i.e., standard deviation).

9   To quantify imitation of movement kinematics we focused the analysis on x-axis data

10   because, much like the constant velocity model that had zero deviation in the y-axis, the

11   perpendicular deviation in the y-axis for the atypical model and typical model was minimal.

12   This was confirmed prior to our analysis by calculating perpendicular deviation using root-

13   mean-square-error (RMSE) with respect to a value of zero (i.e., no deviation). Indeed, we

14   observed the atypical model RMSE was 0.9 mm and typical model RMSE was 1.5 mm.

15   Therefore, and similar to our previous work (Hayes, Andrew, Elliott, Roberts, & Bennett,

16   2012; Hayes, Elliott, & Bennett, 2010, 2013; Hayes, et al., 2014), we felt it was appropriate

17   to conduct an analysis of dependent measures extracted from the primary movement (x-axis)

18   only. Given such minimal deviation in the y-axis, any displacement in this axis by the

19   participant would have most likely been an incidental result of anatomical constraints rather

20   than intentional imitation. To complete the analysis, we identified within the x-axis position

21   data the start and end of the movement. The start was defined as the moment the centre of the

22   cursor moved beyond the perimeter of the 'home' target, and end equated to the moment the

23   participant clicked the lower-button on the stylus. For each imitation trial, the resulting

24   position data were filtered using a low pass 4th order autoregressive filter with an 8 Hz cut-

25   off. The filtered data were next differentiated using a central difference algorithm to obtain

1 velocity. A MATLAB routine then extracted *peak velocity* and *percentage-time-to-peak-*

2 *velocity* from each trial.

3 For both timing and kinematic dependent variables, intra-participant means were

4 calculated from the first six and last six trials performed following observation of the 3 non-

5 human agent models in the 2 target conditions. These data were submitted to separate 2

6 Group (autism; control) x 3 Model (atypical; typical; constant velocity) x 2 Target (target; no-

7 target) x 2 Phase (early; late) repeated measures ANOVA. Significant main and/or

8 interactions effects involving more than two means were analysed using Tukey HSD post-hoc

9 procedure and alpha was set at $p < 0.05$. Additional correlation analysis on relevant

10 significant comparisons indicated by ANOVA were then completed to assess whether the

11 dependent measure correlated with autism severity (i.e., ADOS total score).

12

13 **Results**

14

15 The analysis of *motor timing accuracy* indicated a main effect of model [$F(2, 56) = 51.267$, p

16 < 0.01, $\eta_p^2 = 0.647$]. Timing was more accurate after imitating atypical compared to typical (p

17 $= 0.024$; difference $= 67$ ms) and constant ($p < 0.01$; difference $= 285$ ms) velocity models,

18 and after imitating the typical compared to constant ($p < 0.01$; difference $= 218$ ms) velocity

19 model. As illustrated in Fig. 2a, a 2-way interaction involving group x phase [$F(1, 28) =$

20 9.480, $p < 0.01$, $\eta_p^2 = 0.253$] showed *motor timing accuracy* increased by 175 ms from the

21 early to late phase for the autism group ($p < 0.05$), whereas the performance of the control

22 group deteriorated by 139 ms over the training period ($p < 0.05$). Correlation analysis

23 revealed no relationship between *motor timing accuracy* in the early phase and ADOS total

24 score (Pearson's r(15) $= 0.12$, $p > 0.05$) or late phase and ADOS total score (Pearson's r(15)

25 $= -0.02$, $p > 0.05$).

1   The analysis of *motor timing variability* indicated a main effect of model [F(2, 56) =

2   4.679, $p = 0.01$, $\eta_p^2 = 0.143$], group [F(1, 28) = 11.610, $p = 0.01$, $\eta_p^2 = 0.293$], and a 2-way

3   interaction involving group x phase [F(1, 28) = 4.770, $p < 0.05$, $\eta_p^2 = 0.146$]. As illustrated in

4   Fig. 2b, motor timing was less variable after imitating the typical compared to constant ($p <$

5   0.01; difference 55 ms) velocity model, and when imitating atypical compared to the constant

6   ($p = 0.01$; difference 51 ms) velocity model. Although the main effect indicated the control

7   group (M = 240 ms) was significantly ($p < 0.05$) less variable overall than the autism group

8   (363 ms), the interaction showed that only the autism group significantly ($p < 0.05$) decreased

9   *motor timing variability* (by 99 ms) from the early to late phase of practice. Correlation

10   analysis revealed no relationship between *motor timing variability* in the early phase and

11   ADOS total score (Pearson's r(15) = -0.25, $p > 0.05$) or late phase and ADOS total score

12   (Pearson's r(15) = -0.33, $p > 0.05$).

13

14   Insert Fig. 2 here

15

16   The analysis of *peak velocity* indicated a main effect of model [F(2, 56) = 74.405, $p <$

17   0.01, $\eta_p^2 = 0.727$] and a 2-way interaction involving group x phase [F(1, 28) = 5.000, $p < 0.$

18   05, $\eta_p^2 = 0.152$]. As illustrated in Fig. 3a, *peak velocity* was greater when imitating atypical

19   (0.238 mm/ms) compared to typical (0.192 mm/ms) and constant (0.162 mm/ms) velocity

20   models ($ps < 0.01$). The interaction indicated *peak velocity* increased by 0.024 mm/ms from

21   the early to late phase for the autism group ($p < 0.05$), whereas the decrease of 0.009 mm/ms

22   for the control group was not significant. Correlation analysis revealed no relationship

23   between *peak velocity* in the early phase and ADOS total score (Pearson's r(15) = 0.03, $p >$

24   0.05) or late phase and ADOS total score (Pearson's r(15) = -0.06, $p > 0.05$).

25

1   Insert Fig. 3 about here

2

3   The analysis of *percentage-time-to-peak-velocity* showed a main effect of model [F(2,

4   56) = 41.536, $p < 0.01$, $\eta_p^2 = 0.597$] and an interaction involving group x model [F(2, 56) =

5   8.569, $p < 0.01$, $\eta_p^2 = 0.234$]. As illustrated in Fig. 3b, although the groups did not differ when

6   imitating the typical and constant velocity models ($ps > 0.05$), there was a significant

7   difference when imitating the atypical velocity model ($p < 0.01$). The control group exhibited

8   a *percentage-time-to-peak-velocity* that occurred significantly ($p < 0.01$) earlier in the

9   movement (24 %), which was more similar to the atypical criterion model (18%: dashed line

10   on Fig. 1b), than the autism group (33 %). These effects can be seen in the exemplar velocity

11   traces illustrated in Fig. 4. When imitating the atypical model (back trace Fig. 4a), peak

12   velocity occurred significantly earlier in the movement for the control group (dark grey

13   trace), than the autism group (light grey trace). Whereas peak velocity occurred toward the

14   midpoint of the movement for both groups (autism = light grey trace; control = dark grey

15   trace) when imitating the typical (Fig. 4b) and constant (Fig. 4c) velocity models. Correlation

16   analysis revealed no relationship between *percentage-time-to-peak-velocity* for atypical

17   model and ADOS total score (Pearson's r(15) = 0.22, $p > 0.05$) or typical model and ADOS

18   total score (Pearson's r(15) = 0.05, $p > 0.05$).

19

20   Insert Fig. 4 about here

21

22   **Discussion**

23

24   We examined imitation, and imitation adaption (i.e., performance change from the early to

25   late phase on imitation), of biological motion kinematics using a novel behavioural protocol

1   that required adults with and without autism to observe a model that displayed distinctly

2   different but biologically plausible kinematics. Importantly, the atypical biological motion

3   would not have been represented in the sensorimotor repertoire of observers, and thus could

4   not be imitated by rescaling a typical upper-limb aiming movement. After observing an

5   atypical model, participants in the control group exhibited movements with a *percentage-*

6   *time-to-peak-velocity* that occurred at 24% of the movement trajectory. This early occurrence

7   of peak velocity was similar to that displayed by the atypical model (*percentage-time-to-*

8   *peak-velocity* = 18%), and significantly different to the *percentage-time-to-peak-velocity*

9   exhibited after observing typical (M = 34%) and constant (M = 39%) velocity control models.

10   The presence of temporal correspondence between control participants' movements and the

11   atypical model indicates high fidelity imitation of biological motion kinematics based on

12   lower-level sensorimotor processes (Brass, Bekkering, & Prinz, 2001; Gangitano, Mottaghy,

13   & Pascual-Leone, 2001; Hayes, et al., 2014; Heyes, 2001).

14   Equivalent high fidelity imitation of biological motion kinematics was not found for

15   adults with autism. Although the magnitude of *peak velocity* was similar to control adults,

16   there was a lack of temporal correspondence to the atypical model. The kinematic data

17   showed *percentage-time-to-peak-velocity* occurred at 33% of the movement trajectory, which

18   was significantly different from the control group, but statistically similar to the *percentage-*

19   *time-to-peak-velocity* exhibited when imitating the typical (M = 38%) and constant velocity

20   (M = 39%) control models. In this respect, our data are consistent with other work that

21   demonstrated differences between those with and without autism in imitating the style (e.g., a

22   gentle or harsh hand action) of a movement (Hobson & Lee, 1999; Rogers, et al., 1996; Smith

23   & Bryson, 1994) or movement speed (Stewart, et al., 2013; Wild, et al., 2012). Importantly,

24   however, the present findings extend understanding by showing differences in imitation are

1 directly related to attenuation in representing the temporal occurrence of peak velocity

2 associated with the observed biological motion kinematics.

3   Before interpreting this effect, it is important to highlight that we isolated the

4 examination of biological kinematics using a protocol that controlled higher order factors

5 known to constrain imitation. First, we displayed an atypical model to ensure imitation was

6 associated with representing novel biological kinematics, as opposed to presenting a

7 movement that could be imitated using a pre-existing motor pattern recalled via higher-order

8 semantic (Rumiati et al., 2005) or action-goal (Bekkering, Wohlschlaeger, & Gattis, 2000;

9 Southgate & Hamilton, 2008) processes. Second, because imitation is modulated by social

10 top-down factors (Chartrand & Bargh, 1999; Cook & Bird, 2012; Spengler, et al., 2010;

11 Wang & Hamilton, 2012), we used a non-human agent model that reduced the influence of

12 emotional (Grèzes, Wicker, Berthoz, & de Gelder, 2009) and/or theory of mind (Baron-

13 Cohen et al., 1999) constraints that are inherent in realistic human models. Third, we

14 controlled the influence of end-state target goal attainment (Bekkering, et al., 2000) by

15 displaying a movement trajectory that had no targets in half of the trials. In combination, our

16 use of these control measures minimizes the likelihood that the deficit in imitating biological

17 motion kinematics in adults with autism is attributable to higher-order processes associated

18 with reaching a target, or social imitation.

19   One explanation for the attenuation in imitating biological motion kinematics could be

20 associated with lower-level processes that integrate visuomotor information (Dapretto et al.,

21 2006; Oberman et al., 2005; Stewart, et al., 2013; Théoret et al., 2005; Williams, et al., 2006;

22 Williams, et al., 2004). For example, visuomotor integration of biological motion occurs

23 through specialised visual areas (posterior superior temporal sulcus; (Grossman, Battelli, &

24 Pascual-Leone, 2005; Grossman et al., 2000) and lower-level sensorimotor processes linked

25 to the mirror system (Iacoboni, 2005; Southgate & Hamilton, 2008). These processes are part

1   of a functional network that represents an observed movement by mapping the biological

2   motion characteristics directly onto the motor system (Iacoboni, et al., 1999; Rizzolatti &

3   Craighero, 2004). However, while lower-level processing deficits associated with visuomotor

4   integration during self-other mapping (Stewart, et al., 2013; Williams, et al., 2006; Williams,

5   et al., 2004) could attenuate imitation of atypical biological kinematics, it is notable that

6   adults with autism show intact mapping of biological motion during automatic imitation

7   (Bird, et al., 2007), which is a behavioural protocol that isolates processing to the lower-level

8   mirror system. Moreover, results from neuropsychological work is mixed on whether such a

9   fundamental impairment is present in autism (Hamilton, 2013).

10   Our data revealed an intriguing adaptation effect whereby adults with autism became

11   significantly more accurate at representing movement time, reducing movement time

12   variability, and increasing the magnitude of peak velocity over trials during imitation. This

13   adaptation must have been self-regulated, as opposed to augmented, because external

14   feedback regarding movement time performance was not provided. This change in behaviour

15   can be ascribed to active and functional true imitation, with sensorimotor adaptation most

16   likely a result of attending to, and comparing against, the observed stimulus using

17   feedforward and feedback processes (Byrne & Russon, 1998; Carroll & Bandura, 1982;

18   Kilner, Friston, & Frith, 2007). Moreover, within the group of high-functioning autism

19   participants recruited in the current study, it would seem this adaptation is a general process

20   that is not related to autism severity as determined by correlations with ADOS total score. In

21   addition to modulating the magnitude of peak velocity, the positive change in accuracy for

22   movement timing also reduced the influence of end-state-target-goals such that timing and

23   kinematics changed similarly for target and no-target conditions. Moreover, we also found no

24   evidence that the adult control group prioritised the attainment of an end-state-target-goal,

25   over the imitation of atypical biological kinematics, when present during observation.

1   Although goal-directed imitation effects have been reported in complex movement sequences

2   (Wild, Poliakoff, Jerrison, & Gowen, 2010; Wild, et al., 2012) or a full body point-light

3   model (Hayes, Hodges, Huys, & Williams, 2007), it seems the target was less constraining

4   when individuals observed a point-light non-human agent model performing a single segment

5   movement.

6   The fact that adults with autism became significantly more accurate at imitating

7   movement time, and exhibited a magnitude of peak velocity that was similar to the control

8   group, suggests visual attention was orientated to the information displayed by the non-

9   human agent model. This effect is consistent with data showing visual attention to action

10   features of a model (Vivanti, et al., 2008), and non-human stimuli (Swettenham et al., 1998),

11   is typical in autism, whereas attention to facial features differs from controls (Bird, Catmur,

12   Silani, Frith, & Frith, 2006; Boucher & Lewis, 1992; Vivanti, et al., 2008). Moreover,

13   because no other attention-distracting stimuli were present in our display, it is unlikely that

14   reduced imitation of atypical biological kinematics was associated with visual attention being

15   drawn away from the non-human agent model (Wild, et al., 2012). A more parsimonious

16   explanation is that the selective attention bias to movement time during imitation was

17   controlled via alternative (and efficient) higher-order processes (Hamilton, et al., 2007;

18   Southgate & Hamilton, 2008; Wild, et al., 2012). A possibility is the movement time goal

19   was imitated using processes associated with action comprehension, which are functional in

20   autism (Dinstein et al., 2010), and as such goal attainment was secured using an efficient pre-

21   existing motor pattern. This interpretation is consistent with our kinematic data, which

22   showed individuals with autism executed movements that exhibited typical [peak velocity

23   occurred towards the mid-point of the trajectory (Elliott et al., 2010)] motor control

24   trajectories when imitating both the atypical and typical models.

1   In addition to a goal-directed and action comprehension interpretation, the selective

2   attention bias to movement time may have modulated input to the lower-level mirror system.

3   Input modulation is suggested to impact the activation, or development, of sensorimotor

4   representations via the intentionally mediated orientation of visual attention (Heyes, 2011;

5   Heyes & Bird, 2007; Liepelt & Brass, 2010; Longo, Kosobud, & Bertenthal, 2008).

6   Therefore, because we did not specify within our task instructions what aspect of the model

7   to imitate, the self-selected focus on movement time may have regulated the lower-level

8   processes such that this temporal variable was placed higher on the embedded hierarchy of

9   imitation goals (Hamilton & Grafton, 2007; Hayes, et al., 2014; Wohlschlager, Gattis, &

10   Bekkering, 2003) than atypical kinematics. Although it is unclear if such input modulation is

11   operational in autism (Vivanti & Hamilton, 2014), we have differentially modulated how

12   atypical biological kinematics and movement time is imitated in neurotypical volunteers

13   using pre-specified verbal instructions (Hayes, et al., 2014). For example, the imitation of

14   atypical biological kinematics can be modulated if volunteers are instructed to focus attention

15   on imitating the movement time goal. Likewise, imitation accuracy can be enhanced if

16   selective attention is directed to the kinematics. Therefore, we cannot say for certain if the

17   focus on motor timing in individuals with autism is causally related to deficits in lower-level

18   self-other mapping processes and/or motor ability, or whether the attentional effect is a

19   compensatory strategy. One way to determine if the attenuation in imitating atypical

20   biological kinematics is associated with top-down attentional modulation is to present a

21   similar non-human agent model and employ a selective attention protocol that uses explicit

22   instructions to guide observers to attend and imitate the atypical biological kinematics

23   (Stewart, et al., 2013), as opposed to the observers self-selecting which action-based

24   information to imitate.

1   When considering the findings in respect to the broader context of imitation in autism,

2   it is important to highlight we designed our study to examine 'true imitation'. True imitation

3   is a fundamental developmental process as it underpins the acquisition of novel social, and

4   important sensorimotor skills that facilitate everyday life such as, tying shoe laces, riding a

5   bicycle, or playing ice hockey. Although our data showed an attenuation in the imitation of

6   biological motion kinematics, we did find that movement time accuracy and variability was

7   significantly improved. The implication is that sensorimotor adaption and representation

8   (Gidley Larson, Bastian, Donchin, Shadmehr, & Mostofsky, 2008) of movement time is

9   intact in high-functioning adults with autism. These are first data to show this adaptation in a

10   'true imitation' context and indicates adults with autism do imitate, but they seem to do so in

11   their own way. Therefore, the challenge is to examine the possibility that adults with autism

12   can learn to imitate and represent biological motion kinematics following specific

13   manipulations to the learning context (e.g., practice type, instructions, feedback). If the

14   results are positive, then social and environmental procedures can be implemented by

15   clinicians and practitioners to facilitate the acquisition of social and sensorimotor behaviors

16   in autism.

17   In summary, the data presented here demonstrate, for the first time experimentally,

18   that adults with autism have difficulties imitating the velocity characteristics associated with

19   atypical biological motion kinematics. Compared to control participants, adults with autism

20   became significantly more accurate at imitating movement time across trials. The positive

21   change in behaviour confirmed they actively engaged in the task, and that sensorimotor

22   adaptation during imitation is self-regulated in autism. The bias to movement time suggests

23   the attenuation in imitating biological motion kinematics in autism is perhaps a compensatory

24   strategy due to deficits in lower-level visuomotor processes associated with self-other

1   mapping and/or motor ability, or that selective attention input to the processes that represent

2   atypical biological motion kinematics.

3

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1   **Figure Captions**

2

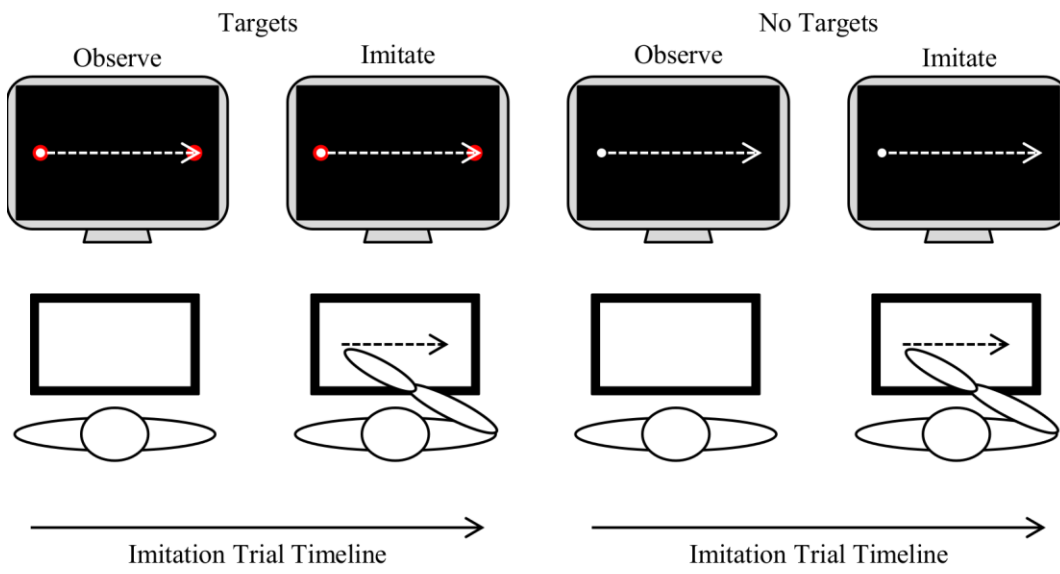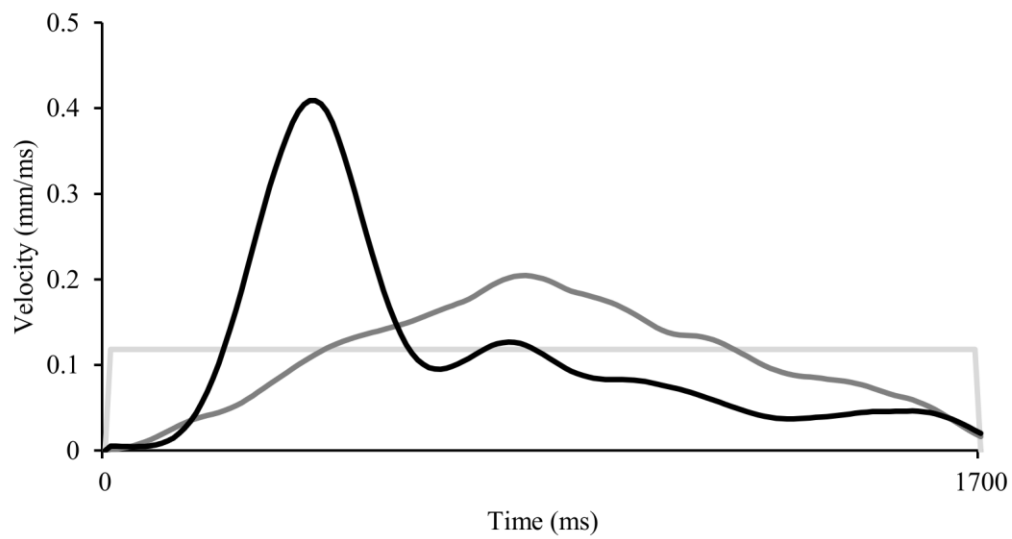3   *Figure 1(a)* A schematic representation of the laboratory/experimental set-up for the

4   imitation task. The black outlined rectangle represents a graphics tablet. The white circle

5   displayed on the CRT monitor represents the model. The single-segment movement is

6   depicted by the arrow (i.e., from the start position to the final position) in the target (red

7   target) and no-target conditions. *(b)* Typical (dark-grey trace), atypical (black trace) and

8   constant (light-grey trace) velocity models presented as a function of movement time.

9

10   *Figure 2 (a)* Timing accuracy and *(b)* Timing variability for the imitation task (error bars

11   represent standard error of the mean) presented as a function of Group, Model and Phase.

12

13   *Figure 3 (a)* Peak velocity and *(b)* percentage-time-to-peak-velocity for the imitation task

14   (error bars represent standard error of the mean) presented as a function of Group, Model and

15   Phase. The dashed lines in Fig. 3a represent the magnitude of peak velocity for the typical

16   (i.e., 0.41 mm/ms) and atypical (i.e., 0.20 mm/ms) models (Fig. 3a). In Fig. 3b, they represent

17   the percentage-time-to-peak-velocity for the typical (i.e., 44%) and atypical (i.e., 18%)
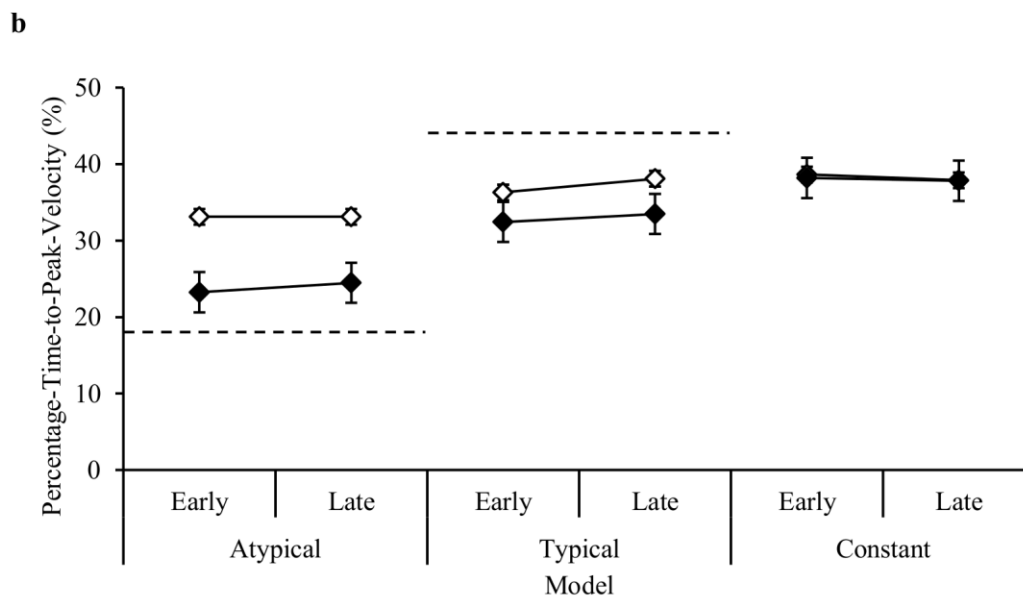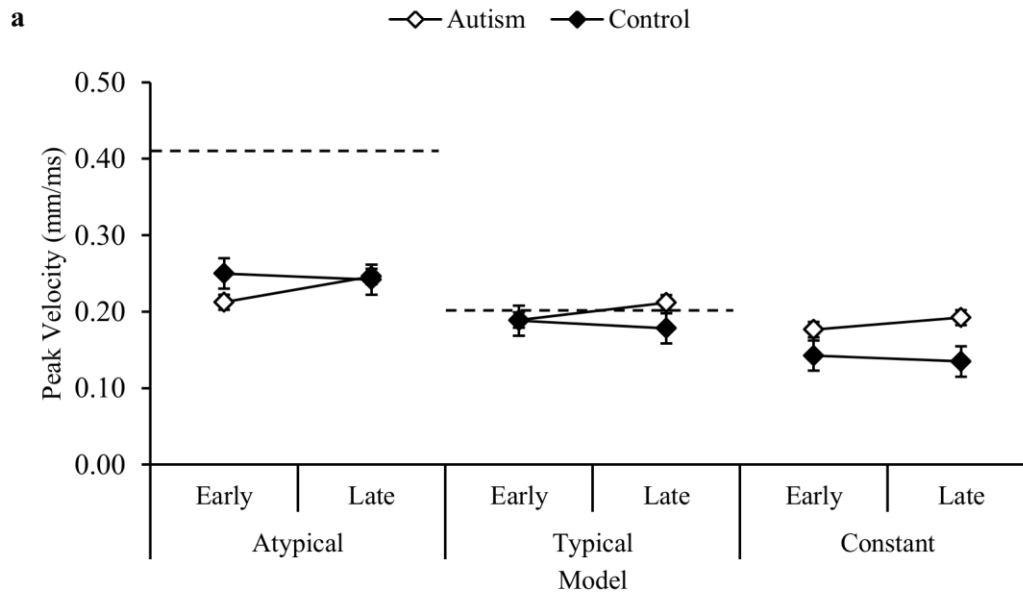
18   models.

19

20   *Figure 4* The black velocity traces represent the atypical (a), typical (b), and constant (c)

21   velocity models. In each panel (a, b, c), the light grey (autism) and dark grey (control)

22   velocity traces display exemplar data from a representative participant imitating each model.
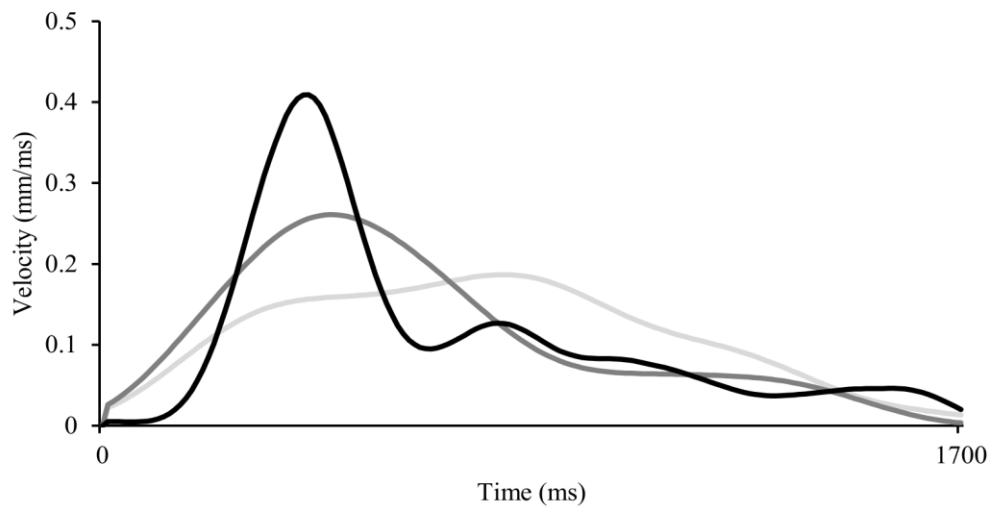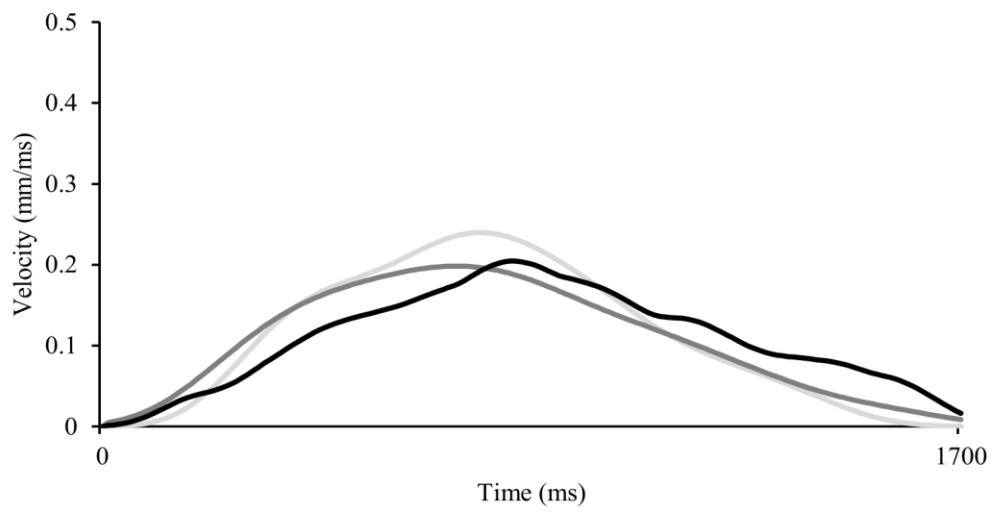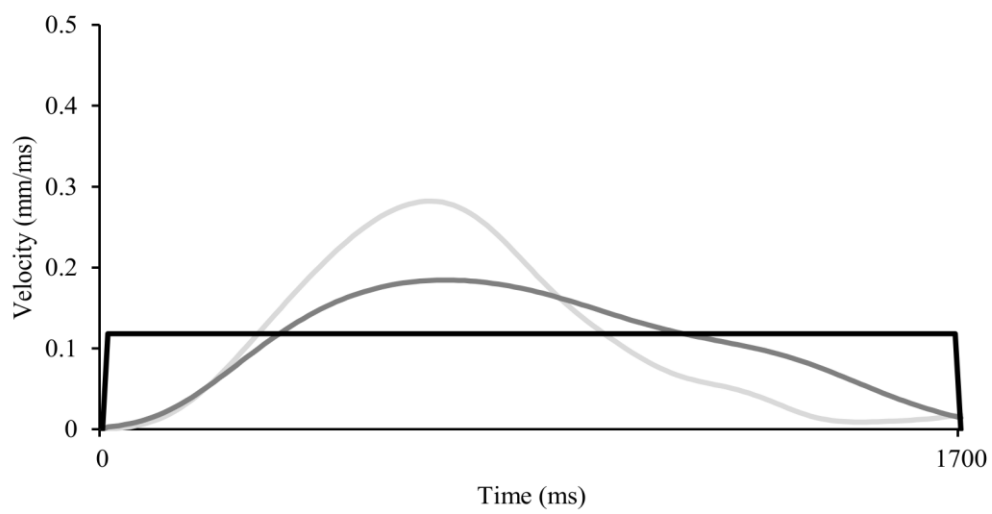
23

a

b

a

b

1

a

b

1

1   *Table 1* Characteristics of autism and neurotypical control participants

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

2