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1 **Facilitating sensorimotor integration via blocked practice underpins imitation learning**
2 **of atypical biological kinematics in autism spectrum disorder**

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4 Nathan C. Foster^{1,2}, Simon J. Bennett², Joe Causer², Digby Elliott³, Geoffrey Bird^{4, 5}, and
5 Spencer J. Hayes^{6*}

6
7 ¹Cognition, Motion and Neuroscience Unit, Fondazione Istituto Italiano di Tecnologia,
8 Genova, Italy

9
10 ²Research Institute for Sport and Exercise Sciences,
11 Liverpool John Moores University, Liverpool, UK

12
13 ³Department of Kinesiology,
14 McMaster University, Ontario, Canada

15
16 ⁴Department of Experimental Psychology, University of Oxford,
17 Oxford, UK

18
19 ⁵Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology
20 and Neuroscience, King's College London, London, UK

21
22 ⁶Department of Psychology and Human Development, Institute of Education, University
23 College London, UK

24
25 *Corresponding author

26 ⁶Department of Psychology and Human Development, Institute of Education,
27 University College London, London, UK

28 spencer.hayes@ucl.ac.uk

Abstract

The reduced efficacy of voluntary imitation in autism is suggested to be underpinned by differences in sensorimotor processing. We examined whether the imitation of novel atypical biological kinematics by autistic adults is enhanced by imitating a model in a predictable blocked practice trial order. This practice structure is expected to facilitate trial-to-trial sensorimotor processing, integration and encoding of biological kinematics. The results showed that neurotypical participants were generally more effective at imitating the biological kinematics across all experimental phases. Importantly, and compared to a pre-test where imitation was performed in a randomised (unpredictable) trial order, the autistic participants learned to imitate the atypical kinematics more effectively following an acquisition phase of repeatedly imitating the same model during blocked practice. Data from the post-test showed that autistic participants remained effective at imitating the atypical biological kinematics when the models were subsequently presented in a randomised trial order. These findings show that the reduced efficacy of voluntary imitation in autism can be enhanced during learning by facilitating trial-to-trial processing and integration of sensorimotor information using blocked practice.

Key words: autism spectrum disorder, imitation, biological motion kinematics, sensorimotor integration, blocked practice

1 Introduction

2 Learning novel actions through voluntary imitation is a fundamental part of human
3 development, and is facilitated by intentional, attentional and sensorimotor processes.
4 During voluntary imitation (henceforth imitation), an individual observes a model that
5 typically prescribes a higher-order action-goal (e.g., to use chop sticks), as well as the lower-
6 level kinematic properties (e.g., velocity of the digits) constraining the means of achieving
7 the action-goal. In the action-observation phase of imitation, an action-goal and lower-level
8 kinematics are encoded within a sensorimotor system linking perception to action (Prinz,
9 1997). After observation, processes associated with sensorimotor planning are engaged to
10 control the specification of forces required for initial execution of the to-be-imitated
11 movement pattern. During, and after, movement execution, efferent and afferent
12 sensorimotor information is integrated and processed (by feedforward and feedback control
13 mechanisms) to support encoding (Wolpert, Diedrichsen & Flanagan, 2011). Over repeated
14 imitation trials, an action-representation is developed and refined so that an imitated
15 movement becomes similar to the observed biological motion characteristics displayed by
16 the model. While the process of imitation is operational across typical development
17 (Anisfeld, 2005; Jones, 2009; Ray & Heyes, 2011), it has been claimed that autistic
18 individuals show difficulty imitating the lower-level biological kinematic properties of an
19 observed action (DeMyer et al., 1972; Hayes, Andrew, Elliott, Gowen, & Bennett, 2016;
20 Hobson & Lee, 1999; Rogers, Bennetto, McEvoy, & Pennington, 1996; Stewart, McIntosh, &
21 Williams, 2013; Wild, Poliakoff, Jerrison, & Gowen, 2012).

22 In a previous examination of the imitation of biological kinematics in autism, we
23 randomly presented two models that displayed the same movement amplitude and
24 movement time goal (1700 ms), but different underlying kinematics (Hayes, Andrew, et al.,
25 2016). A control model had typical kinematics with a bell-shaped velocity profile (peak
26 velocity that occurred at ~50% of the movement trajectory). This model could be imitated by
27 rescaling a typical movement profile from an existing motor repertoire (Carmo, Rumiati,
28 Siugzdaite, & Brambilla, 2013; Rumiati et al., 2005). As predicted, we showed no difference

1 between the autistic and neurotypical groups imitation of movement kinematics displayed in
2 the control model. An experimental model had a novel atypical kinematic profile, where peak
3 velocity occurred at 18% of the movement trajectory. This model required participants to
4 represent the atypical kinematics during action-observation in order to reorganise the
5 sensorimotor system to plan and execute the appropriate motor response during imitation.
6 Unlike the neurotypical group that successfully imitated the atypical kinematics (Hayes,
7 Andrew, et al., 2016), the autistic group produced a movement more similar to the
8 kinematics of the typical model. Still, despite failing to imitate the novel atypical kinematics,
9 the autism group became significantly more accurate and consistent at imitating the 1700
10 ms movement time goal. This specific adaptation effect associated with learning the overall
11 movement time goal indicates the autistic participants were actively engaged in imitation
12 learning and therefore likely to have followed the task instructions to “watch and then copy
13 the movement displayed by a white dot on the computer monitor”. Accordingly, we can infer
14 that attention (to the stimuli) and intention (to produce the observed action), which both
15 modulate voluntary imitation (Hayes et al., 2014), were not factors that limited imitation of
16 the movement time goal.

17 Further insight into the operation of sensorimotor processes in autism is evident from
18 automatic imitation studies (Bird, Leighton, Press, & Heyes, 2007; Edey et al., 2016;
19 Hamilton, Brindley, & Frith, 2007; Press, Richardson, & Bird, 2010; Schulte-Rüther et al.,
20 2017; Sowden, Koehne, Catmur, Dziobek, & Bird, 2016; Spengler, Bird, & Brass, 2010) in
21 which autistic adults have been shown to generate sensorimotor response times similar to
22 matched-controls when observing task irrelevant biological action stimulus (e.g., a human
23 hand lifting an index finger). In other words, movement observation had a direct automatic
24 influence on motor execution (Brass, Bekkering, & Prinz, 2001), thereby confirming the
25 sensorimotor processes responsible for processing biological motion during action-
26 observation are operational in autism (Nackaerts et al., 2012; Saygin, Cook, & Blakemore,
27 2010). The implication for voluntary imitation is that the difficulty imitating atypical biological
28 kinematics is not solely associated with a specific imitation mechanism that directly

1 represents and encodes biological motion during the action-observation phase (Bernier,
2 Dawson, Webb, & Murias, 2007; Williams, Whiten, & Singh, 2004; Williams, Whiten,
3 Suddendorf, & Perrett, 2001). Rather, there may be differences in how other general
4 sensorimotor learning processes (Chetcuti, Hudry, Grant, & Vivanti, 2019; Hamilton, 2013;
5 Leighton, Bird, Charman, & Heyes, 2008) are engaged to represent and refine the observed
6 and executed biological kinematics during repeated imitation trials.

7 For example, by presenting the typical and atypical kinematic models in a
8 randomised trial order (Hayes, Andrew, et al., 2016), sensorimotor information from trial n
9 (e.g., atypical model) would often be different to trial $n+1$ (e.g., typical model). Therefore,
10 executing different sensorimotor actions would have led to 'intratask interference' (Battig,
11 1972). In a motor learning context, this form of interference is called the contextual
12 interference effect, which is defined 'as the effect on learning of the degree of functional
13 interference found in a practice situation when several tasks must be learned and practiced
14 together' (Magill & Hall, 1990, p. 244). Although practising multiple task variations of a
15 sensorimotor action engages processes that facilitate long-term retention and transfer of the
16 action (Brady, 1998; Edwards, Elliott, & Lee, 1986; Magill & Hall, 1990; Shea & Morgan,
17 1979), motor performance during the practice period is often attenuated (i.e., decrease in
18 accuracy; increase in variability). Attenuation occurs because intratask interference affects
19 the efficacy of integrating, and consolidating, different sensorimotor information sources
20 across trials because the expected (efference) and actual (reafferent) sensorimotor
21 consequences are different (Immink & Wright, 2001). In addition to a contribution from
22 processes underlying integration and consolidation, performance may have been affected
23 because imitating the typical and atypical models in a random trial order would have
24 engaged greater attention-demanding and effortful motor planning processes (Li & Wright,
25 2000), which are already known to be compromised in autism (Glazebrook, Elliott, & Lyons,
26 2006; Rinehart, Bradshaw, Brereton, & Tonge, 2001).

27 Therefore to further examine sensorimotor planning and integration processes in
28 voluntary imitation in autism, we investigated imitation learning (pre-test, acquisition-phase,

1 and post-test) of a novel motor behaviour using a blocked practice protocol. Compared to
2 random practice (Hayes, Andrew, et al., 2016), the acquisition-phase was arranged such
3 that the same atypical model is presented consecutively across all practice trials. We
4 expected imitation performance to be facilitated because functional task difficulty is lower
5 during blocked practice (i.e., see 'Challenge Point' framework; Guadagnoli & Lee, 2004),
6 with only one sensorimotor action plan (i.e., the atypical movement) being represented
7 across all trials. In addition to lower task difficulty, the blocked trial order should optimise
8 (Magill & Hall, 1990; Immink & Wright, 2001; Kantak & Winstein, 2012) the comparison and
9 processing of expected (efference copy - feedforward control) and actual (reafference -
10 feedback control) sensorimotor consequences from trial n to trial $n+1$ (Elliott et al., 2001;
11 Wolpert et al., 2011). Over repeated trials, an internal action model is expected to be refined
12 and encoded so that the movement imitated by an observer becomes similar to the atypical
13 biological kinematics displayed by the model.

14 In the present study, autistic and neurotypical participants completed a learning
15 protocol where they imitated two models that displayed either atypical or typical biological
16 kinematics. Because we were principally interested in the effects of practice structure on
17 imitation performance, we quantified attention (Wild et al., 2012) by recording the eye
18 movements of participants to ensure that overt visual attention was located on the model
19 during the action observation phase of imitation. Specifically, in a pre-test and post-test, both
20 models were presented in a randomised trial order such that imitation context was
21 unpredictable. Following the pre-test, participants performed an acquisition phase where
22 they imitated the atypical and typical models presented in a blocked practice trial order
23 where each model was repeatedly imitated for a set number of trials (i.e., they trained on
24 one profile before being trained on the other). This design was implemented to establish
25 whether blocked practice allows autistic participants to learn to imitate the atypical
26 kinematics (imitation training was performed in a random trial order in Hayes, Andrew, et al.,
27 2016). Moreover, by transferring imitation from a blocked (acquisition) to random (post-test)
28 trial order, we aimed to evaluate if the processing benefits developed during blocked

1 practice generalise to a test where imitation of typical and atypical kinematics are required in
2 a random trial order.

3 To this end, we specified five sets of a priori hypotheses to test separate aspects of
4 imitation via orthogonal planned comparisons (see below). This statistical technique allowed
5 us to be very clear on what questions we wanted to answer by a priori isolating differences
6 between sets of specific means within these planned contrasts. This approach offers an
7 advantage because it provides more statistical power against making type-II errors,
8 therefore leading to a greater likelihood of detecting real differences between means of
9 interest while still protecting alpha (see Thompson, 1990). The first set of planned
10 comparisons tested the hypothesis that autistic individuals will in general be less effective at
11 voluntary imitation than neurotypical individuals. The second and third sets of planned
12 comparisons examined whether imitating in a blocked practice trial order underpins
13 sensorimotor adaptation in autism by facilitating the integration and encoding of atypical
14 biological kinematics. We compared imitation of the atypical model in the pre-test
15 (randomised trial order) against the middle-acquisition (blocked practice), as well as early-
16 acquisition (blocked practice) against late-acquisition (blocked practice). If the blocked
17 practice trial order facilitates sensorimotor adaptation in autism, we expect imitation of
18 atypical kinematics to be significantly different (i.e., closer to the atypical model) when
19 compared to imitating in the random trial order (pre-test), and when comparing imitation after
20 completing all blocked practice trials (i.e., late-acquisition). Finally, the fourth and fifth sets of
21 planned comparisons examined whether imitating the atypical model in a blocked practice
22 trial order facilitated sensorimotor planning and learning in autism. For sensorimotor
23 planning, we compared imitation during the late-acquisition block (blocked trial order)
24 against the post-test (random trial order). If voluntary imitation differences in autism are a
25 result of sensorimotor integration rather than planning, we expect to find no significant
26 change in imitation performance from the late-acquisition block to the post-test. For
27 sensorimotor learning, we compared imitation during the pre-test (random trial order) against
28 the post-test (random trial order). If imitating in a blocked practice trial order facilitates

1 sensorimotor adaptation and the refinement of an internal action model, we expect to show a
2 significant change in imitation performance, and therefore learning, between the pre-test and
3 post-test.

4

5 Method

6 Participants

7 Twenty neurotypical participants (15 male; 5 female) and 20 autistic participants (15
8 male; 5 female) volunteered for the study. The participants were recruited from an autistic
9 society, and the host university. The participants were provided with a participant information
10 sheet and given the opportunity to consent to be part of the study. All volunteers were right-
11 handed, which was established via an in-house self-report process where a researcher
12 asked the participants a set of pre-experimental questions ("which hand do you write with";
13 "which hand do you throw with"; which hand do you use to brush your teeth"). Furthermore,
14 participants were screened, via self-report, for the following exclusion criteria: dyspraxia,
15 dyslexia, epilepsy and other neurological or psychiatric conditions. The autistic participants
16 had a diagnosis of autism, Asperger's syndrome or autism spectrum disorder by an
17 independent clinician. Diagnosis was confirmed by a researcher trained (with research-
18 reliability status) in the administration of module 4 of the Autism Diagnostic Observation
19 Schedule 2 (ADOS-2) (Lord et al., 2012). All autistic participants met the threshold for autism
20 spectrum disorder on the ADOS-2 total classification score, and on the communication and
21 social interaction subscales. Groups were equated for age, as well as full-scale, verbal and
22 performance IQ, which was measured via the Wechsler Abbreviated Scale of Intelligence
23 (WASI) (Wechsler, 1999). Sample characteristics are presented in Table 1. In addition to the
24 autistic volunteers who participated in the study, we also engaged with a group (n = 6; 1
25 female; 5 male) of autistic advocates who helped to develop the experimental methods via a
26 participatory research process (Fletcher-Watson et al., 2019, Nicolaidis et al., 2011). During
27 engagement, advocates offered their opinion on the apparatus, number of trials, task
28 instructions, how the participant information sheets were constructed, as well as the

1 proposed research question. Feedback from the participatory engagement process was
 2 used to refine the methods of the current experiment. Finally, the experiment was designed
 3 in accordance with the 1964 declaration of Helsinki and approved by the local research
 4 ethics committee.

5

6 Table 1. Characteristics of autism and neurotypical participants.

	Autism (<i>n</i> = 20)		Neurotypical (<i>n</i> = 20)		<i>t</i> test
	Mean (SD)	Range	Mean (SD)	Range	<i>p</i> value
Chronological age in years	27 (8)	18-48	25 (8)	18-46	<i>p</i> = 0.509
Full scale IQ	110 (10)	93-129	110 (10)	85-128	<i>p</i> = 0.893
Verbal IQ	112 (12)	87-134	111 (8)	92-122	<i>p</i> = 0.858
Performance IQ	106 (10)	89-123	105 (10)	82-128	<i>p</i> = 0.803
Gender	15M : 5F		15M : 5F		

7

8

9 Apparatus

10 Participants sat facing a 21-inch CRT monitor (Iiyama Vision Master 505), operating
 11 with a resolution of 1280 x 1024 pixels and a refresh rate of 85 Hz, located on a table at a
 12 viewing distance of 900 mm. Connected to the monitor was a desktop PC (Hewlett Packard
 13 Compaq 8000), graphics tablet and a hand-held stylus (Wacom Intuos Pro XL).

14 Experimental stimuli were generated on the host PC using the COGENT toolbox (developed
 15 by John Romaya at the Laboratory of Neurobiology at the Wellcome Department of Imaging
 16 Neuroscience) implemented in MATLAB (Mathworks Inc.). Movement of the left eye was
 17 recorded at 250 Hz using an EyeLink eye tracker (SR Research) with remote optics. The
 18 host PC and EyeLink were synchronized using a TTL signal.

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Stimuli

To examine the imitation of biological kinematics, participants observed point-light models that displayed a single white-dot (diameter = 6.25 mm) that moved from the home-position on the left-hand side of the screen to the right-hand end-position (Figure 1a). The movement occurred in the horizontal axis only, with an amplitude of 200 mm and total movement time duration of 1700 ms. Two models, which were created by a human volunteer, displayed typical or atypical velocity profiles. The method of using a human volunteer to generate both models was critical because it ensured the kinematics were biological and could be reproduced by the participants. The typical model displayed a bell-shaped velocity profile (Elliott et al., 2010; Flash & Hogan, 1985), which is characteristic of most upper-limb movements (displacement time-series is displayed as the dashed trace in Figure 1b), and had a magnitude of peak velocity that was 0.19 mm/ms and a peak that occurred at 44 % of the movement duration. The atypical model (black trace in Figure 1b) had a magnitude of peak velocity that was 0.33 mm/ms that occurred at 18 % of the movement duration.

Insert Figure 1 around here.

Procedure

The imitation task consisted of a familiarisation period, a pre-test, an acquisition-phase and a post-test. The familiarisation period simulated the general experimental conditions and instructions used during the experimental imitation trials. Participants were instructed to “watch and pay attention to the dot’s trajectory, with the intention to then copy the trajectory”. The dot was a single point-light white dot (diameter = 6.25 mm) that displayed a horizontal left to right movement (Figure 1a), that had the same movement duration (1700 ms), and amplitude (200 mm), as the two experimental models, but had a constant velocity profile. The constant velocity (0.18 mm/ms) model ensured construct

1 validity by preventing participants experiencing biological kinematics before the experimental
2 imitation trials in the three follow-up phases. Participants were not informed about the
3 duration of the movement or the type of stimuli. After observing the model, the participants
4 practised imitation by moving a stylus on a graphics tablet so that a cursor, representing the
5 stylus on a CRT monitor, moved from a home-position red target (diameter = 12.50 mm)
6 located on the left-hand side of the monitor and ended on the right-hand side of the monitor
7 as per the movement displayed by the model. Participants confirmed they understood the
8 model, the instruction regarding how to imitate a model, and the sensorimotor relationship
9 between the movement of the stylus on the graphics tablet and the corresponding
10 movement of the cursor on the CRT monitor.

11 Following the familiarisation period, and prior to the experimental phases,
12 participants performed a standardised set-up routine in order to record eye movements
13 whilst observing a model. First, an automated system ‘calibration’ procedure recorded the
14 raw eye data to gaze position on the CRT monitor. Participants were required to fixate gaze
15 on a small white spot at the centre of a black circular target, which appeared randomly for
16 1000 ms at each location of a standard 9-point grid. Following calibration, an experimenter
17 performed a visual ‘validation’ procedure within the software package to confirm the
18 accuracy of the fixations during calibration. Calibration was only accepted if the average
19 error was $< 1^\circ$ of visual angle.

20 Following the eye recording set-up, the pre-test consisted of 12 imitation trials (6
21 atypical, 6 typical) presented in a randomised trial order that reduced the predictability of an
22 upcoming model. Participants were instructed to “watch and pay attention to the dot’s
23 trajectory, with the intention to then copy the trajectory”. In the acquisition-phase, the groups
24 performed a block of 30 imitation trials of the atypical model, and 30 imitation trials of the
25 typical model. The presentation order of the two blocks was counterbalanced across
26 participants. Participants received the same instructions to “watch and pay attention to the
27 dot’s trajectory, with the intention to then copy the trajectory”. The blocked practice trial order

1 was used to facilitate trial-to-trial sensorimotor integration and planning. Finally, participants
2 completed a post-test that replicated the exact procedures of the pre-test.

3

4 Data Reduction

5 Behavioural Data

6 To quantify imitation of movement kinematics we focused the analysis on x-axis data
7 only (Hayes, Andrew, Elliott, Roberts, & Bennett, 2012; Hayes, Elliott, & Bennett, 2010,
8 2013; Hayes, Roberts, Elliott, & Bennett, 2014). The perpendicular deviation in the y-axis for
9 the atypical model and typical model was minimal as confirmed by a root mean square error
10 of 0.9 mm for the atypical model and 1.55 mm for the typical model. We identified within the
11 x-axis position data the start and end of the movement. The start was defined as the
12 moment the centre of the cursor moved beyond the perimeter of the home-position, and end
13 equated to the moment the participant clicked the upper-button on the stylus. For each
14 imitation trial, the resulting position data were filtered using a low pass 4th order
15 autoregressive filter with an 8 Hz cut-off. The filtered data were then differentiated using a
16 central difference algorithm to obtain velocity. A MATLAB routine extracted percentage time
17 to peak hand velocity (tPHV) from each trial. This dependent variable provides a discrete
18 kinematic measure/marker that accurately represents whether participants imitated a key
19 timing characteristic (peak velocity) of the atypical and typical models (Hayes et al., 2014).

20

21 Eye Movement Data

22 To quantify eye behaviour during the action-observation phase of imitation we
23 focused the analysis on the x-axis data recorded from the left-eye. Synchronisation signals
24 (TTL from host computer) were used to identify the start and end of stimulus presentation
25 and the corresponding eye movement during each trial. Saccades were identified in the x-
26 axis eye position data using the proprietary algorithm in the EyeLink software. The criterion
27 for saccade identification was a velocity threshold of 30 deg/s, acceleration threshold of
28 8000 deg/s², and a motion threshold of 0.15 deg. Saccades plus an additional five data

1 points (equivalent to 20 ms) at the beginning and end of the identified saccade trajectory
2 were then removed from the eye velocity trace. The removed data were replaced by a linear
3 interpolation routine based on the smooth eye velocity before and after the saccade (Bennett
4 & Barnes, 2003). The desaccaded smooth eye velocity was then low-pass filtered using a
5 moving average zero-phase filter (40 ms window). To quantify how well the eye matched the
6 velocity trajectory of the observed model, we extracted percentage-time-to-peak smooth eye
7 velocity for each trial. This discrete measure of smooth eye movement provides a means to
8 quantify pursuit of the observed model, and thus the locus of overt visual attention, which
9 normally coincides with the moving model (Lovejoy et al., 2009), albeit sometimes with a
10 slight lead (Van Donkelaar et al., 2002). Covertly attending to other areas or locations would
11 be possible, although effortful and unlikely given that there were no other relevant cues
12 within the model presentation. Therefore, a good match between the temporal occurrence of
13 peak smooth eye velocity and peak velocity of the model stimulus, would provide a simple
14 and clear indication that participants pursued the observed model stimuli and thus engaged
15 with the task.

16

17 Data Analysis

18 For all dependent variables, intra-participant means were calculated from the
19 kinematic data in the imitation phases, and from the eye movement data in the action-
20 observation phases. For the pre-test and post-test, means were calculated from the 6 trials
21 performed during the imitation of atypical, and typical biological kinematics. For acquisition,
22 means were calculated from trials that represented the early (1-6), middle (13-18) and late
23 (25-30) stages of acquisition. In order to examine the a priori questions associated with
24 imitation learning, each dependent variable was first submitted to a separate 2 Group
25 (autism; neurotypical) x 2 Model (atypical; typical) x 5 Phase (pre-test; early-acquisition;
26 middle-acquisition; late-acquisition; post-test) mixed design ANOVA. We then conducted 5
27 sets of orthogonal planned comparisons to address specific a priori hypotheses/questions
28 for each dependant variable. The first set of planned comparisons are associated with

1 variance pooled from all phases of the imitation protocol. The second set of separate
2 planned comparisons compared imitation behaviour from the pre-test (random trial order) to
3 middle-acquisition (blocked trial order) for the autism and neurotypical groups. The third set
4 of planned comparisons examined imitation behaviour across acquisition by comparing
5 early-acquisition (blocked trial order) against the pooled behaviour of the middle/late-
6 acquisition (blocked trial order) for the autism and neurotypical groups. The fourth set of
7 planned comparisons examined imitation behaviour from the late stage (blocked trial order)
8 of acquisition to the post-test (random trial order). The final set of planned comparisons
9 investigated learning by examining imitation behaviour from the pre-test (random trial order)
10 to the post-test (random trial order). Alpha was set at $p < 0.05$, and Cohen's d was used to
11 express the size of the effect.

12 To establish whether the degree of sensorimotor learning measured in the final set of
13 planned comparisons (i.e., pre-test to post-test) was related to the magnitude of
14 sensorimotor adaptation across acquisition (i.e., planned comparison three), we first
15 computed the percentage change ($\% \Delta$) between the mean percentage-time-to-peak-hand-
16 velocity in the pre-test and post-test (i.e., $\% \Delta = ((\text{post } \bar{x} - \text{pre } \bar{x}) / \text{pre } \bar{x}) * 100$) for both the
17 atypical and typical models. The same was also completed for the mean percentage-time-to-
18 peak-hand-velocity in early-acquisition compared to the pooled behaviour of the middle/late-
19 acquisition periods (i.e., $\text{Mid/Late} = (\text{Mid} + \text{Late}) / 2$; $\% \Delta = ((\text{Mid/Late } \bar{x} - \text{Early } \bar{x}) / \text{Early}$
20 $\bar{x}) * 100$). We then correlated the percentage change scores ($\% \Delta$) for the autism and
21 neurotypical groups separately. A significant positive correlation demonstrates that greater
22 adaptation from pre-test to post-test is related to becoming more accurate during the
23 blocked acquisition period. Whereas a nonsignificant relationship would suggest the blocked
24 trial order during acquisition is not contributing towards imitation learning.

25

26 Results

27 Percentage time to peak hand velocity (tPHV)

1 tPHV data for both groups across all phases of the imitation learning protocol are
2 illustrated in Figure 2 (a, atypical; b, typical). The first set of planned comparisons are
3 associated with variance pooled from all phases of the imitation protocol for each group.
4 First, there was a significant difference in general imitation behaviour between the autism
5 and neurotypical groups [$F(1, 38) = 7.05, p = 0.01, d = 0.47$]. When examining imitation
6 across the two models, the autism [$F(1, 38) = 17.95, p < 0.001, d = 0.90$] and neurotypical
7 [$F(1, 38) = 47.73, p < 0.001, d = 1.63$] groups showed significant differences in behaviour
8 when imitating the atypical (autism $M = 28.46 \pm 8.98$; neurotypical $M = 20.99 \pm 7.67$) and
9 typical (autism $M = 36.76 \pm 9.88$; neurotypical $M = 34.52 \pm 9.29$) models.

10
11 Insert Figure 2 around here.
12

13 The second set of separate planned comparisons compared imitation behaviour from
14 the pre-test (random trial order) to middle-acquisition (blocked trial order) for the autism and
15 neurotypical groups. Middle-acquisition was selected as it was deemed an appropriate stage
16 to examine sensorimotor adaptation following the completion of half of the imitation trials.
17 For the neurotypical group, there was no significant differences in behaviour when imitating
18 either model across the two phases [atypical: $F(1, 38) = 0.40, p = 0.53, d = 0.14$; typical: F
19 $(1, 38) = 0.09, p = 0.76, d = 0.10$]. The percentage change when imitating the atypical model
20 was $\% \Delta = 5$, and the typical model was $\% \Delta = 2$. Although the autism group demonstrated no
21 significant change ($\% \Delta = 2$) in behaviour when imitating the typical model [$F(1, 38) = 0.11, p$
22 $= 0.75, d = 0.08$], there was a significant change ($\% \Delta = 17$) leading to peak velocity
23 occurring earlier in the movement when imitating the atypical model [$F(1, 38) = 9.47, p =$
24 $0.004, d = 0.66$].

25 The third set of planned comparisons examined imitation behaviour across
26 acquisition by comparing early-acquisition (blocked trial order) against the pooled behaviour
27 of the middle/late-acquisition (blocked trial order) for the autism and neurotypical groups.
28 There were no significant changes across these phases for the neurotypical group when

1 imitating either model [atypical: $F(1, 38) = 0.88, p = 0.36, d = 0.08$; typical: $F(1, 38) = 0.04,$
2 $p = 0.84, d = 0.09$]. The percentage change when imitating the atypical model was $\% \Delta = 5,$
3 and the typical model was $\% \Delta = 1$. Although the autism group demonstrated no significant
4 change ($\% \Delta = 3$) in behaviour when imitating the typical model [$F(1, 38) = 0.26, p = 0.61, d$
5 $= 0.14$], there was a significant change ($\% \Delta = 9$) leading to peak velocity occurring earlier in
6 the movement when imitating the atypical model [$F(1, 38) = 4.62, p = 0.04, d = 0.31$].

7 The fourth set of planned comparisons examined imitation behaviour from the late
8 stage (blocked trial order) of acquisition to the post-test (random trial order). There were no
9 significant changes across these phases for the neurotypical group [atypical: $F(1, 38) =$
10 $0.67, p = 0.42, d = 0.13$; typical: $F(1, 38) = 2.11, p = 0.15, d = 0.24$] or the autism group
11 [atypical: $F(1, 38) = 3.29, p = 0.08, d = 0.22$; typical: $F(1, 38) = 2.60, p = 0.12, d = 0.30$]
12 when imitating either model. The percentage change when imitating the atypical model was
13 $\% \Delta = 7$ for autism group and $\% \Delta = 4$ for the neurotypical group. When imitating the typical
14 model, the autism group showed $\% \Delta = 7$, and the neurotypical group $\% \Delta = 7$.

15 The final set of planned comparisons investigated learning by examining imitation
16 behaviour from the pre-test (random trial order) to the post-test (random trial order). There
17 was no overall learning effect in the neurotypical group for either model [atypical: $F(1, 38) =$
18 $0.38, p = 0.54, d = 0.13$; typical: $F(1, 38) = 0.43, p = 0.52, d = 0.20$]. Although the autism
19 group showed no learning of the typical model [$F(1, 38) = 0.07, p = 0.79, d = 0.07$], they
20 demonstrated a significant learning effect for the atypical model [$F(1, 38) = 6.29, p = 0.02, d$
21 $= 0.47$]. The percentage change when imitating the atypical model was $\% \Delta = 13$ for autism
22 group and $\% \Delta = 5$ for the neurotypical group. When imitating the typical model, the autism
23 group showed $\% \Delta = 2$, and the neurotypical group $\% \Delta = 5$.

24

25 Percentage time to peak smooth eye velocity (tPSEV)

26 tPSEV data for both groups across all phases of the imitation learning protocol are
27 illustrated in Figure 3 (a, autism; b, neurotypical). The first set of planned comparisons are
28 associated with variance pooled from all phases of the imitation protocol. First, there was no

1 significant difference in tPSEV when examining behaviour at the group level [$F(1, 29) =$
2 $0.04, p = 0.84, d = 0.02$]. When examining tPSEV as a function of observing the different
3 models, the autism [$F(1, 29) = 169.93, p < 0.001, d = 2.81$] and neurotypical [$F(1, 29) =$
4 $243.44, p < 0.001, d = 4.97$] groups showed significant differences in behaviour when
5 observing the atypical (autism $M = 31.67 \pm 6$; neurotypical $M = 30.37 \pm 4.03$) and typical
6 (autism $M = 50.55 \pm 7.55$; neurotypical $M = 52.25 \pm 5.03$) models.

7
8 Insert Figure 3 around here.

9
10 The second set of separate planned comparisons compared tPSEV from the pre-test
11 (random trial order) to middle-acquisition (blocked trial order) for the autism and neurotypical
12 groups. There were no significant changes across these phases when observing either
13 model for the neurotypical group [atypical: $F(1, 29) = 0.05, p = 0.83, d = 0.10$; typical: $F(1,$
14 $29) = 0.001, p = 0.97, d = 0.01$] or the autism group [atypical: $F(1, 29) = 0.18, p = 0.68, d =$
15 0.11 ; typical: $F(1, 29) = 2.31, p = 0.14, d = 0.45$]. The percentage change for the
16 neurotypical group when observing the atypical model was $\% \Delta = 2$, and the typical model
17 was $\% \Delta = <1$, and for the autism group when observing the atypical model was $\% \Delta = 2$, and
18 the typical model was $\% \Delta = 6$.

19 The third set of planned comparisons examined tPSEV across acquisition by
20 comparing early-acquisition (blocked trial order) against the pooled behaviour of the
21 middle/late-acquisition (blocked trial order) for the autism and neurotypical groups. There
22 were no significant changes across these phases when observing either model for the
23 neurotypical group [atypical: $F(1, 29) = 0.15, p = 0.70, d = 0.15$; typical: $F(1, 29) = 0.83, p =$
24 $0.37, d = 0.26$] or the autism group [atypical: $F(1, 29) = 3.55, p = 0.07, d = 0.57$; typical: F
25 $(1, 29) = 0.001, p = 1.00, d = 0.001$]. The percentage change for the neurotypical group when
26 observing the atypical model was $\% \Delta = 3$, and the typical model was $\% \Delta = 3$, and for the
27 autism group when observing the atypical model was $\% \Delta = 13$, and the typical model was
28 $\% \Delta = <1$.

1 The fourth set of planned comparisons examined tPSEV from the late stage (blocked
2 trial order) of acquisition to the post-test (random trial order). When observing the atypical
3 model, tPSEV occurred earlier ($\% \Delta = 10$) for the autism group in the post-test compared to
4 the late stage of acquisition [$F(1, 29) = 4.31, p = 0.05, d = 0.53$]. The autism group did not
5 demonstrate a significant change ($\% \Delta = 3$) when observing the typical model [$F(1, 29) =$
6 $0.53, p = 0.47, d = 0.18$]. There were no significant changes across these phases when
7 observing either model for the neurotypical group [atypical: $F(1, 29) = 0.01, p = 0.93, d =$
8 0.04 ; typical: $F(1, 29) = 0.34, p = 0.57, d = 0.23$]. The percentage change when observing
9 the atypical model was $\% \Delta = <1$, and the typical model was $\% \Delta = 2$.

10 The final set of planned comparisons investigated learning by examining tPSEV from
11 the pre-test (random trial order) to the post-test (random trial order). When observing the
12 atypical model, peak-smooth-eye-velocity occurred earlier ($\% \Delta = 8$) for the autism group in
13 the post-test compared to the pre-test [$F(1, 29) = 6.75, p = 0.01, d = 0.66$]. The autism
14 group did not demonstrate a significant change ($\% \Delta = 4$) when observing the typical model
15 [$F(1, 29) = 2.06, p = 0.16, d = 0.48$]. There were no significant changes across these
16 phases when observing either model for the neurotypical group [atypical: $F(1, 29) = 0.70, p$
17 $= 0.41, d = 0.36$; typical: $F(1, 29) = 2.25, p = 0.14, d = 0.41$]. The percentage change when
18 observing the atypical model was $\% \Delta = 3$, and the typical model was $\% \Delta = 4$.

19

20 Relationship between changes in imitation accuracy across acquisition, and changes in 21 imitation accuracy from pre-test to post-test.

22 Pearson's correlation analyses indicated a significant relationship between the
23 magnitude of adaptation ($\% \Delta$) during acquisition, and magnitude of adaptation ($\% \Delta$) from
24 pre-test to post-test, for the imitation of the atypical model by the autism group ($r = 0.454, p$
25 $= 0.04$; Fig. 4a) but not the neurotypical group ($r = -0.145, p = 0.54$; Fig. 4c). As illustrated in
26 Figure 4, there was a positive relationship whereby autistic participants who demonstrated
27 the greatest (or lowest) magnitude of sensorimotor adaptation across acquisition (see Y-axis
28 on Figure 4a) also exhibited a greater (or lower) change in imitation from pre-test to post-test

1 (see X-axis on Figure 4a). There were no significant relationships for either group (autism
2 (Fig. 4b: $r = 0.053$, $p = 0.83$; Fig. 4d neurotypical: $r = 0.390$, $p = 0.09$) when imitating the
3 typical model.

4

5 Insert Figure 4 around here.

6

7 Discussion

8 Although voluntary imitation is generally different in autistic compared to neurotypical
9 individuals (DeMyer et al., 1972; Rogers & Pennington, 1991; Vivanti & Hamilton, 2014),
10 there is evidence that certain general sensorimotor processes underlying imitation are
11 operational (Bird et al., 2007; Hamilton et al., 2007; Hayes, Andrew, et al., 2016). To better
12 understand how sensorimotor processes function during the imitation, we examined
13 performance in autistic and neurotypical volunteers following a period of blocked practice at
14 learning to imitate a biological model with an atypical velocity profile. Learning was assessed
15 by measuring performance change from the pre-test to post-test where imitation was
16 performed in random trial orders. Any significant adaptation effects following of blocked
17 practice would be generalised to a trial order that was randomised in the post-test.

18 The first set of planned comparisons confirmed a general difference in imitation
19 behaviour between autistic and neurotypical participants, thereby suggesting certain
20 sensorimotor processing operations in autism impact the efficacy of how novel actions are
21 imitated (DeMyer et al., 1972; Hayes, Andrew, et al., 2016; Hobson & Lee, 1999; Rogers et
22 al., 1996; Stewart et al., 2013; Wild et al., 2012). Nonetheless, both groups did scale hand
23 and eye kinematics such that peak velocity occurred earlier in the movement trajectory when
24 imitating the atypical compared to the typical model. As well as replicating previous findings
25 in neurotypical participants (Andrew, Bennett, Elliott, & Hayes, 2016; Hayes, Dutoy, Elliott,
26 Gowen, & Bennett, 2016), this is the first evidence showing that autistic individuals can
27 imitate novel atypical biological kinematics that would not have existed in their motor
28 repertoire.

1 The results from the second and third sets of planned comparisons suggest that the
2 imitation of atypical kinematics demonstrated by the autism group was underpinned by
3 processes that facilitated sensorimotor integration and adaptation across blocked practice
4 (Magill & Hall, 1990). Compared to the neurotypical group that successfully imitated the
5 atypical model at pre-test and middle-acquisition, the second planned comparison indicated
6 the autism group exhibited a significant 17% change (5 units of tPHV; pre-test, M = 32;
7 middle, M = 27) in imitation behaviour at the middle-acquisition phase of imitating the
8 atypical model during blocked practice. This significant change is important because in our
9 previous work (Hayes, Andrew, et al., 2016) where a group of comparable autistic adults
10 imitated atypical, typical and constant velocity kinematics presented randomly across 84
11 trials, we showed no adaptation effects across a similar number of practice trials (tPHV; pre-
12 test, M = 33; late phase, M = 33). The third planned comparison, which examined changes
13 in imitation from early-acquisition to middle/late-acquisition where trials were received only in
14 a blocked practice trial order, indicated the autism group significantly adapted tPHV by 9%.
15 This finding indicates that the imitation adaptation effects observed in the second and third
16 planned comparisons were not merely a consequence of switching the learning environment
17 from a randomised to blocked practice trial order. Moreover, the fact that we showed no
18 such change in our previous work (Hayes, Andrew, et al., 2016) indicates that the
19 sensorimotor adaptation effect found here is unlikely to be underpinned by processes
20 governing general practice effects.

21 Before interpreting the adaptation effects associated with the hand kinematics, it is
22 noteworthy to comment that our measure of smooth pursuit eye velocity (tPSEV) was
23 appropriately scaled to the different models by both groups [atypical (autism M = 32;
24 neurotypical M = 30); typical (autism M = 51; neurotypical M = 52)]. These data show that
25 the high-acuity region of the fovea was maintained within the vicinity of both models during
26 pursuit (see Lovejoy et al., 2009), and would have provided retinal and extra-retinal input on
27 the observed biological kinematics that could contribute to subsequent configuration of the
28 limb movement. Second, neither group significantly changed tPSEV when imitating the

1 atypical model in the pre-test (random trial order) compared to middle-acquisition (blocked
2 trial order), or from early-acquisition to middle/late-acquisition (NB. both had blocked trial
3 order). At 13%, the change in tPSEV from early-acquisition to middle/late-acquisition for the
4 autism group was close to significance ($p = 0.07$). This change was based on tPSEV
5 occurring later in the middle/late phase (33%) compared to the early phase (29%).
6 Conversely, the significant 9% change in hand kinematics for autism group indicated the
7 opposite effect, with tPHV equal to 26% in the middle/late phase and 29% in the early
8 phase. The implication is that the significant adaptation effect in our measure of hand
9 kinematics by the autism group when imitating the atypical model (across acquisition) is
10 unlikely to simply be related to smooth pursuit eye movements.

11 Together with our previous work (Hayes, Andrew, et al., 2016), we suggest the
12 imitation adaptation effect observed for the hand kinematics was underpinned by the way
13 the blocked practice trial order engaged the underlying sensorimotor processes over
14 repeated attempts at imitating the atypical model. Along with decreasing functional task
15 difficulty because only one sensorimotor action plan is represented (Guadagnoli & Lee,
16 2004), the trial order most likely facilitated imitation by optimising sensorimotor control and
17 integration processes engaged to specify the forces required to initially execute the
18 movement (Magill & Hall, 1990). Moreover, by keeping sensorimotor information similar
19 between consecutive trials, the comparison and processing of expected (efference copy;
20 feedforward control) and actual (reafference; feedback control) sensorimotor consequences
21 from trial n can be integrated more effectively. This blocked practice structure therefore
22 optimises feedforward and feedback control mechanisms during motor execution (Kantak &
23 Winstein, 2012), and subsequent sensorimotor consolidation and planning for trial $n+1$
24 (Elliott et al., 2001; Wolpert et al., 2011), which enables an internal action model
25 representing the atypical kinematics to be refined and encoded.

26 Further evidence that sensorimotor adaptation was optimised by facilitating the
27 integration and encoding of atypical biological kinematics is apparent from the fourth and
28 fifth sets of planned comparisons. The fourth set indicated no significant changes in

1 behaviour for either group when imitation was compared from late-acquisition (blocked trial
2 order) to the post-test (randomised trial order). This is in contrast to the significant change
3 found in the fifth set, where the autism group successfully imitated the atypical kinematics at
4 post-test compared to pre-test. These combined effects indicate the processing changes
5 that occurred during blocked practice underpinned the encoding of an internal action model
6 that was operational when the autism group was transferred back to a randomised trial order
7 in the post-test. This learning effect and subsequent positive transfer indicate that
8 differences in voluntary imitation in autism (DeMyer et al., 1972; Rogers & Pennington,
9 1991; Vivanti & Hamilton, 2014) are not solely due to sensorimotor planning problems
10 (Glazebrook, Elliott, & Lyons, 2006; Rinehart, Bradshaw, Brereton, & Tonge, 2001)
11 associated with imitating a novel action (Hayes, Andrew, et al., 2016; Stewart et al., 2013;
12 Wild et al., 2012). Rather, it would seem that while the underlying visuomotor system
13 activated during voluntary imitation in autism is functional, operational imitation of atypical
14 biological kinematics requires the practice conditions to be structured optimally (e.g.,
15 'Challenge Point' framework; Guadagnoli & Lee, 2004) in order to facilitate sensorimotor
16 integration, and learning.

17 In summary, we have shown that the imitation difficulties in autism (i.e., pre-test
18 difference between the autism and control groups) are in part related to sensorimotor
19 processing and integration. Despite there being general differences in imitation efficacy
20 between autistic and control individuals, we have shown for the first time that the autistic
21 sensorimotor system can be modulated by structuring the voluntary imitation environment in
22 a predictable manner that enhances trial-to-trial sensorimotor processing, integration and
23 encoding of atypical biological motion. The fact that the significant adaptation effect occurred
24 from a very short period of training raises the possibility for creating other types of
25 sensorimotor protocols (i.e., autism specific motor interventions used in pre-school and
26 educational settings) based on the processing benefits of blocked practice.

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3

1 Table/Figure Legends

2 Table 1. Characteristics of autism and neurotypical participants.

3

4 Figure 1. (a) A schematic representation of the laboratory/experimental set-up for the
5 imitation task. The black outlined rectangle represents a graphics tablet. The white circle
6 displayed on the CRT monitor represents the model. The single-segment movement is
7 depicted by the arrow (i.e., from the start position to the final position). (b) Displacement
8 time-series displaying the typical (dashed trace) and atypical (black trace) velocity models.

9

10 Figure 2. Percentage-time-to-peak-hand-velocity for the imitation task (error bars represent
11 standard error of the mean) presented as a function of group and phase for the atypical
12 model (a) and the typical model (b). Dashed line represents the model.

13

14 Figure 3. Percentage-time-to-peak-smooth-eye-velocity for the eye during imitation task
15 (error bars represent standard error of the mean) presented as a function of group and
16 phase for the atypical model (a) and the typical model (b). Dashed line represents the
17 model.

18

19 Figure 4. Correlation between the magnitude of sensorimotor adaptation across acquisition
20 (y-axis) and the magnitude of sensorimotor adaptation from pre-test to post-test (x-axis)
21 when imitating the atypical model (autism: A; neurotypical: C) and the typical model (autism:
22 B; neurotypical: D).

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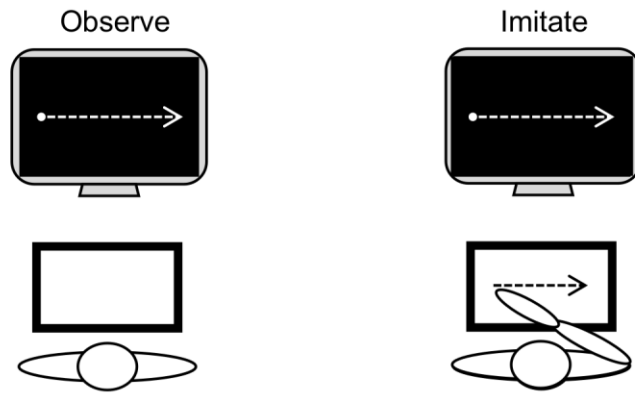
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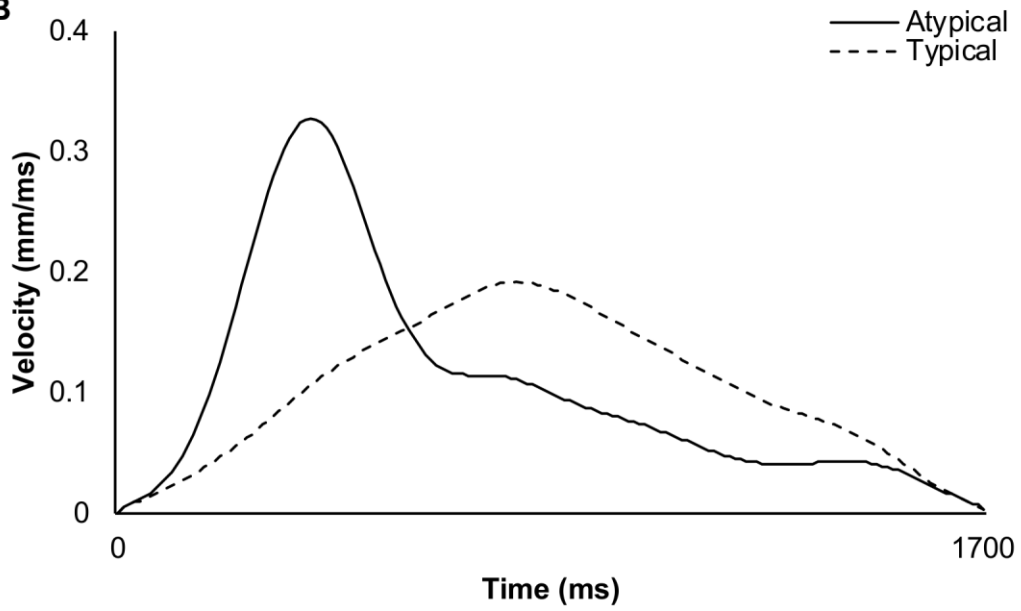
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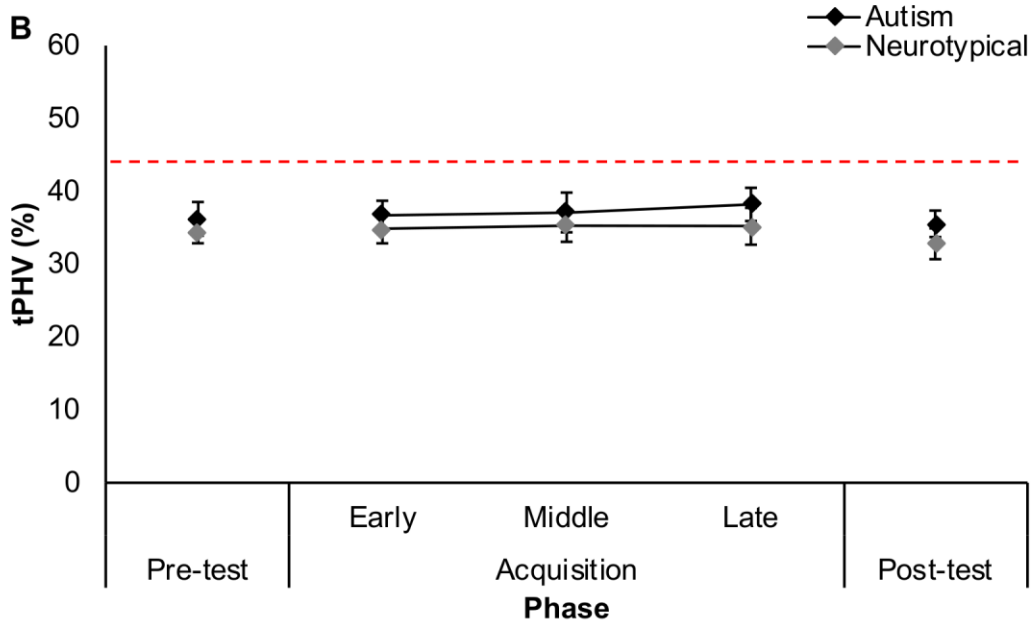
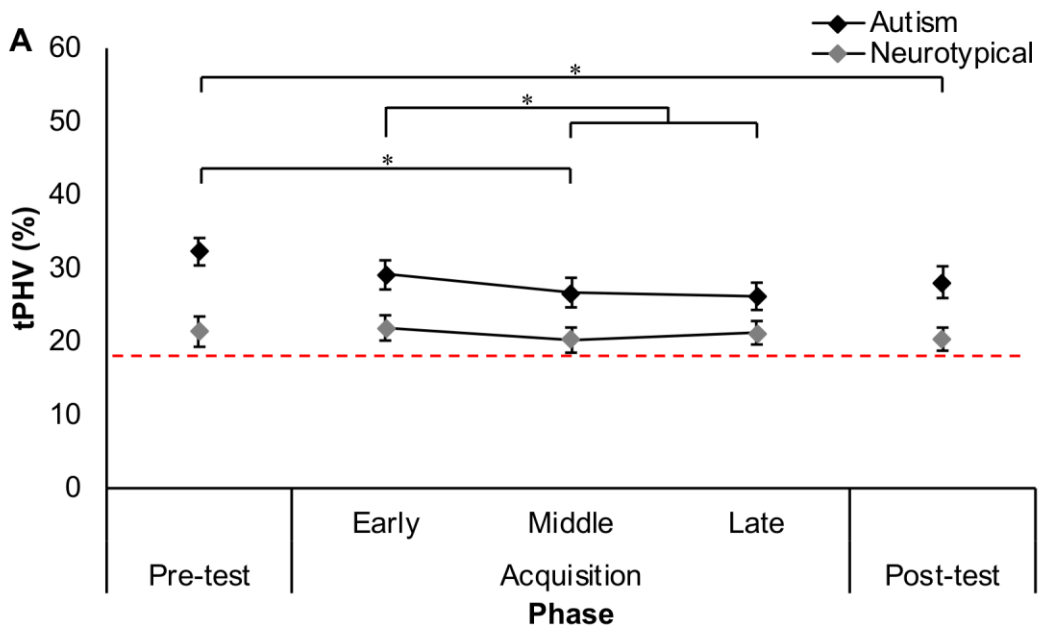


Imitation Trial Timeline →

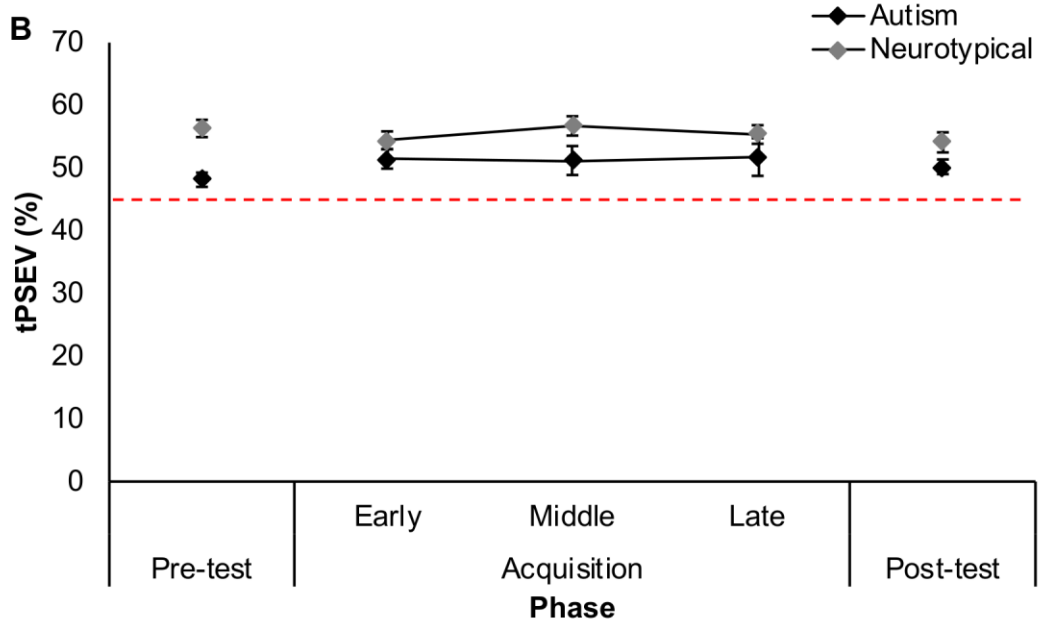
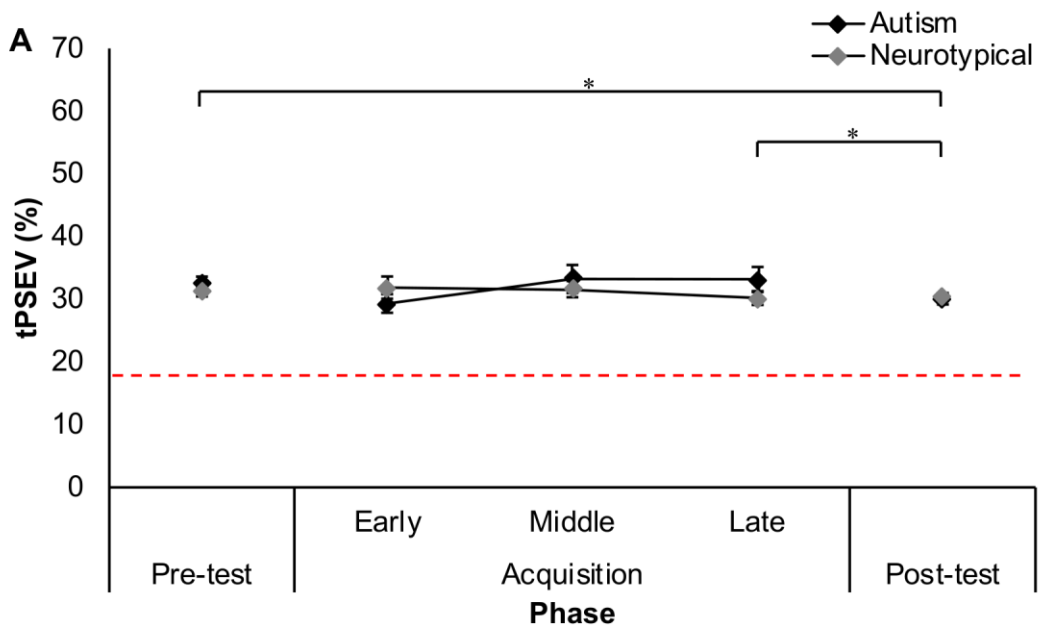
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