Original Article

autism

Autism
2020, Vol. 24(6) 1494–1505

DOI: 10.1177/1362361320908104
journals.sagepub.com/home/aut
SAGE

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

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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.

Lay Abstract

Autistic people sometimes find it difficult to copy another person's movement accurately, especially if the movement is unfamiliar or novel (e.g. to use chop sticks). In this study, we found that autistic people were generally less accurate at copying a novel movement than non-autistic people. However, by making a small adjustment and asking people to copy this movement for a set number of attempts in a predictable manner, we showed that autistic people did successfully learn to copy a new movement. This is a very important finding for autistic people because rather than thinking they cannot copy new movements, all that needs to be considered is for parents/guardians, teachers and/or support workers to make a small adjustment so that learning occurs in a predictable manner for new skills to be successfully acquired through copying. The implications from this study are wide-ranging as copying (imitation) and motor learning are important developmental processes for autistic infants and children to acquire in order to interact within the world. Therefore, practising these behaviours in the most effective way can certainly help the developmental pathway.

Keywords

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

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Introduction

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

In a previous examination of the imitation of biological kinematics in autism, we randomly presented two models that displayed the same movement amplitude and movement time goal (1700 ms) but different underlying kinematics (Hayes, Andrew, et al., 2016). A control model had typical kinematics with a bell-shaped velocity profile (peak velocity that occurred at ~50% of the movement trajectory). This model could be imitated by rescaling a typical movement profile from an existing motor repertoire (Carmo et al., 2013; Rumiati et al., 2005). As predicted, we showed no difference between the autistic and neurotypical groups' imitation of movement kinematics displayed in the control model. An experimental model had a novel atypical kinematic profile, where peak velocity occurred at 18% of the movement trajectory. This model required participants to represent the atypical kinematics during action-observation phase in order to reorganise the sensorimotor system to plan and execute the appropriate motor response during imitation. Unlike the neurotypical group that successfully imitated the atypical kinematics (Hayes, Andrew, et al., 2016), the autistic group produced a movement more similar to the kinematics of the typical model. Still, despite failing to imitate the novel atypical kinematics, the autism group became significantly more accurate and consistent at imitating the 1700-ms movement time goal. This specific adaptation effect associated with learning the overall movement time goal indicates the autistic participants were actively engaged in imitation learning and therefore likely to have followed the task instructions to 'watch and then copy the movement displayed by a white dot on the computer monitor'. Accordingly, we can infer that attention (to the stimuli) and intention (to produce the observed action), which both modulate voluntary imitation (Hayes et al., 2014), were not factors that limited imitation of the movement time goal.

Further insight into the operation of sensorimotor processes in autism is evident from automatic imitation studies (Bird et al., 2007; Edey et al., 2016; Hamilton et al., 2007; Press et al., 2010; Schulte-Rüther et al., 2017; Sowden et al., 2016; Spengler et al., 2010), in which autistic adults have been shown to generate sensorimotor response times similar to matched-controls when observing task-irrelevant biological action stimulus (e.g. a human hand lifting an index finger). In other words, movement observation had a direct automatic influence on motor execution (Brass et al., 2001), thereby confirming the sensorimotor processes responsible for processing biological motion during action-observation phase are operational in autism (Nackaerts et al., 2012; Saygin et al., 2010). The implication for voluntary imitation is that the difficulty imitating atypical biological kinematics is not solely associated with a specific imitation mechanism that directly represents and encodes biological motion during the action-observation phase (Bernier et al., 2007; Williams et al., 2001, 2004). Rather, there may be differences in how other general sensorimotor learning processes (Chetcuti et al., 2019; Hamilton, 2013; Leighton et al., 2008) are engaged to represent and refine the observed and executed biological kinematics during repeated imitation trials.

For example, by presenting the typical and atypical kinematic models in a randomised trial order (Hayes, Andrew, et al., 2016), sensorimotor information from trial n (e.g. atypical model) would often be different to trial $n + 1$ (e.g. typical model). Therefore, executing different sensorimotor actions would have led to 'intratask interference' (Battig, 1972). In a motor learning context, this form of interference is called the contextual interference effect, which is defined 'as the effect on learning of the degree of functional interference found in a practice situation when several tasks must be learned and practiced together' (Magill & Hall, 1990, p. 244). Although practising multiple task variations of a sensorimotor action engages processes that facilitate long-term retention and transfer of the action (Brady, 1998; Edwards et al., 1986; Magill & Hall, 1990; Shea & Morgan, 1979), motor performance during the practice period is often attenuated (i.e. decrease in accuracy, increase in variability). Attenuation occurs because intratask interference affects the efficacy of integrating, and consolidating, different

sensorimotor information sources across trials because the expected (efference) and actual (reafferent) sensorimotor consequences are different (Immink & Wright, 2001). In addition to a contribution from processes underlying integration and consolidation, performance may have been affected because imitating the typical and atypical models in a random trial order would have engaged greater attention-demanding and effortful motor planning processes (Li & Wright, 2000), which are already known to be compromised in autism (Glazebrook et al., 2006; Rinehart et al., 2001).

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

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

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

Method

Participants

A total of 20 neurotypical participants (15 males, 5 females) and 20 autistic participants (15 males, 5 females) volunteered for the study. The participants were recruited from an autistic society and the host university. The

Table 1. Characteristics of autism and neurotypical participants.

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

SD: standard deviation.

participants were provided with a participant information sheet and given the opportunity to consent to be part of the study. All volunteers were right-handed, which was established through an in-house self-report process where a researcher asked the participants a set of pre-experimental questions ('which hand do you write with'; 'which hand do you throw with'; 'which hand do you use to brush your teeth'). Furthermore, participants were screened, through self-report, for the following exclusion criteria: dyspraxia, dyslexia, epilepsy and other neurological or psychiatric conditions. The autistic participants had a diagnosis of autism, Asperger's syndrome or autism spectrum disorder by an independent clinician. Diagnosis was confirmed by a researcher trained (with research-reliability status) in the administration of module 4 of the Autism Diagnostic Observation Schedule 2 (ADOS-2; Lord et al., 2012). All autistic participants met the threshold for autism spectrum disorder on the ADOS-2 total classification score and on the communication and social interaction subscales. Groups were equated for age, as well as full-scale, verbal and performance IQ, which was measured through the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999). Sample characteristics are presented in Table 1. In addition to the autistic volunteers who participated in the study, we also engaged with a group ($n = 6$; 1 female, 5 males) of autistic advocates who helped to develop the experimental methods through a participatory research process (Fletcher-Watson et al., 2019; Nicolaidis et al., 2011). During engagement, advocates offered their opinion on the apparatus, number of trials, task instructions, how the participant information sheets were constructed, as well as the proposed research question. Feedback from the participatory engagement process was used to refine the methods of the current experiment. Finally, the experiment was designed in accordance with the 1964 Declaration of Helsinki and approved by the local research ethics committee.

Apparatus

Participants sat facing a 21-inch CRT monitor (Iiyama Vision Master 505), operating with a resolution of 1280 × 1024 pixels and a refresh rate of 85 Hz, located on a table at a viewing distance of 900 mm. Connected to the monitor was a desktop PC (Hewlett Packard Compaq 8000), graphics tablet and a hand-held stylus (Wacom Intuos Pro XL). Experimental stimuli were generated on the host PC using the COGENT toolbox (developed by John Romaya at the Laboratory of Neurobiology at the Wellcome Department of Imaging Neuroscience) implemented in MATLAB (Mathworks Inc.). Movement of the left eye was recorded at 250 Hz using an EyeLink eye tracker (SR Research) with remote optics. The host PC and EyeLink were synchronised using a TTL (transistor–transistor logic) signal.

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 1(a)). 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 dashed trace in Figure 1(b)), 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 1(b)) had a magnitude of peak velocity that was 0.33 mm/ms that occurred at 18% of the movement duration.

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

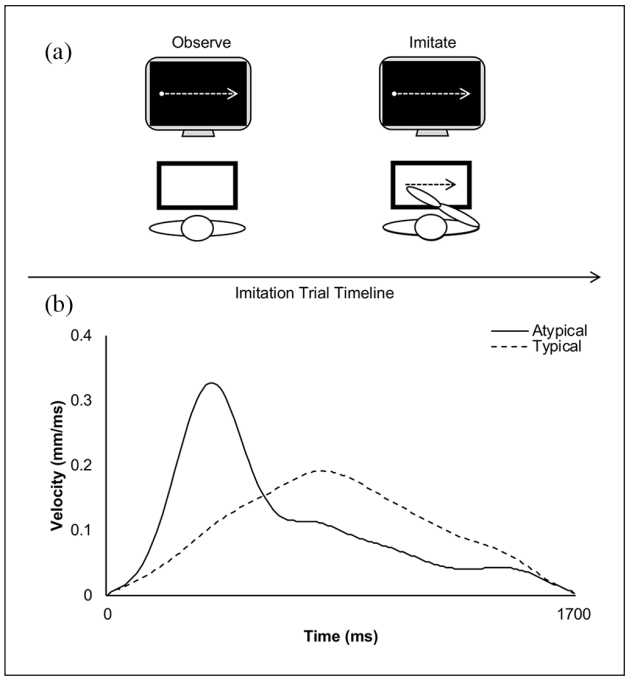

Figure 1. (a) A schematic representation of the laboratory/experimental set-up for the imitation task. The black outlined rectangle represents a graphics tablet. The white circle displayed on the CRT monitor represents the model. The single-segment movement is depicted by the arrow (i.e. from the start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

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

Following the familiarisation period, and prior to the experimental phases, participants performed a standardised set-up routine in order to record eye movements while observing a model. First, an automated system 'calibration' procedure recorded the raw eye data to gaze position on the CRT monitor. Participants were required to fixate gaze on a small white spot at the centre of a black circular target, which appeared randomly for 1000 ms at each location of a standard nine-point grid. Following calibration, an experimenter performed a visual 'validation' procedure within the software package to confirm the accuracy of the fixations during calibration. Calibration was only accepted if the average error was below 1° of visual angle.

Following the eye recording set-up, the pre-test consisted of 12 imitation trials (6 atypical, 6 typical) presented in a randomised trial order that reduced the predictability of an upcoming model. Participants were instructed to 'watch and pay attention to the dot's trajectory, with the intention to then copy the trajectory'. In the acquisition phase, the groups performed a block of 30 imitation trials of the atypical model and 30 imitation trials of the typical model. The presentation order of the two blocks was counterbalanced across participants. Participants received the same instructions to 'watch and pay attention to the dot's trajectory, with the intention to then copy the trajectory'. The blocked practice trial order was used to facilitate trial-to-trial sensorimotor integration and planning. Finally, participants completed a post-test that replicated the exact procedures of the pre-test.

Data reduction

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

Eye movement data. To quantify eye behaviour during the action-observation phase of imitation, we focused the analysis on the *x*-axis data recorded from the left eye. Synchronisation signals (TTL from host computer) were used

to identify the start and end of stimulus presentation and the corresponding eye movement during each trial. Saccades were identified in the *x*-axis eye position data using the proprietary algorithm in the EyeLink software. The criterion for saccade identification was a velocity threshold of 30 deg/s, acceleration threshold of 8000 deg/s^2 and a motion threshold of 0.15 deg. Saccades plus an additional five data points (equivalent to 20 ms) at the beginning and end of the identified saccade trajectory were then removed from the eye velocity trace. The removed data were replaced by a linear interpolation routine based on the smooth eye velocity before and after the saccade (Bennett & Barnes, 2003). The desaccaded smooth eye velocity was then low-pass filtered using a moving average zero-phase filter (40 ms window). To quantify how well the eye matched the velocity trajectory of the observed model, we extracted percentage-time-to-peak-smooth-eye-velocity (tPSEV) for each trial. This discrete measure of smooth eye movement provides a means to quantify pursuit of the observed model, and thus the locus of overt visual attention, which normally coincides with the moving model (Lovejoy et al., 2009), albeit sometimes with a slight lead (Van Donkelaar & Drew, 2002). Covertly attending to other areas or locations would be possible, although effortful and unlikely given that there were no other relevant cues within the model presentation. Therefore, a good match between the temporal occurrence of peak smooth eye velocity and peak velocity of the model stimulus would provide a simple and clear indication that participants pursued the observed model stimuli and thus engaged with the task.

Data analysis

For all dependent variables, intra-participant means were calculated from the kinematic data in the imitation phases and from the eye movement data in the action-observation phases. For the pre-test and post-test, means were calculated from the six trials performed during the imitation of atypical and typical biological kinematics. For acquisition, means were calculated from trials that represented the early (1–6), middle (13–18) and late (25–30) stages of acquisition. In order to examine the a priori questions associated with imitation learning, each dependent variable was first submitted to a separate 2 group (autism, neurotypical) × 2 model (atypical, typical) × 5 phase (pre-test, early-acquisition, middle-acquisition, late-acquisition, post-test) mixed-design ANOVA. We then conducted five sets of orthogonal planned comparisons to address specific a priori hypotheses/questions for each dependant variable. The first set of planned comparisons are associated with variance pooled from all phases of the imitation protocol. The second set of separate planned comparisons compared imitation behaviour from the pre-test (random trial order) to middle-acquisition (blocked trial order) for the autism and neurotypical groups. The third set of planned comparisons examined imitation behaviour across acquisition by comparing early-acquisition (blocked trial order) against the pooled behaviour of the middle/late-acquisition (blocked trial order) for the autism and neurotypical groups. The fourth set of planned comparisons examined imitation behaviour from the late stage (blocked trial order) of acquisition to the post-test (random trial order). The final set of planned comparisons investigated learning by examining imitation behaviour from the pre-test (random trial order) to the post-test (random trial order). Alpha was set at $p < 0.05$, and Cohen's *d* was used to express the size of the effect.

To establish whether the degree of sensorimotor learning measured in the final set of planned comparisons (i.e. pre-test to post-test) was related to the magnitude of sensorimotor adaptation across acquisition (i.e. Planned Comparison 3), we first computed the percentage change (%Δ) between the mean tPHV in the pre-test and post-test (i.e. $\%\Delta = ((\text{post}\,\bar{x} - \text{pre}\,\bar{x}) / \text{pre}\,\bar{x}) \times 100$) for both the atypical and the typical models. The same was also completed for the mean tPHV in early-acquisition compared to the pooled behaviour of the middle/late-acquisition periods (i.e. Mid/Late = (Mid + Late) / 2; $\%\Delta = ((\text{Mid/Late}\ \bar{x} - \text{Early}\ \bar{x}) / \text{Early}\ \bar{x}) \times 100$). We then correlated the percentage change scores (%Δ) for the autism and neurotypical groups separately. A significant positive correlation demonstrates that greater adaptation from pre-test to post-test is related to becoming more accurate during the blocked acquisition period, whereas a nonsignificant relationship would suggest the blocked trial order during acquisition is not contributing towards imitation learning.

Results

tPHV

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

The second set of separate planned comparisons compared imitation behaviour from the pre-test (random trial order) to middle-acquisition (blocked trial order) for the autism and neurotypical groups. Middle-acquisition was

selected as it was deemed an appropriate stage to examine sensorimotor adaptation following the completion of half of the imitation trials. For the neurotypical group, there was no significant differences in behaviour when imitating either model across the two phases (atypical: $F(1, 38)=0.40$, $p=0.53$, $d=0.14$; typical: $F(1, 38)=0.09$, $p=0.76$, $d=0.10$). The percentage change when imitating the atypical model was %Δ=5 and the typical model was %Δ=2. Although the autism group demonstrated no significant change (%Δ=2) in behaviour when imitating the typical model ($F(1, 38)=0.11$, $p=0.75$, $d=0.08$), there was a significant change (%Δ=17) leading to peak velocity occurring earlier in the movement when imitating the atypical model ($F(1, 38)=9.47$, $p=0.004$, $d=0.66$).

The third set of planned comparisons examined imitation behaviour across acquisition by comparing early-acquisition (blocked trial order) against the pooled behaviour of the middle/late-acquisition (blocked trial order) for the autism and neurotypical groups. There were no significant changes across these phases for the neurotypical group when imitating either model (atypical: $F(1, 38)=0.88$, $p=0.36$, $d=0.08$; typical: $F(1, 38)=0.04$, $p=0.84$, $d=0.09$). The percentage change when imitating the atypical model was %Δ=5 and the typical model was %Δ=1. Although the autism group demonstrated no significant change (%Δ=3) in behaviour when imitating the typical model ($F(1, 38)=0.26$, $p=0.61$, $d=0.14$), there was a significant change (%Δ=9) leading to peak velocity occurring earlier in the movement when imitating the atypical model ($F(1, 38)=4.62$, $p=0.04$, $d=0.31$).

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

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

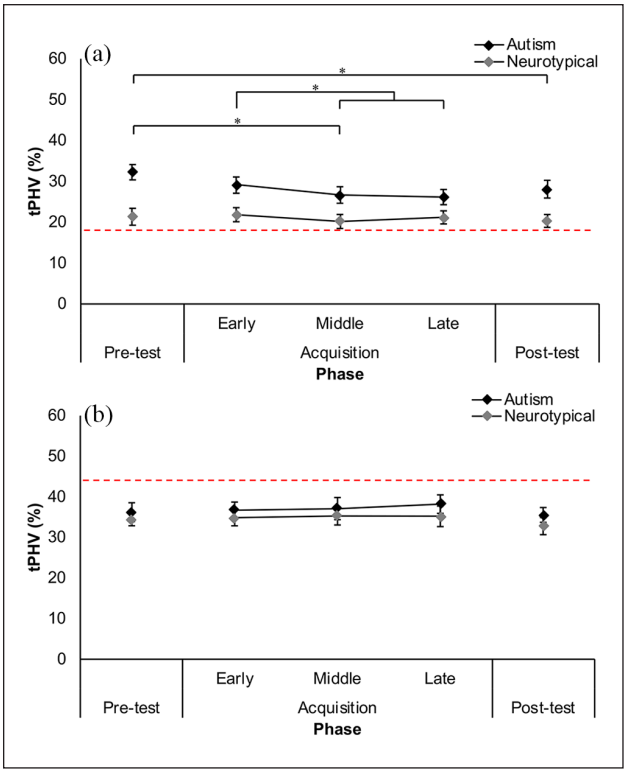

Figure 2. Percentage-time-to-peak-hand-velocity (tPHV) for the imitation task (error bars represent standard error of the mean) presented as a function of group and phase for the (a) atypical model and (b) typical model. Dashed line represents the model.

tPSEV

tPSEV data for both groups across all phases of the imitation learning protocol are illustrated in Figure 3 ((a) autism, (b) neurotypical). The first set of planned comparisons is associated with variance pooled from all phases of the imitation protocol. First, there was no significant difference in tPSEV when examining behaviour at the group level ($F(1, 29)=0.04$, $p=0.84$, $d=0.02$). When examining tPSEV as a function of observing the different models, the autism ($F(1, 29)=169.93$, $p<0.001$, $d=2.81$) and neurotypical ($F(1, 29)=243.44$, $p<0.001$, $d=4.97$) groups showed significant differences in behaviour when observing the atypical (autism $M=31.67 \pm 6$, neurotypical $M=30.37 \pm 4.03$) and typical (autism $M=50.55 \pm 7.55$, neurotypical $M=52.25 \pm 5.03$) models.

The second set of separate planned comparisons compared tPSEV from the pre-test (random trial order) to middle-acquisition (blocked trial order) for the autism and neurotypical groups. There were no significant changes across these phases when observing either model for the

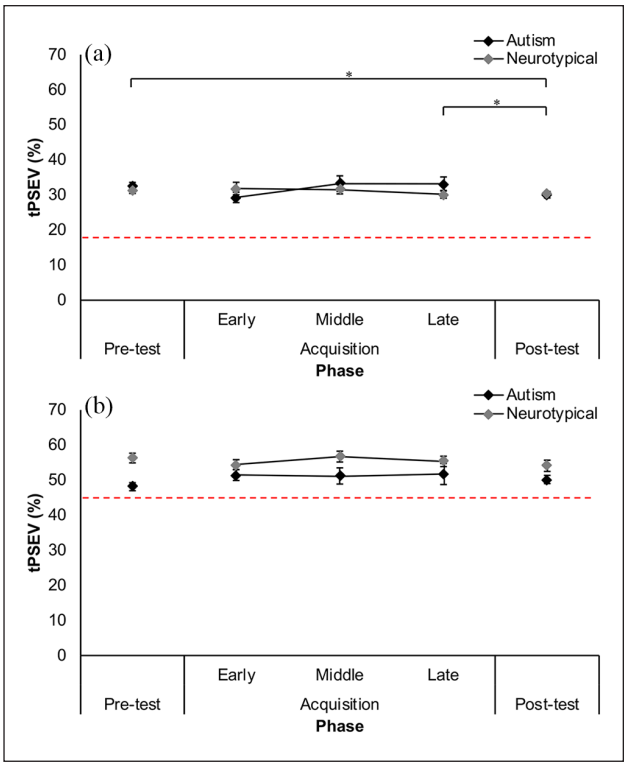

Figure 3. Percentage-time-to-peak-smooth-eye-velocity (tPSEV) for the eye during imitation task (error bars represent standard error of the mean) presented as a function of group and phase for the (a) atypical model and (b) typical model. Dashed line represents the model.

neurotypical group (atypical: $F(1, 29)=0.05$, $p=0.83$, $d=0.10$; typical: $F(1, 29)=0.001$, $p=0.97$, $d=0.01$) or the autism group (atypical: $F(1, 29)=0.18$, $p=0.68$, $d=0.11$; typical: $F(1, 29)=2.31$, $p=0.14$, $d=0.45$). The percentage change for the neurotypical group when observing the atypical model was %Δ=2 and the typical model was %Δ=<1, and for the autism group when observing the atypical model was %Δ=2 and the typical model was %Δ=6.

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

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

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

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

Pearson's correlation analyses indicated a significant relationship between the magnitude of adaptation (%Δ) during acquisition, and magnitude of adaptation (%Δ) from pre-test to post-test, for the imitation of the atypical model by the autism group ($r=0.454$, $p=0.04$; Figure 4(a)) but not the neurotypical group ($r=-0.145$, $p=0.54$; Figure 4(c)). As illustrated in Figure 4, there was a positive relationship whereby autistic participants who demonstrated the greatest (or lowest) magnitude of sensorimotor adaptation across acquisition (see *y*-axis on Figure 4(a)) also exhibited a greater (or lower) change in imitation from pre-test to post-test (see *x*-axis on Figure 4(a)). There were no significant relationships for either group (autism, Figure 4(b): $r=0.053$, $p=0.83$; neurotypical, Figure 4(d): $r=0.390$, $p=0.09$) when imitating the typical model.

Discussion

Although voluntary imitation is generally different in autistic compared to neurotypical individuals (DeMyer et al., 1972; Rogers & Pennington, 1991; Vivanti & Hamilton, 2014), there is evidence that certain general sensorimotor processes underlying imitation are operational (Bird et al., 2007; Hamilton et al., 2007; Hayes, Andrew, et al., 2016). To better understand how sensorimotor processes function

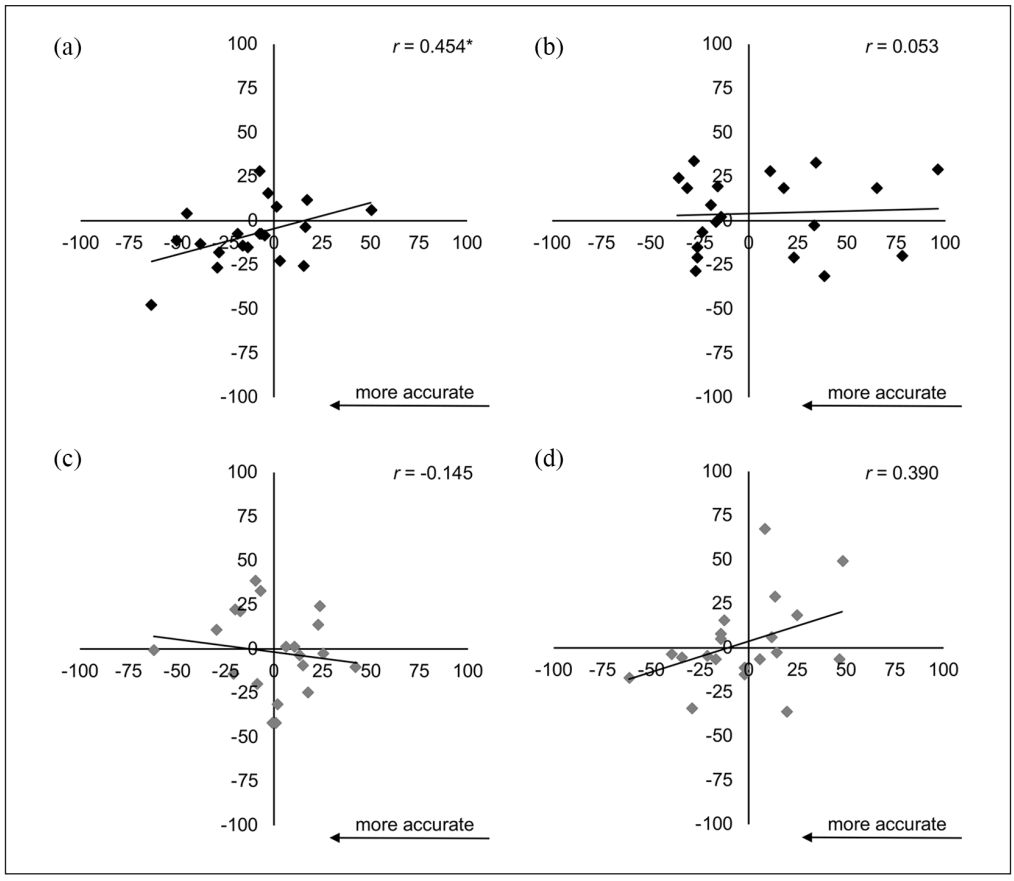

Figure 4. Correlation between the magnitude of sensorimotor adaptation across acquisition (*y*-axis) and the magnitude of sensorimotor adaptation from pre-test to post-test (*x*-axis) when imitating the atypical model ((a) autism, (c) neurotypical) and the typical model ((b) autism, (d) neurotypical).

during the imitation, we examined performance in autistic and neurotypical volunteers following a period of blocked practice at learning to imitate a biological model with an atypical velocity profile. Learning was assessed by measuring performance change from the pre-test to post-test where imitation was performed in random trial orders. Any significant adaptation effects following blocked practice would be generalised to a trial order that was randomised in the post-test.

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

The results from the second and third sets of planned comparisons suggest that the imitation of atypical kinematics demonstrated by the autism group was underpinned by processes that facilitated sensorimotor integration and adaptation across blocked practice (Magill & Hall, 1990). Compared to the neurotypical group that successfully imitated the atypical model at pre-test and middle-acquisition, the second planned comparison indicated the autism group exhibited a significant 17% change (5 units of tPHV: pre-test M = 32, middle M = 27) in imitation behaviour at the middle-acquisition phase of imitating the atypical model during blocked practice. This significant change is important because in our previous work (Hayes, Andrew, et al., 2016) where a group of comparable autistic adults imitated atypical, typical and constant velocity kinematics presented randomly across 84 trials, we showed no adaptation effects across a similar number of practice trials (tPHV: pre-test M = 33, late-phase M = 33). The third planned comparison, which examined changes in imitation from early-acquisition to middle/late-acquisition where trials were received only in a blocked practice trial order, indicated the autism group significantly adapted tPHV by 9%. This finding indicates that the imitation adaptation effects observed in the second and third planned comparisons

were not merely a consequence of switching the learning environment from a randomised to blocked practice trial order. Moreover, the fact that we showed no such change in our previous work (Hayes, Andrew, et al., 2016) indicates that the sensorimotor adaptation effect found here is unlikely to be underpinned by processes governing general practice effects.

Before interpreting the adaptation effects associated with the hand kinematics, it is noteworthy to comment that our measure of smooth pursuit eye velocity (tPSEV) was appropriately scaled to the different models by both groups (atypical: autism M = 32, neurotypical M = 30; typical: autism M = 51, neurotypical M = 52). These data show that the high-acuity region of the fovea was maintained within the vicinity of both models during pursuit (see Lovejoy et al., 2009) and would have provided retinal and extra-retinal input on the observed biological kinematics that could contribute to subsequent configuration of the limb movement. Second, neither group significantly changed tPSEV when imitating the atypical model in the pre-test (random trial order) compared to middle-acquisition (blocked trial order) or from early-acquisition to middle/late-acquisition (NB: both had blocked trial order). At 13%, the change in tPSEV from early-acquisition to middle/late-acquisition for the autism group was close to significance ($p = 0.07$). This change was based on tPSEV occurring later in the middle/late phase (33%) compared to the early phase (29%). Conversely, the significant 9% change in hand kinematics for autism group indicated the opposite effect, with tPHV equal to 26% in the middle/late phase and 29% in the early phase. The implication is that the significant adaptation effect in our measure of hand kinematics by the autism group when imitating the atypical model (across acquisition) is unlikely to simply be related to smooth pursuit eye movements.

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

Further evidence that sensorimotor adaptation was optimised by facilitating the integration and encoding of atypical biological kinematics is apparent from the fourth and fifth sets of planned comparisons. The fourth set indicated no significant changes in behaviour for either group when imitation was compared from late-acquisition (blocked trial order) to the post-test (randomised trial order). This is in contrast to the significant change found in the fifth set, where the autism group successfully imitated the atypical kinematics at post-test compared to pre-test. These combined effects indicate the processing changes that occurred during blocked practice underpinned the encoding of an internal action model that was operational when the autism group was transferred back to a randomised trial order in the post-test. This learning effect and subsequent positive transfer indicate that differences in voluntary imitation in autism (DeMyer et al., 1972; Rogers & Pennington, 1991; Vivanti & Hamilton, 2014) are not solely due to sensorimotor planning problems (Glazebrook et al., 2006; Rinehart et al., 2001) associated with imitating a novel action (Hayes, Andrew, et al., 2016; Stewart et al., 2013; Wild et al., 2012). Rather, it would seem that while the underlying visuomotor system activated during voluntary imitation in autism is functional, operational imitation of atypical biological kinematics requires the practice conditions to be structured optimally (e.g. 'Challenge Point' framework; Guadagnoli & Lee, 2004) in order to facilitate sensorimotor integration and learning.

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

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship and/or publication of this

article: An author of this work has received funding from the European Union's Horizon 2020 research and innovation programme under the Marie Skłodowska-Curie grant agreement number 754490.

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