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RESEARCH ARTICLE

# Observational learning of atypical biological kinematics in autism

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## Abstract

Observing and voluntarily imitating the biological kinematics displayed by a model underpins the acquisition of new motor skills via sensorimotor processes linking perception with action. Differences in voluntary imitation in autism could be related to sensorimotor processing activity during action-observation of biological motion, as well as how sensorimotor integration processing occurs across imitation attempts. Using an observational practice protocol, which minimized the active contribution of the peripheral sensorimotor system, we examined the contribution of sensorimotor processing during action-observation. The data showed that autistic participants imitated both the temporal duration and atypical kinematic profile of the observed movement with a similar level of accuracy as neurotypical participants. These findings suggest the lower-level perception-action processes responsible for encoding biological kinematics during the action-observation phase of imitation are operational in autism. As there was no task-specific engagement of the peripheral sensorimotor system during observational practice, imitation difficulties in autism are most likely underpinned by sensorimotor integration issues related to the processing of efferent and (re)afferent sensorimotor information during trial-to-trial motor execution.

## Lay Summary

Learning a skill by imitating a model (i.e., teacher, parent, or carer) occurs in many everyday situations such as a classroom or home. Imitation can be difficult for some autistic people, especially if a skill is new or complex. These difficulties are said to be based on how autistic people watch a skill and subsequently process what they watched in order to imitate the skill. In this study, we tasked autistic people to learn a new skill by only watching (not imitating) a model during a period of practice. Autistic people imitated how the model moved (movement time and style) similarly to non-autistic people. This finding is very important because it showed that the reported difficulty in imitation is not based on how autistic people watch and process a model (i.e., how they move), but a result of sensory-motor difficulties related to how they plan and get ready to imitate a model. Parents/carers, clinicians, teachers, and/or support workers should therefore consider autistic motor difficulties when teaching new motor skills and everyday skills via modeling.

**KEYWORDS**
autism spectrum disorder, biological motion kinematics, imitation, observational practice, sensorimotor integration

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## INTRODUCTION

Humans have an exceptional ability to learn sensorimotor behaviors by observing and then imitating another person (i.e., a model) performing a movement. Voluntary imitation (henceforth imitation) enables new or adapted internal action-plans to be represented by engaging sensorimotor, attentional, and intentional processes (Iacoboni, 2009; Keysers et al., 2018; Schaal et al., 2003). When observing to imitate a model, individuals represent both the displayed biological motion properties and the action-goal in a sensorimotor system linking perception with action (Becchio et al., 2018; Buccino et al., 2004; Prinz, 1997). This system is tuned to agents that display a biological (e.g., human) origin (Press et al., 2011), including kinematics (Candidi et al., 2008) and form (Brass et al., 2001) and controls the processing of task-relevant biological information (i.e., velocity, grip aperture), with the resulting representations being used by sensorimotor planning processes to generate an action-plan (Wolpert & Kawato, 1998) to reproduce the observed action. During motor reproduction, the expected sensorimotor consequences (efference) of an action and the actual sensory feedback (reafference) are integrated and processed by feedforward and feedback control mechanisms to organize sensorimotor control, and to consolidate an executed sensorimotor action. Across repeated imitation attempts, the predicted and actual movement outcomes are compared leading to updated sensorimotor action-plans (Buccino et al., 2004; Carroll & Bandura, 1990) that underpin the reproduction of movements that are similar to the observed biological kinematics displayed by a model.

Although the processes subserving imitation are operational across typical development (Anisfeld, 2005; Jones, 2009; Ray & Heyes, 2011), autistic individuals can exhibit processing differences that lead to specific motor imitation (Andrew et al., 2016; DeMyer et al., 1972; Hobson & Lee, 1999; Rogers et al., 1996; Stewart et al., 2013; Tunçgenç et al., 2021; Wild et al., 2012) difficulties when reproducing lower-level biological kinematics displayed by a model (note, other forms of imitation such as automatic imitation are operational in autism; see de Hamilton et al., 2007; Vanvuchelen et al., 2013). These differences are likely to be underpinned by a network of interacting processes, such that autistic participants can sometimes show less perceptual sensitivity to the underlying biological motion properties (e.g., velocity) of an observed movement (Cook et al., 2009). Processing differences include visual attention (Klin et al., 2009), social cognition (de Hamilton, 2008; Klin & Jones, 2008; Spengler et al., 2010; Tunçgenç et al., 2021), and sensorimotor control (Cook et al., 2013).

Importantly, however, imitation of biological motion in autism is still attenuated when modulatory factors related to social cognition (e.g., the model is a single point-light dot) and goal-directed imitation are controlled (i.e., the model does not perform a movement to an end-state target). This can be seen in a study where we examined imitation of biological kinematics using two single point-light models that displayed the same movement amplitude (200 mm) and absolute movement time (1700 ms), but different underlying biological kinematics (Hayes et al., 2016a). One model displayed typical kinematics where peak velocity occurred at 44% of the movement trajectory (bell-shaped profile), whereas the other model displayed atypical kinematics with peak velocity occurring at 18% of the movement trajectory. The typical kinematics could be imitated by rescaling a movement from an existing motor repertoire, whereas the atypical kinematics required participants to observe and represent the atypical kinematics to reorganize the sensorimotor system for trial-to-trial movement reproduction. Both the autistic and neurotypical groups successfully reproduced the absolute movement time goal, which indicated they attended to the modeled stimulus and learned to imitate the absolute timing parameter (de Hamilton et al., 2007; Vivanti & Hamilton, 2014). However, the autism group was significantly less accurate at imitating the novel atypical biological kinematics.

The methodology used by Hayes et al. (2016a) did not enable us to determine whether attenuated imitation was underpinned by sensorimotor processing difficulties or less effective sensorimotor integration at the single-trial level. Specifically, the two models were presented in a randomized trial order where sensorimotor information on trial $n$ (e.g., *atypical* model) could be different to trial $n + 1$ (e.g., *typical* model). Accordingly, the underlying sensorimotor control processes engaged to compare efferent and reafferent sensorimotor information to update a sensorimotor action-plan (Buccino et al., 2004; Carroll & Bandura, 1990) may have operated less effectively in autism when the subsequent trial (trial $n + 1$) was different to trial $n$. This may have negatively impacted upon sensorimotor integration (Lidstone & Mostofsky, 2021; Marko et al., 2015; Nebel et al., 2016) and led to the development of a less effective action-plan used for planning and controlling movement reproduction. This was confirmed in a subsequent study (Foster et al., 2020b) that used a blocked-practice trial order (Shea & Morgan, 1979) whereby participants imitated the same model (i.e., *atypical* model) across all practice trials (i.e., trial $n$ was the same as $n + 1$). With repeated practice attempts, the autism group became significantly more accurate at imitating the atypical kinematics, which suggested imitation was enhanced by having an opportunity to refine and update an action-plan on a trial-by-trial basis. However, they remained significantly less accurate at imitating the atypical kinematics than the neurotypical group, despite exhibiting similar smooth pursuit eye movements when overtly attending to the model during action-observation. It therefore seems reasonable to suggest that other perception-action processes may be contributing to attenuated imitation in autism. In particular,

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it could be that autistic participants exhibit difficulties in sensorimotor integration related to the processing of efferent and (re)afferent information during trial-to-trial motor execution.

Here, then, we used an observational practice protocol to investigate the operational nature of processes underpinning the representation of atypical biological kinematics in autism. Unlike the imitation protocol used in our previous work, which involved action-observation followed by movement reproduction, observational practice requires participants to learn only by observing a novel stimuli across practice trials (Bird et al., 2005; Mattar & Gribble, 2005; Vogt, 1995). In this context, there is no requirement to physically reproduce the observed movement during learning, thus enabling us to directly examine the imitation of atypical biological kinematics based solely on the contribution of the perception-action processes engaged during action-observation. If the neural circuitry encoding biological kinematics operates differently in autism (Williams et al., 2001, 2004), we expect an autism group to be significantly less accurate at reproducing the atypical biological kinematics than the neurotypical group. If previously reported autistic differences in imitation are the result of a sensorimotor system that disrupts the integration of a visual input and motor output, we expect no significant difference between the autism and neurotypical groups at reproducing the atypical biological kinematics as there is no requirement to physically reproduce the model during observational practice.

# METHOD

## Participants

An opportunity sample of autistic volunteers was recruited from an autism society in the United Kingdom, and the host University. Volunteers were provided with a participant information sheet to read, followed by an opportunity to ask questions to clarify the experimental procedures, and then a period to consider whether they would like to consent to participate. Twenty autistic (2 female; 18 male) volunteers participated in the study. Following the autism recruitment, we used IQ, age and gender to recruit and match 20 neurotypical (2 female; 18 male) volunteers from a host University. Data on socioeconomic status and educational attainment levels were not recorded. All participants were screened via 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). All autistic participants met the threshold for autism spectrum disorder on the ADOS-2 total classification score, and the communication, and social interaction subscales. Groups were equated for age, as well as full-scale, verbal, and performance IQ as measured via the Wechsler Abbreviated Scale of Intelligence (WASI). Group characteristics are presented in Table 1.

## 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. The monitor was connected to a desktop PC (HP Compaq 8000 Elite), which received input from a hand-held stylus on a graphics tablet (Wacom Intuos Pro XL). Experimental stimuli were generated on the desktop PC using the COGENT toolbox (developed by John Romaya at the Laboratory of Neurobiology at the Wellcome Department of Imaging Neuroscience) implemented in MATLAB (Mathworks, Inc.). 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 synchronized using a TTL signal.

## Stimuli

Participants observed two prerecorded models in which a single white dot (diameter = 6.25 mm) moved along a horizontal trajectory of 200 mm in a criterion movement time of 1700 ms, but with either a *typical or atypical* velocity profiles (see Figure 1a,b). The two models were created by a human volunteer who practiced performing

**TABLE 1** Characteristics of autism and neurotypical participants.

| | Autism ($n = 20$) | | Neurotypical ($n = 20$) | | |
|---|---|---|---|---|---|
| | Mean (*SD*) | Range | Mean (*SD*) | Range | *t* Test *p* value |
| Chronological age in years | 25 (7) | 18–44 | 25 (7) | 18–45 | $p = 0.845$ |
| Full scale IQ | 107 (9) | 91–125 | 109 (8) | 94–123 | $p = 0.396$ |
| Verbal IQ | 106 (11) | 88–130 | 109 (8) | 96–125 | $p = 0.214$ |
| Performance IQ | 106 (11) | 82–128 | 107 (12) | 82–128 | $p = 0.891$ |
| Sex assigned at birth | 18M: 2F | | 18M: 2F | | |

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the movements using a hand-held stylus on a graphics tablet (see Foster et al., 2020b), and thus displayed human biological motion. For the *typical* model, the volunteer practiced performing unconstrained goal-directed aiming movements that created a typical bell-shaped velocity profile (Flash & Hogan, 1985) where the peak occurred at 44% of the movement trajectory, and had a magnitude of 0.19 mm/ms (dashed trace in Figure 1a). For the *atypical* model (see Hayes et al., 2014 for a description of the method of creating atypical models), the volunteer practiced performing novel atypical movements to create a positively skewed velocity profile (black trace in Figure 1a) where the peak occurred at 18% of the movement trajectory and had a magnitude of 0.33 mm/ms. We did not present end-state targets in order to constrain observational practice to processes related to representing biological kinematics, rather than being influenced by top-down processes engaged to achieve the end-state goal (Bekkering et al., 2000; Hayes et al., 2016b).

## Procedure

The experimental procedure was refined via participatory research. Six adult autistic advocates, with previous experience in supporting our research (see Foster et al., 2020b), gave feedback on the current study to help shape the experimental set-up, task instructions, and how the participant information sheets were created to ensure participants were able to make an informed decision to consent.

Before participating, all participants completed a familiarization period that replicated the general methodological conditions used in the main experiment. Participants performed four imitation trials, each showing a constant velocity stimulus moving with the same movement duration (1700 ms) and amplitude (200 mm) as the experimental models. Importantly, velocity in the horizontal $x$ axis was constant at 0.12 mm/ms, with no deviations in the perpendicular $y$ axis. This model ensured the construct validity by preventing participants from experiencing biological kinematics before the experimental trials. Participants were instructed to observe the horizontal trajectory of the model with the intention to overtly reproduce the movement following action-observation. They were not informed about the movement duration or the nature of the stimulus. To imitate the model, participants moved the stylus on the tablet so that the cursor moved from the home-position to the end-position as per the movement displayed by the model. All participants confirmed they observed the

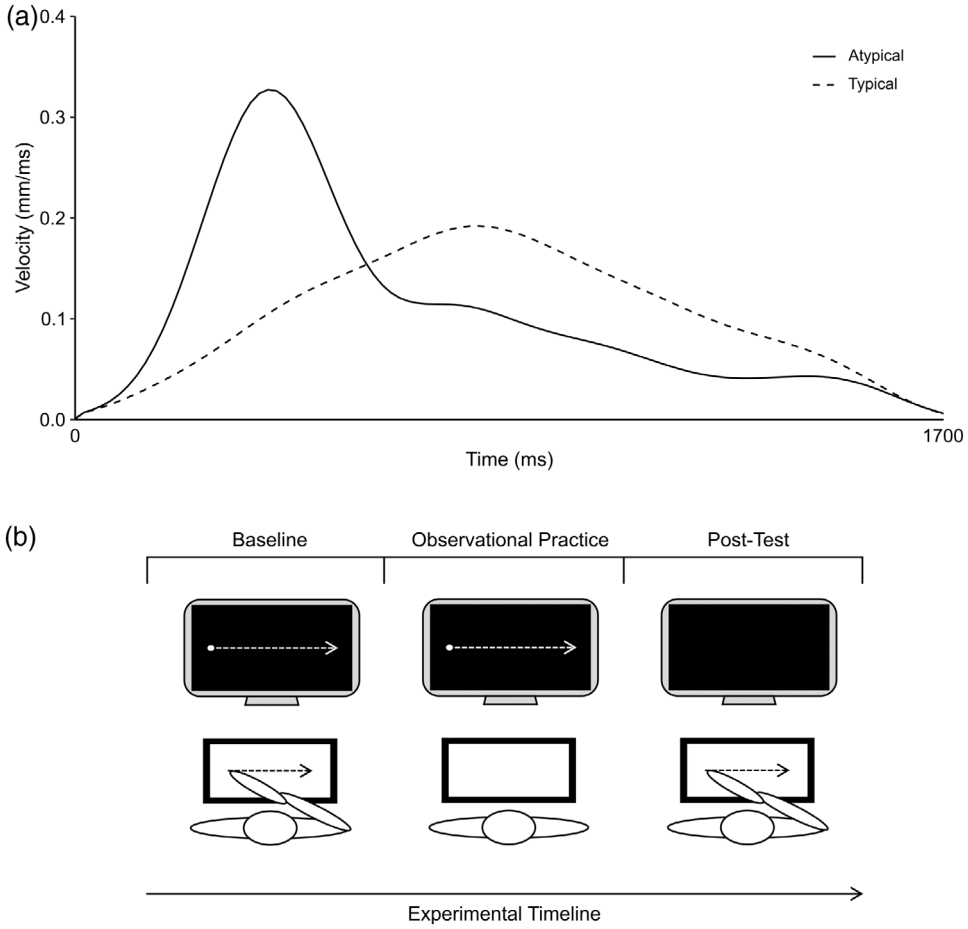


**FIGURE 1** (a) Displacement time-series displaying typical (dashed trace) and atypical (black trace) models. (b) A schematic representation of the experimental set-up and design. The black outlined rectangle represents a graphics tablet. The white circle displayed on the CRT monitor represents a point-light dot model. The single-segment movement displayed by a model is depicted by the arrow (i.e., from the home-position to the end-position). During baseline, participants observed and reproduced a model displaying a typical model. During observational-practice, participants observed a model displaying an atypical model. During post-test, participants recalled and reproduced the movement displayed by the atypical model during observational practice.

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model, understood the instructions on how to imitate the model, and the sensorimotor association between the stylus on a graphics tablet and the corresponding movement of the cursor on the monitor.

The experiment consisted of a 3-phase adapted version of an observational practice protocol used by Foster et al. (2018), whereby participants performed a baseline, observational practice, and a post-test (see Figure 1b). The baseline consisted of 10 imitation trials, where on each trial participants were instructed to observe the horizontal trajectory of the *typical* model with the intention to overtly reproduce the movement following action-observation. No information was presented to the participants regarding the nature of the *typical* model. We made the decision not to baseline using the *atypical* model because we wanted to control the influence of any carry-over effects. Specifically, using the *atypical* model at baseline would have influenced the subsequent observational practice as participants would already have had an opportunity to observe (i.e., action-observation) and imitate (i.e., motor-execution) the *atypical* model. The observational practice phase consisted of 30 consecutive action-observation trials where on each trial participants were instructed to watch the movement trajectory of the *atypical* model very carefully, with the intention to reproduce the movement in the post-test. The post-test consisted of 10 trials and participants were instructed to reproduce the *atypical* model from memory (participants did not observe the model) using the stylus on the graphics tablet. Following the post-test, all participants completed a verbal debrief session to confirm they distinguished the difference between the two experimental models and that they had intended to reproduce the *atypical* velocity profile as accurately as possible in the post-test. Eye movements of all participants were recorded as they observed the two stimuli during the experiment (40 trials). Some data were subsequently excluded (7 autism: 2 neurotypical) due to recording difficulties resulting from participants wearing prescription spectacles.

## Data reduction

### Hand movement data

Using a custom written MATLAB routine, we identified the start and end of each movement reproduction from the *x*-axis position data. The start of a movement was defined as the moment the center of the cursor moved beyond the perimeter of the home-position, whereas movement end corresponded to the moment the participant clicked the button on the stylus. Using these moments, we then extracted the time-series position data for each baseline, and post-test trial. The position data for each trial were processed using a low-pass 4th order autoregressive filter with an 8 Hz cut-off (Foster et al., 2018, 2020b). The filter minimized measurement noise in the experimental time-series data before differentiation using a 2-point central difference algorithm to obtain velocity.

To quantify movement reproduction during action-execution, movement duration (*MT*) from the time-series data was extracted from each trial (baseline, and post-test). Further analysis of movement kinematics was focused on the *x*-axis data only, which reflected the primary movement axis. A MATLAB routine extracted *percentage-time-to-peak-hand-velocity* (*tPHV*) from each trial. This kinematic variable was chosen as it provides a discrete measure that reflects whether participants accurately reproduced the timing characteristics (peak velocity) of the observed movement (Hayes et al., 2014). Additionally, we calculated root-mean-square error (*RMSE*) of the hand velocity data as a measure of overall reproduction error across the entire movement trajectory. A MATLAB routine resampled the hand velocity time-series data from each trial, and the two experimental models, to 100 data points. The routine then calculated the root-mean-squared deviations for each trial between the 100 points associated with a performed movement trajectory and the 100 points associated with the respective model. For each trial, an error score was generated, where perfect reproduction returns a value of 0.

### Eye movement data

To quantify eye movements during action-observation of the baseline and the subsequent observational practice, we focused the analysis on the *x*-axis data taken from the left-eye. Synchronization signals (TTL) generated by the COGENT toolbox at the start and end of stimulus presentation were used to identify the corresponding eye movement. 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. The de-saccaded smooth eye velocity was then low-pass filtered using a moving average zero-phase filter (40 ms window). To quantify how well eye velocity matched the velocity trajectory of the observed model, we extracted *percentage-time-to-peak-smooth-eye-velocity* (tPSEV) for each trial.

## Data analysis

To account for individual differences, we examined trial-level motor (*MT, tPHV*, and *RMSE*) and ocular (*tPSEV*) behavior as a function of observational practice

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using a mixed effects modeling approach ("glmmTMB" R package; Brooks et al., 2017). For measures of motor behavior, the fixed effects were *phase* (baseline, post-test) and *group* (autism and neurotypical), whereas for ocular behavior the fixed effect were *phase* (baseline, early [first 10 trials of observational practice], late [last 10 trials of observational practice]) and *group* (autism and neurotypical). We included all main and interaction fixed-effect terms, as well as a random slope for phase and intercept, although the slope was removed if the model failed to converge. For *MT*, we fitted a linear mixed model, whereas for *tPHV*, *RMSE*, *and tPSEV* we fitted a generalized linear mixed model (gamma family with log link). Gamma distributions model positively skewed, non-negative data (Ng & Cribbie, 2017) and produced better performance both in terms of log-likelihood and BIC than other distributions that allow for skewness (e.g., inverse Gaussian, lognormal, etc.). The fit of the full model was compared to an intercept-only model using the likelihood ratio test (LRT) and pseudo *R*-squared (r2_nakagawa function, "performance" R package; Lüdecke et al., 2021).The significance of fixed effects in the selected model were determined using type III ANOVA (Wald approximation; *Anova* function, "car" R package; Fox & Weisberg, 2019). Significant main and/or interaction effects were then investigated using Holm–Bonferroni adjusted pairwise comparisons ("emmeans" R package; results are presented as estimated marginal means ± standard error).

# RESULTS

## Hand movement data

The full model [MT ~ phase*group+(phase|participant)] fit significantly better than the intercept-only model [LRT: $\chi^2 = 214.81$, $df = 2$, $p < 0.001$] and had a total explanatory power of $R^2_{\text{conditional}} = 0.66$. There was no significant main effect of group [$\chi^2 = 0.94$, $df = 1$, $p = 0.33$] or phase × group interaction [$\chi^2 = 1.78$, $df = 1$, $p = 0.18$]. The significant main effect of phase [$\chi^2 = 13.12$, $df = 1$, $p < 0.001$] indicated movement time was shorter by 246.43 ms in the post-test (1779.72 ± 66.73 ms) compared with baseline (2026.14 ± 58.67 ms). As illustrated in Figure 2a, the autism group (gray line) improved motor

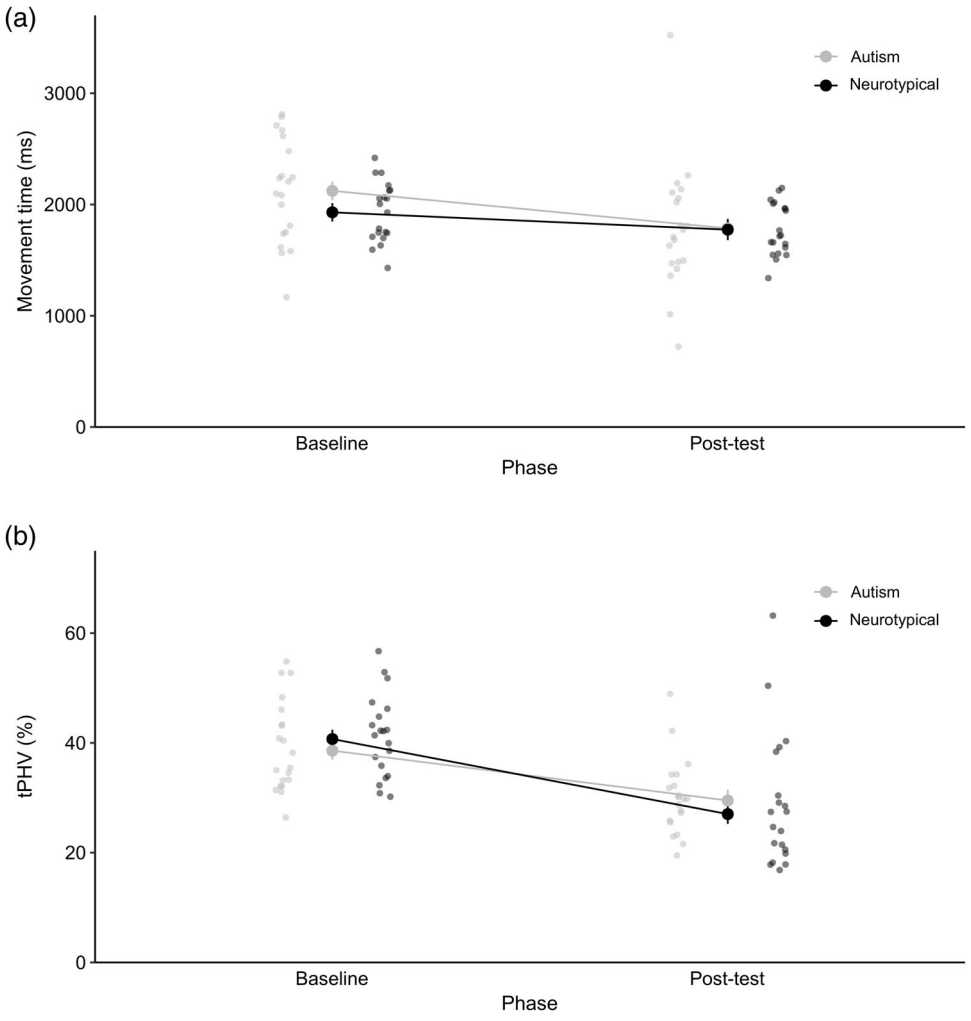


**FIGURE 2** (a) Movement time as a function of group and phase. (b) Percentage-time-to-peak-hand velocity as a function of group and phase. The plots show the estimated marginal mean ± SE from the full mixed-effect model fits to single trial data. Individual data points represent single subject means.

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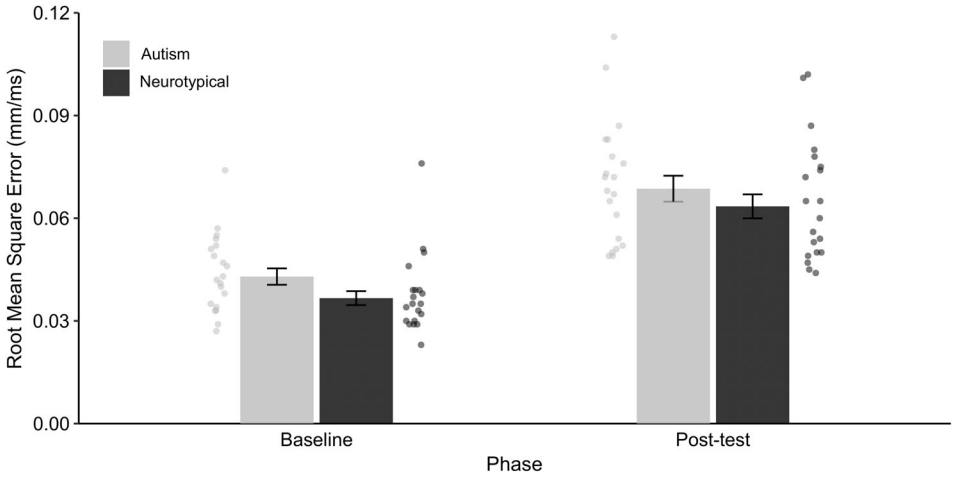


**FIGURE 3** Root mean square error as a function of group and phase. The plot shows the estimated marginal mean ± SE from the full mixed-effect model fit to single trial data. Individual data points represent single subject means.

performance by 16% as MT decreased from 2122.75 ± 82.96 ms at baseline to 1785.57 ± 94.39 ms in the post-test. The neurotypical group (black line) improved motor performance by 8% as MT decreased from 1929.53 ± 82.98 ms to 1773.87 ± 94.35 ms.

The full model [tPHV ~ phase × group + (phase|participant)] fit significantly better than the intercept-only model [LRT: $\chi^2 = 159.49$, $df = 2$, $p < 0.001$] and had a total explanatory power of $R^2_{\text{conditional}} = 0.54$. There was no significant main effect of group [$\chi^2 = 0.09$, $df = 1$, $p = 0.76$] or phase × group interaction [$\chi^2 = 1.76$, $df = 1$, $p = 0.18$]. The main effect of phase was significant [$\chi^2 = 40.49$, $df = 1$, $p < 0.001$] and indicated peak velocity occurred 11.38 ± 1.70 units earlier in the movement trajectory in the post-test (28.27 ± 1.32) compared with baseline (39.64 ± 1.18). As illustrated in Figure 2b, the autism group changed by 24% from *tPHV* of 38.58 ± 1.62 at baseline to 29.51 ± 1.95 in the post-test. The neurotypical group changed by 34% from *tPHV* of 40.70 ± 1.71 at baseline to 27.03 ± 1.79 in the post-test.

The full model [RMSE ~ phase × group + (phase|participant)] fit significantly better than the intercept-only model [LRT: $\chi^2 = 144.83$, $df = 2$, $p < 0.001$] and had a total explanatory power of $R^2_{\text{conditional}} = 0.63$. There was no significant phase × group interaction [$\chi^2 = 0.59$, $df = 1$, $p = 0.44$]. There was a significant main effect of group [$\chi^2 = 4.10$, $df = 1$, $p = 0.04$], although post hoc testing on the response scale was not significant ($RMSE_{ASD} - RMSE_{TD} = 0.006$; SE = 0.003; $z = 1.81$; $p = 0.07$). The main effect for phase was significant [$\chi^2 = 94.48$, $df = 1$, $p < 0.001$] and indicated RMSE was 0.026 ± 0.003 greater in the post-test (0.066 ± 0.003) compared with baseline (0.040 ± 0.002). As shown in Figure 3, the autism group changed by 60% from baseline (0.043 ± 0.002) to post-test (0.069 ± 0.004). While the neurotypical group changed by 73% from baseline (0.037 ± 0.002) to post-test (0.063 ± 0.004). As illustrated in Figure 4a, at baseline, both groups (autism = gray trace; neurotypical = black trace) executed bell-shaped velocity profiles that were like the velocity profile displayed by the typical model (red trace). During the post-test (Figure 4b), both groups (autism: gray trace; neurotypical: black trace) executed velocity profiles where peak velocity occurred earlier in the movement trajectory and therefore more like the velocity profile displayed by the atypical model (red trace).

## Eye movement data

The full mixed model with a random intercept [tPSEV ~ phase × group + (1|participant)] had the highest total explanatory power with $R^2_{\text{conditional}} = 0.54$. There was no significant main effect of group [$\chi^2 = 0.35$, $df = 1$, $p = 0.56$] or phase × group interaction [$\chi^2 = 2.27$, $df = 2$, $p = 0.32$], but there was a significant main effect of phase [$\chi^2 = 963.36$, $df = 2$, $p < 0.001$]. Post hoc testing indicated that *tPSEV* decreased by 20.71 ± 0.82 from 50.30 ± 0.85 at baseline to 29.59 ± 0.51 in the early phase of observational practice ($z = 25.27$; $p < 0.001$). There was no difference in *tPSEV* between the early phase and late phase (30.52 ± 0.52) of observational practice ($z = -1.60$; $p = 0.11$). As illustrated in Figure 5, *tPSEV* of the autism group changed from 49.85 ± 1.29 at baseline to 30.12 ± 0.78 in the early phase of observational practice. For the neurotypical group, *tPSEV* changed from 50.75 ± 1.11 at baseline to 29.10 ± 0.64 in the early phase of observational practice. At the late phase of observational practice, *tPSEV* was 30.94 ± 0.80 in the autism group and 30.10 ± 0.66 in the neurotypical was.

# DISCUSSION

We used an observational practice protocol to investigate the operational nature of processes engaged during action-observation to represent, and reproduce, atypical biological kinematics in autism. Consistent with our previous imitation studies in autism (Foster et al., 2020b; Hayes et al., 2016a), which used the same stimuli, there was no significant group difference in smooth pursuit eye

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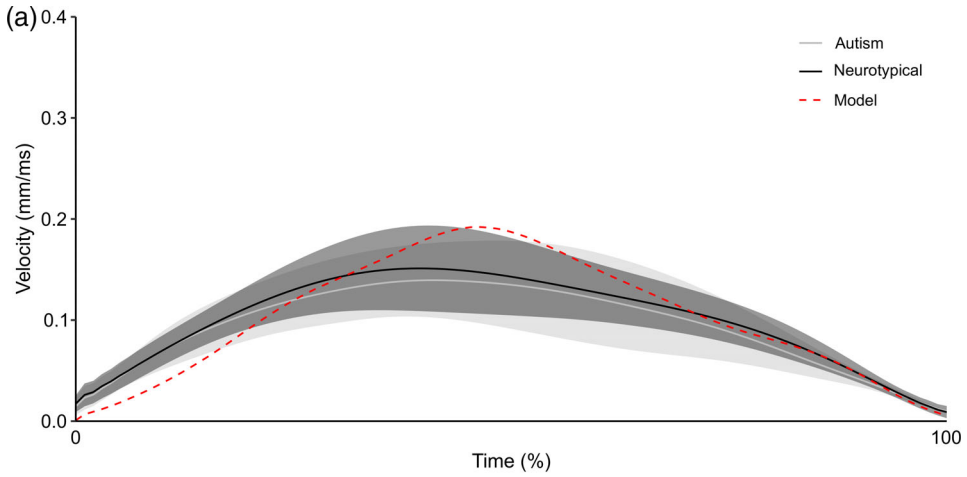


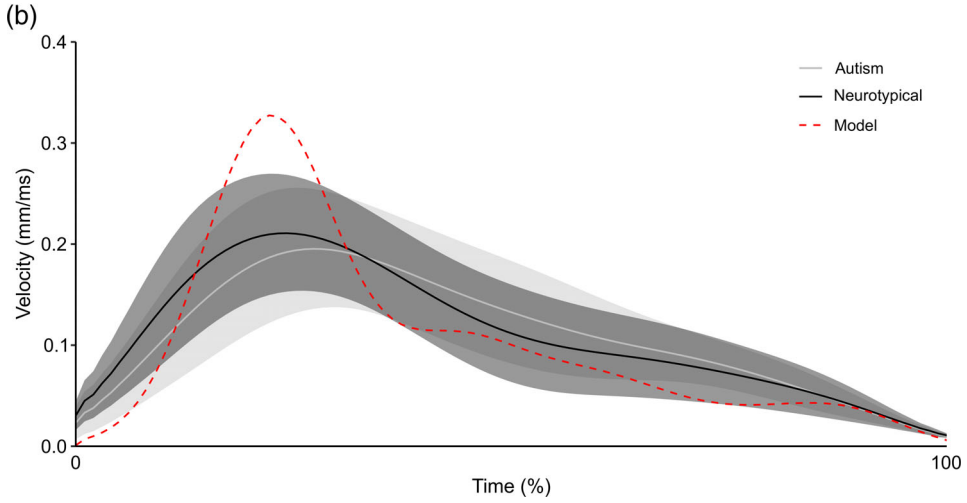


**FIGURE 4** Normalized displacement time-series and standard deviation (shaded area) for the autism (gray line) and neurotypical (black line) groups during baseline (a) and the post-test (b). The observed model is represented by the dashed red line.

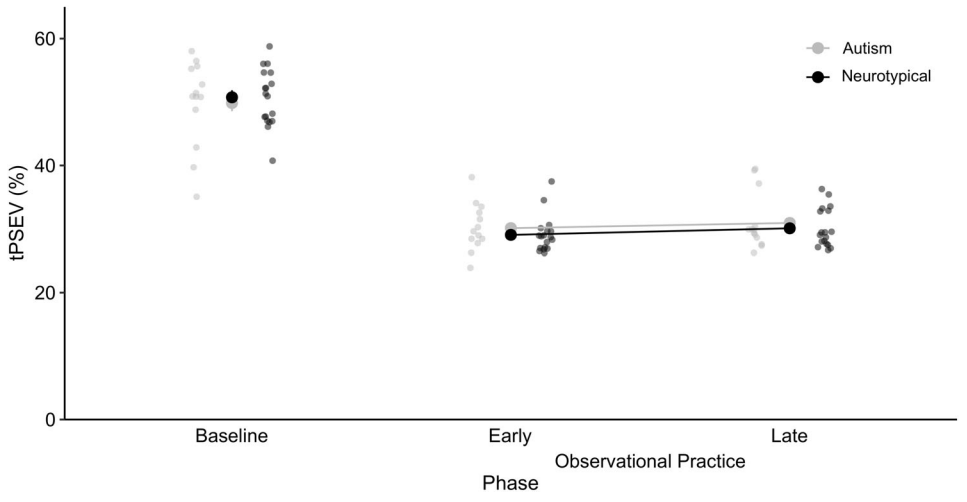


**FIGURE 5** Percentage-time-to-peak-smooth-eye-velocity as a function of group and phase. The plot shows the estimated marginal mean ± SE from the intercept only mixed-effect model fit to single trial data. Individual data points represent single subject means.

velocity when observing the typical and atypical models. For both groups, there was a 20% change in the time of peak smooth eye velocity (*tPSEV*) from viewing the *typical* model at baseline to the *atypical* model at the early and late phases of observational practice. This task-specific scaling of smooth pursuit to the *atypical* model suggests that overt visual attention was located close to the moving trajectory displayed by the model (Lovejoy et al., 2009), thus ensuring access to retinal and extra-retinal input (Leigh & Zee, 2015) required to process and represent the atypical biological kinematics as a sensorimotor action-plan. The effectiveness of observational practice for subsequent planning and control of movement reproduction (Desmurget & Grafton, 2000;

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Elliott et al., 2010; Wolpert et al., 2011) was evident in the movement time data, which indicated both groups significantly improved motor timing performance in the post-test (autism group reduced MT by 18%; neurotypical group by 8%). Similar improvements in motor timing performance are reported in autism following imitation (Hayes et al., 2016a), or motor learning (Hayes et al., 2018) with augmented knowledge-of-results (Bilodeau & Bilodeau, 1958) presented after each trial.

In addition to changes in movement time, the hand kinematic data indicated that both groups reproduced movements in the post-test where peak velocity occurred significantly earlier in the movement trajectory (autism = 30%; neurotypical = 27%) compared with baseline (autism = 39%; neurotypical = 41%). This was also evident across the entire movement trajectory, with both groups demonstrating a higher but similar *RMSE* when reproducing the *atypical* kinematics in the post-test compared with *typical* kinematics at baseline. The data illustrates that both groups had more difficulty reproducing the novel *atypical* model (Rokni et al., 2007) but importantly they still exhibited mean velocity traces with a positive skew in the timing of peak velocity (Figure 4b). A further way to examine the acquisition of atypical kinematics would be to modify our protocol so that movements at baseline are quantified from executing atypical kinematics, rather than typical kinematics, as per the current study. This modified experimental design would allow the direct examination of behavior change as a function of learning of a novel atypical stimulus via observational practice. In terms of our current results, the data for the neurotypical group is consistent with our previous finding (Andrew et al., 2016; Hayes et al., 2014; Hayes et al., 2016b) that neurotypical learners integrate visual information within a perception-action matching mechanism (Prinz, 1997) containing visual-motor integration processes (Bird et al., 2005; Cross et al., 2009; Higuchi et al., 2012; McGregor & Gribble, 2015). These processes underpin the representation of *atypical* biological kinematics as an action-plan that controls subsequent movement reproduction. The fact that the autism group reproduced the observed atypical kinematics in the post-test suggests the aforementioned visual-motor integration processes recruited during observational practice (Cross et al., 2009; Higuchi et al., 2012) were operational in our group of autistic adults that did not present with other neurological conditions or learning difficulties. Together, our findings add to evidence (Bastiaansen et al., 2011; Dinstein et al., 2010; Enticott et al., 2013; Fan et al., 2010; Marsh & de Hamilton, 2011; Pokorny et al., 2015; Raymaekers et al., 2009; Sowden et al., 2016) that there is not a global deficit (cf. Oberman & Ramachandran, 2007; Williams et al., 2001, 2004) of the action-observation network processing in autism.

It is important to appraise how our experimental protocol (i.e., observational practice of a non-social, atypical model) offered new opportunities for examining motor imitation. First, the learning effects occurred following the use of a single point-light dot model that displayed no social characteristics. In this context, we controlled the presence of social top-down signals that can differentially modulate how sensorimotor information is processed and represented during imitation (Cook & Bird, 2012; de Hamilton, 2008; Spengler et al., 2010; Tunçgenç et al., 2021). Although we found operational processing of *atypical* kinematics, additional research should be designed to gain a broader understanding of how the action-observation processes operate when autistic individuals learn to imitate biological kinematics displayed by different social agents (e.g., parent, teacher, sibling) across various contexts (e.g., home, school, nursery) and for learning different motor skills (e.g., play, fundamental movement skills, handwriting). Second, our effects resulted from a period of observational practice, which removed the task-specific sensorimotor efference and reafference (Hayes et al., 2014; Mattar & Gribble, 2005) known to modulate sensorimotor integration leading to atypical motor learning (Dowell et al., 2009; Foster et al., 2020a) and motor imitation (Hayes et al., 2016a; Foster et al., 2020b). It is therefore important to establish whether motor imitation difficulties in autism can be targeted by structuring autism-specific learning protocols so that observational practice precedes blocked-practice imitation (Foster et al., 2020b). This schedule of practice should provide learners with an opportunity to gain the additive effects of observational practice and imitation learning (see Deakin & Proteau, 2000; Shea et al., 2000). We are mindful, however, that our opportunity sample of 20 volunteers were predominantly male autistic adults without language or cognitive impairment. To be able to generalize across a broader spectrum of autistic individuals, we need to gain a better understanding of the benefits of autism-specific learning protocols by widening participation to include volunteers from under represented genders (Tubío-Fungueiriño et al., 2021), those with learning difficulties, severity levels, and those who are non-verbal (Tager-Flusberg & Kasari, 2013).

To conclude, the autism and neurotypical groups showed comparable motor timing and movement reproduction of *atypical* biological kinematics following observational practice of point-light models with no social characteristics. By isolating the effects to the perception-action processes that represent atypical biological kinematics, we question the assumption of a global deficit in action-observation network processing that generally affects motor imitation in autism. The acquisition of new internal action-plans that controlled the execution of novel atypical movements provides new insights into the pervasive sensorimotor integration differences often reported in motor imitation in autism.

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## DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

## ETHICS STATEMENT

The experiment was designed in accordance with the principals of the Declaration of Helsinki (2013) and was approved by the University research ethics committee.

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