

Gesture Imitation in Schizophrenia

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Recent evidence suggests that individuals with schizophrenia (SZ) are impaired in their ability to imitate gestures and movements generated by others. This impairment in imitation may be linked to difficulties in generating and maintaining internal representations in working memory (WM). We used a novel quantitative technique to investigate the relationship between WM and imitation ability. SZ outpatients and demographically matched healthy control (HC) participants imitated hand gestures. In Experiment 1, participants imitated single gestures. In Experiment 2, they imitated sequences of 2 gestures, either while viewing the gesture online or after a short delay that forced the use of WM. In Experiment 1, imitation errors were increased in SZ compared with HC. Experiment 2 revealed a significant interaction between imitation ability and WM. SZ produced more errors and required more time to imitate when that imitation depended upon WM compared with HC. Moreover, impaired imitation from WM was significantly correlated with the severity of negative symptoms but not with positive symptoms. In sum, gesture imitation was impaired in schizophrenia, especially when the production of an imitation depended upon WM and when an imitation entailed multiple actions. Such a deficit may have downstream consequences for new skill learning.

Key words: Working Memory/Social cognition/Sequence learning/Mirror Mechanism/Simulation

Introduction

Imitation plays a key role in the acquisition and fine-tuning of many competencies including motor skills and language. It also plays a central role in understanding the goals, intentions, and desires of others.¹ In imitation, the observation of another's action is used to organize a matching action by the observer.^{2,3} It has been suggested that this correspondence problem is mediated

by the “mirror system,”⁴ comprising neurons that fire both when an action is performed and when a similar or identical action is observed.⁵ In humans, the mirror system includes a network of premotor and parietal regions.^{6,7}

Impaired imitation ability and its consequences have been extensively explored in autism,⁸ and the role of disturbed imitation in other psychiatric disorders has also been noted.⁹ However, to date imitation ability in schizophrenia has not been systematically examined. The few previous studies that included imitation tasks have been motivated by known social impairments in schizophrenia¹⁰ or have examined imitation in the context of emotion expression,^{11,12} but imitation itself was not the main focus of these studies. Imitation and its potential importance to schizophrenia were addressed explicitly for the first time in a recent study,¹³ in which 3 types of imitation were examined: hand and mouth movements and facial expressions. They found that imitation impairments in schizophrenia were pervasive and not limited to social and emotional contexts. While it is not surprising that schizophrenia patients would have difficulty in imitating emotional facial expressions, it was noteworthy that they also failed to imitate simple manual gestures and mouth movements. Based on these results, Park et al¹³ suggested that the imitation deficit in schizophrenia was extensive and could account for a wide range of social impairments. However, there are several unresolved issues that must be addressed.

The first issue concerns the assessment of imitation accuracy. This problem has hampered decades of imitation research in autism.⁸ In Park et al.'s study,¹³ trained raters assessed imitation performance with high interrater reliability but such ratings do not yield fine-grained measurements and are not free of subjective biases. Second, matching of another's action to one's own often depends upon the maintenance of mental representation in working memory (WM). Because WM deficits are central to

schizophrenia,¹⁴ its role in imitation needs to be considered. Third, learning by imitation in daily life almost always requires learning not just individual actions but the proper sequence of multiple actions. This distinction is consequential because these 2 dimensions of imitation learning—the individual component actions and information about their sequence—probably depend on somewhat distinct neural mechanisms.^{15,16} The order in which we generate output is of great importance whether it involves movements, language, or thoughts. For example, in the domain of language, compare the sentences, “Sarah beat Joe” with “Joe beat Sarah.” Although both sentences contain the same words, the order in which we utter them makes a dramatic difference to the meaning. Appropriate sequencing of motor output requires the coordination and organization of complex processes, including the linking and ordering of each movement component. Therefore, a complete characterization of imitation ability in schizophrenia should address performance with sequences of actions. It is important to note that sequence learning has also been shown to be impaired in schizophrenia.^{17,18}

In the present study, we aimed to elucidate the cognitive underpinnings and consequences of imitation impairments in schizophrenia. Learning via imitation requires successful integration of multiple perceptual and cognitive processes. The action sequences that are to be imitated must be encoded. Furthermore, in learning skills that require the sequencing of a series of actions, the representation for each action must also be held in WM until the time to respond. Solid evidence points to deficits in guidance of behavior by WM or internal representation in schizophrenia^{14,19,20} and such deficits could undermine imitation, especially in sequencing. Two studies investigated the role of WM load and delay in imitation accuracy^{21,22} using a series of quasi-random dot movements in healthy adults. Increasing WM demand resulted in reduced imitation accuracy, especially in older adults.²¹ Findings from these studies^{21,22} suggest that WM affects imitation performance. Weeks and colleagues²³ investigated imitation of hand shapes both while the stimulus image was displayed online and after a delay. Initially, imitation performance was impaired by the delay between the presentation of the model action and the production of the action but with practice, imitation learning became more robust in the delay condition. Together, these findings suggest that WM interacts significantly with imitation performance in healthy individuals, but the potential effects of WM on imitation in those with reduced WM (eg, schizophrenia) are unknown.

In the present study, we investigated imitation of novel abstract manual gestures. Novel gestures pose a particular imitation challenge and constitute a powerful test of imitation ability.^{24,25} In contrast to the production of meaningful, familiar gestures, imitation of abstract, novel gestures cannot rely on prior knowledge from long-term memory.^{24,26} Instead, with novel gestures, imitation

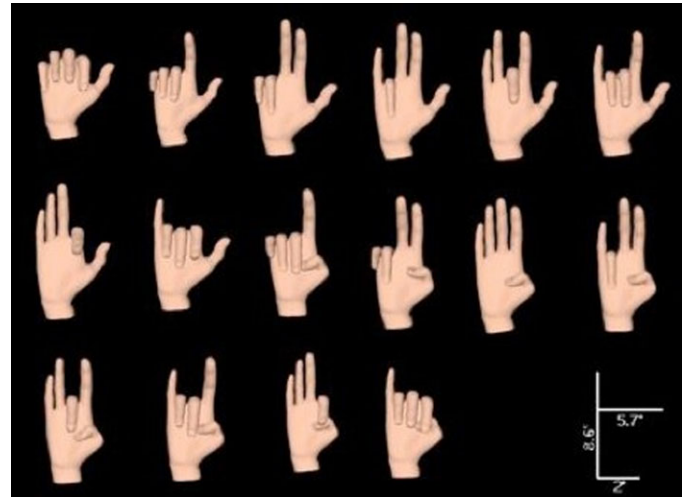


Fig. 1. Hand gestures used to construct gesture sequences in Experiment 1 and Experiment 2.

requires a direct match between the observed gesture and the observer’s motor system, and information for the match must be maintained in WM until the appropriate response is made. Moreover, to imitate a complex action, an observer must capture both the spatial and temporal dynamics. We used a technique sensitive to both spatial and temporal parameters of the model action²⁷ which renders a substantial advantage over the subjective ratings methods to test the hypothesis that schizophrenia patients would be impaired in imitation accuracy and that this deficit would be exacerbated by WM demands.

Methods

Participants

Fifteen outpatients who met the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV²⁸) criteria for schizophrenia (SZ) according to the Structured Clinical Interview for DSM-IV²⁹ were recruited from private psychiatric facilities in Nashville. Fifteen healthy controls (HCs) without any history of DSM-IV Axis I disorder in themselves or their families were recruited through advertisements in Nashville and Waltham. SZ and HC were matched for age, education, and IQ (Wechsler Abbreviated Scale of Intelligence³⁰). All participants had normal or corrected-to-normal vision and were right-handed according to the Edinburgh Handedness Inventory.³¹ Exclusion criteria for both SZ and HC were alcohol or drug dependence, a history of head injury, neurological, or movement disorder. No participant had prior experience with American sign language (ASL), a necessary exclusion criterion because our stimuli were similar to letters in the ASL alphabet (figure 1). All participants provided written informed consent in accordance with the Declaration of Helsinki and were compensated. The protocol was approved by the Vanderbilt University Institutional Review Board

Table 1. Demographic and Clinical Information

	Controls, (n = 14)	Schizophrenia, (n = 14)
Age (years)	40.0 (10.2)	40.2 (8.6)
% Women	50%	50%
Years of education	15.1 (2.9)	14.4 (2.4)
IQ (WASI)	94.7 (10.2)	92.7 (19.2)
SAPS	—	14.2 (13.9)
SANS	—	22.4 (14.75)
CPZ-equivalent dose (mg/kg/day)	—	536.76 (387.04)
Duration of illness	—	19.8 (8.6)
Number of hospitalizations	—	5.4 (6.9)

Note: Values represent the means (SD). SAPS, Scale for the Assessment of Positive Symptoms; SANS, Scale for the Assessment of Negative Symptoms; WASI, Wechsler Abbreviated Scale of Intelligence.

and by the Brandeis University Committee for the Protection of Human Subjects. Results from 2 participants (1 medicated SZ and 1 HC) were eliminated from analysis because hardware failure resulted in a partial loss of their data. Therefore, results from the remaining 14 SZ and 14 HC are reported.

All patients were taking atypical antipsychotic medications (ziprasidone, risperidone, clozapine, or aripiprazole) except for one patient who was unmedicated at the time of testing. No participant was taking anticholinergic drugs. Symptoms ratings for SZ were obtained using the Scale for the Assessment of Positive Symptoms (SAPS³²) and the Scale for the Assessment of Negative Symptoms (SANS³³) (for demographic information, see table 1).

Imitation Tasks

General Method. Model sequences were constructed from individual hand gestures drawn from a pool of 16 different combinations of digit flexions and extensions from Gold et al.²⁷ These hand gestures (see figure 1) could be readily combined to form many different novel sequences. Stimulus sequences were displayed using the Vizard VR Toolkit (WorldViz). For each sequence, a photorealistic rendering of a right hand was displayed. The model hand was oriented so that its palm faced the subject. At its longest, from the wrist to the distal tip of the middle finger, each stimulus hand subtended 8.6° of visual angle. The model hand was sufficiently compact that during observation of a gesture sequence, participants did not have make substantial shifts in gaze.

Participants imitated gestures while wearing a right-handed, one-size 5DT DataGlove 5 Ultra (Fifth Dimension Technologies) along with a hand sensor and a lower arm sensor from the Patriot motion tracking system (Polhemus). Sequences of hand gestures were generated by

a Matlab program whose input were sets of ordered hand gestures and whose output was a seamless sequence in which each individual gesture morphed smoothly into the next.²⁷

Participants viewed the stimuli while seated at a table with their right elbow on a rest and their forearm and digits extended straight up. Their own palm faced toward them, matching the posture with which the stimulus hand was displayed. This match of postures eliminated the need for a visuospatial transformation when imitating the stimulus.

At the start of the testing session, the data glove was calibrated for each participant. During the calibration, participants saw 4 images of hand postures, each with a different combination of digit flexions and extensions. Participants reproduced each gesture, as many times as possible in the 10-s period during which the stimulus hand images were visible.

Following calibration, the actual experiment began. Every gesture stimulus began and ended with the display of an open hand (ie, a hand with all fingers fully extended). These open-hand gestures provided a reference point from which the transition times for the first and the last gestures could be established. This made it possible to compare the timing of a sequence's first and last gestures to the timing of the other gestures. Participants' reproduction of the open-hand gestures was not included in the data analysis.

Experiment 1. Single-Gesture Test

Participants viewed and imitated each of the 16 single hand gestures (figure 1). Each trial consisted of a gesture starting and ending with an open hand, immediately after which the participants imitated. Each gesture was presented for 4 s and imitated 3 times in succession. Participants were given an extra second at the end of the display to complete their imitation. Any imitation that did not start and end with an open hand was excluded from analysis; these instances were extremely rare. There were 48 trials.

Experiment 2. Multiple-Gesture Tests

The task used in Experiment 1 was modified in 2 ways. First, the number of gestures to be imitated was increased to 2 gestures per trial. Having to encode and then imitate a pair of gestures in proper order increases the demand on WM. Second, participants imitated these gestures under 2 conditions, an "online condition" and a "WM condition." In the online condition, participants imitated each sequence of gestures concurrently with the stimulus display. In the WM condition, the trial structure was identical to the online condition except that participants imitated each sequence from memory after a 2-s delay. The order of presentation of the 2 conditions was counterbalanced across participants.

In each trial, a gesture sequence was displayed for 5.5 s, followed by 2-s delay. After the delay, a tone signaled the participants to begin to imitate. This procedure was repeated with the same gesture sequence 8 times in a row. Then a new gesture sequence was given for the next set of 8 repetitions. Gestures sequences were trial unique. In the WM condition, participants had to complete their imitations within 10 s from the end of the displayed sequence. In the online condition, participants were allowed one additional second following the stimulus gesture sequence to complete their imitation. Any imitation that did not start and end with an open hand was excluded from analysis; these instances were extremely rare. There were 128 trials.

Results

We used a multistage algorithm that determines the differences between the imitation model and the participant's reproduction.²⁷ Both the model and the imitation sequence contain a specific number of component gestures. To compare an imitation to a model sequence, the algorithm first segments both the imitation and model into components and then compares the 2 resulting sets of individual components. Once the component static gestures have been identified, the participant's imitation is compared with the original model in terms of both the spatial accuracy of digit flexion/extension and response timing.²⁷ This analysis established 3 main categories of imitation errors. Gesture-level errors were defined by one or more flexion differences between an imitation and the model gesture that it was meant to reproduce. Sequence-level errors were defined by a shift in a gesture's serial position within a sequence, relative to the model sequence. Unmatched errors were scored when the segmentation algorithm was unable to match an imitated gesture to one that had appeared in the model. Unmatched errors occur when participants produced either too many or too few gestures relative to the model sequence. These 3 types of errors were summed to generate a total error score.

We also extracted one important timing measure, the premotor planning time (PMPT),²⁷ which is defined as the interval between the signal to initiate a gesture and the actual start of the imitation. PMPT is different from a simple reaction time that involves a single highly practiced response.

Statistical analyses were performed with SPSS. Where sphericity assumptions were violated, Greenhouse-Geisser corrections were applied. All tests were 2-tailed unless otherwise specified.

Experiment 1

Total error score for single gesture imitation (gesture-level errors in this case) and the PMPT were computed.

Spatial Errors. There was a significant main effect of group ($F_{1,25} = 5.979$, $P = .02$, $\eta_p^2 = 0.19$); SZ made significantly more errors than HC. Figure 2a presents the average number of errors produced as a function of repeated presentations. Errors decreased significantly with repetition ($F_{2,50} = 9.406$, $P < .001$, $\eta_p^2 = 0.23$). The 2 groups did not differ in the change in imitation accuracy across repetitions ($F_{2,50} = 1.1$, $P = .34$).

Premotor Planning Time. Figure 2b presents the mean PMPT as a function of gesture imitation repetition. There was a main effect of repetition ($F_{2,50} = 5.07$, $P = .011$, $\eta_p^2 = 0.17$). There was no significant interaction between PMPT and group ($F_{2,50} = 0.65$, $P = .52$), indicating that the improvement in speed of gesture initiation did not differ between groups.

There was no main effect of group ($F_{1,25} = 0.42$, $P = .51$). Both SZ ($M = 1784.55$, $SE = 81.88$) and HC ($M = 1706.09$, $SE = 84.97$) took approximately 1700 ms to initiate the gesture. Note that the planning time measure is taken from the start of the initial open hand, which is displayed on the screen for 1 s before transitioning to the first gesture.

Relationship between Clinical Symptoms and Imitation Measures. No associations were observed between clinical symptoms or chlorpromazine (CPZ) equivalent dose and imitation errors (SAPS $r = .19$, $P = .95$; SANS $r = -.29$, $P = .30$; CPZ $r = -.62$, $P = .06$) or PMPT (SAPS $r = -.17$, $P = .56$; SANS $r = -.23$, $P = .44$; CPZ $r = -.45$, $P = .20$).

Experiment 2

We examined the number of gestures produced, total errors scores, and PMPT. Unfortunately, because of very low error rates, it was not possible to examine the 3 types of errors separately.

Number of Gestures Produced. Each gesture sequence contained 4 components: an open hand at the outset, a pair of gestures, and an open hand at the end. There were no significant differences between groups ($F_{1,26} = 1.89$, $P = .18$). Both SZ ($M = 3.62$, $SE = 0.094$) and HC ($M = 3.77$, $SE = 0.094$) produced about 4 components per trial. There was no group-by-condition interaction ($F_{1,26} = 0.58$, $P = .45$). This result ruled out the possibility that any potential differences between the groups' imitation accuracy reflects some gross motor impairment that prevented the production of gestures; both groups could imitate the requisite number of gestures.

Spatial Errors. We examined the number of spatial errors as a function of sequence repetition (figure 3a). There was no main effect of groups, but there was a main effect of WM condition ($F_{1,26} = 7.38$, $P = .012$,

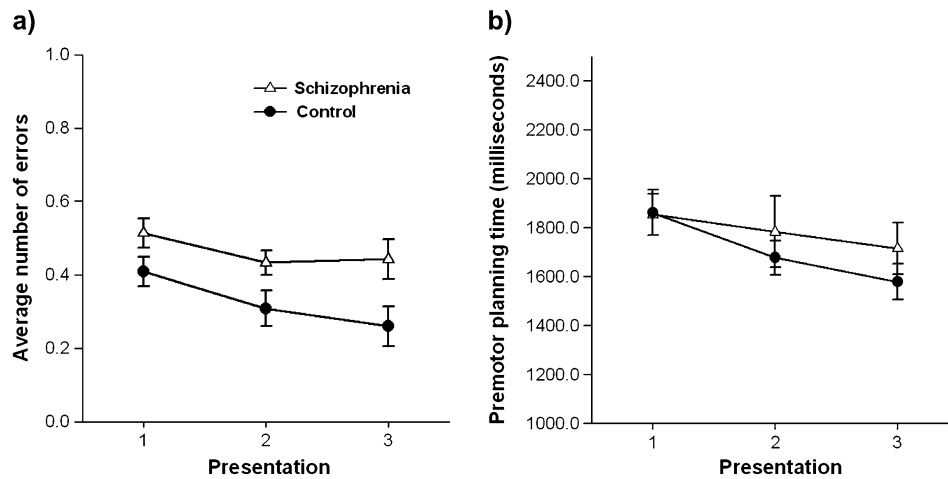


Fig. 2. a) Average number of errors (per gesture) across each presentation of the stimulus model. (b) Average premotor planning time (per gesture) across each presentation of the stimulus model for controls (filled circles) and individuals with schizophrenia (open triangles) for Experiment 1.

$\eta_p^2 = 0.22$); more errors were made during the WM than in the online condition. There was a significant condition-by-group interaction ($F_{1,26} = 6.50, P = .017, \eta_p^2 = 0.20$). Post hoc tests confirmed that the effect of WM was only significant for SZ ($F_{1,13} = 1.66, P = .005$). There was also a significant sequence learning effect, with the number of errors decreasing with repetition ($F_{7,182} = 8.79, P < .001, \eta_p^2 = 0.25$). The effect of presentation repetition also interacted with condition ($F_{1,182} = 5.02, P < .001, \eta_p^2 = 0.16$). Post hoc tests confirmed that this resulted from a significant sequence-learning effect that was seen only in the WM condition ($F_{7,182} = 11.07, P < .001$). There was no group-by-presentation repetition interaction ($F_{7,182} = 1.6, P = .12$).

Premotor Planning Time. Figure 3b shows the mean PMPT as a function of sequence repetition. A significant

sequence learning effect on PMPT was observed ($F_{7,182} = 2.58, P = .014, \eta_p^2 = 0.09$). There was a significant condition-by-group interaction ($F_{1,26} = 10.01, P = .004, \eta_p^2 = 0.28$). Post hoc tests confirmed that the longer PMPT in SZ compared with HC was only observed in the WM condition ($F_{1,26} = 8.741, P = .007$). In the online condition, SZ and HC did not differ in PMPT. This effect may seem odd at first glance but PMPT is not a simple reaction time. It is the interval between the signal to initiate a gesture (tone) and the actual start of the imitation. Therefore, PMPT reflects the time taken to generate the mental representation for imitation. In the WM condition, participants have 2 s (delay period) to prepare for action and HC are presumably ready to move as soon as they hear the tone. In contrast, in the online condition, there is no delay period so there is no preparation time before the participants have to initiate the gesture.

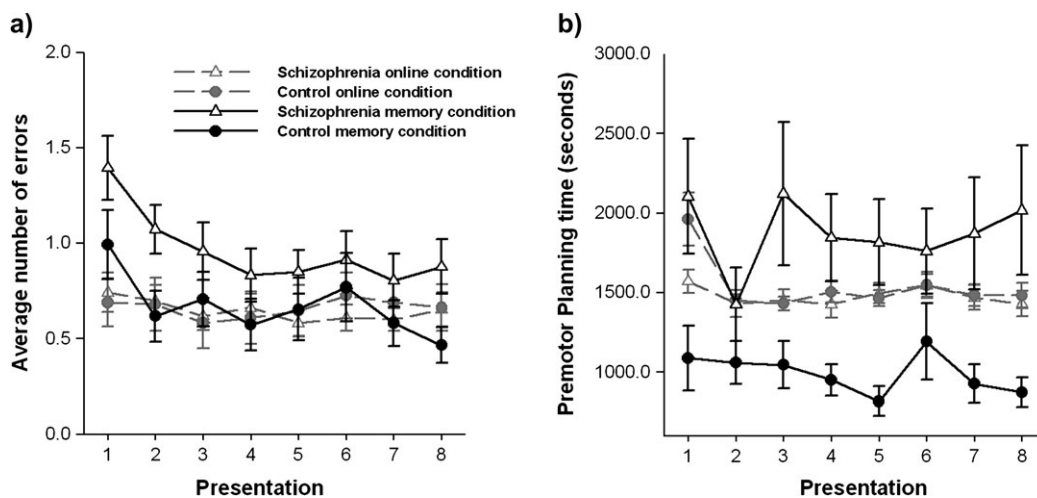


Fig. 3. a) Average number of errors (per gesture) across each presentation of the stimulus model. (b) Average premotor planning time (per gesture) across each presentation of the stimulus model for controls (filled circles) and individuals with schizophrenia (open triangles) for Experiment 2.

Table 2. Spearman's Nonparametric Correlations (r) between Mean Imitation Errors, PMPT, and Clinical Symptoms Scores for Individuals with Schizophrenia from Experiment 2

	Mean Imitation Errors	Mean PMPT
WM condition		
SAPS	0.24	0.32
SANS	0.51*	0.61*
CPZ-equivalent dose	-0.09	0.10
Online condition		
SAPS	0.20	-0.18
SANS	0.48	0.19
CPZ-equivalent dose	-0.14	0.17

Note: Abbreviations are explained in the first footnote to table 1. $P < .05$.

WM deficits in SZ may prevent them from being able to use the 2-s delay in the WM condition to prepare their imitation.

Relationship between Clinical Symptoms and Imitation Ability

We examined correlations between imitation performance and the severity of clinical symptoms. There was a significant correlation between SANS total score and increased spatial errors in the WM condition ($r = .51, P = .03$) and with longer PMPT in the WM condition ($r = .61, P = .02$). These results indicate that the severity of negative symptoms is associated with both temporal and spatial parameters of imitation in the WM condition. In contrast, we saw no significant correlations between SAPS and any measures of imitation. There were no significant correlations for the online condition and no significant correlations between the CPZ-equivalent dose and any of the imitation measures. See table 2.

Discussion

We investigated gesture imitation in schizophrenia using a novel technology that allowed us to objectively and quantitatively assess imitation ability. We also sought to examine the cognitive underpinnings of imitation ability and to relate it to clinical symptoms of schizophrenia.

A number of key findings emerge from the present study. First, our results show that SZ are impaired in gesture imitation as assessed by an objective quantitative method.²⁷ SZ were less accurate when imitating novel manual gestures than HC. This result supports the previous findings¹³ and confirms that imitation deficits in schizophrenia are not limited to facial expressions of emotion but represent a more pervasive problem.

In Experiment 2, we investigated the role of WM in imitation by introducing a delay between the presentation of a gesture sequence and the participant's reproduction. Learning a new skill, whether social or

nonsocial, often requires the novice learner to observe an action and then internally model and maintain a mental representation of the action in WM until the appropriate moment to reproduce the action. Given the profound WM deficits in schizophrenia,¹⁴ we hypothesized that requiring imitations to be based on WM would be particularly challenging for SZ. Indeed, imitation errors increased in SZ when they had to draw upon WM rather than being able to imitate concurrently with the target gestures. This pattern was not observed in HC. Interestingly, SZ, but not HC, showed increased PMPT when WM demand was introduced. Similar increases in latency have been observed in SZ in other paradigms. For example, the latency of visually guided saccades to a target is normal in SZ.^{20,34} However, their saccadic latencies are significantly increased when a saccade must be guided by an internal representation of the target, eg, in memory-guided saccade paradigms^{20,34} or in tasks that require response inhibition.³⁵ We hypothesize that the increase in PMPT in SZ under WM demands reflects increased time taken to prepare the intended imitation by reactivating the mental representations of the gesture sequence. In contrast, HC may even benefit from the delay because they are able to use this period to prepare for action. Taken together, these results suggest that WM deficits influence both the accuracy and the planning time of imitated gestures in schizophrenia.

The gesture sequences in Experiment 2 and the single gestures in Experiment 1 were each presented multiple times. This allowed us to examine learning with repetitions and to estimate the improvement in imitation performance with repeated presentation of gestures. In both experiments, both groups showed a decrease in errors and in PMPT with the repetition of gesture imitation. This suggests that both groups benefited from repeated presentations. In Experiment 2, both imitation accuracy and PMPT were worse under WM condition in SZ, but there was no interaction between gesture repetition and WM condition. An inspection of imitation performance as a function of repeated stimulus presentations and reproductions suggests that the majority of imitation errors, for both groups, occurred in the first presentation of a gesture sequence (see figure 3a); a similar pattern is also seen in the single-gesture imitations in Experiment 1. Imitation of novel, unfamiliar actions demands the generation of a new internal representation of the stimulus and a new mapping of that representation onto a motor plan. SZ did improve their imitation performance with repeated presentations, which suggests that after their initial difficulties in generating an internal representation of the target gesture, the representation remains stable enough to guide action. Alternatively, it is possible that with repeated practice, internal representation is generated with greater accuracy and less noise.

In Experiment 2, we found that negative but not positive symptoms were associated with imitation impairments under WM load. This finding replicates the association between negative symptoms and imitation accuracy reported in a previous study.¹³ Negative symptoms are characterized by difficulties in initiating and sustaining many forms of behavior including actions, speech, and thoughts. Difficulties in generating and sustaining internal representation of the external world might contribute to both imitation difficulties and negative symptoms. In this regard, it is noteworthy that the neural circuitry that supports imitation, particularly the prefrontal and premotor cortex^{4,36} overlaps with the regions that are implicated in the negative symptoms of schizophrenia.^{37,38} Moreover, the role of the prefrontal cortex in WM is well established.¹⁹ Taken together, the relationship between negative symptoms and imitation ability in schizophrenia appears reliable. It should be noted, however, that we did not find a significant correlation between symptom ratings and single-gesture imitation ability in Experiment 1. As the imitation task in Experiment 1 was very easy, this lack of association may be related to the restricted range of imitation errors.

Our finding of a significant correlation between negative symptoms and WM-dependent imitation also suggests a possible route by which negative symptoms are linked to social functioning in schizophrenia. Imitation provides one means to connect our internal states to the external world in which we are anchored. Imitation allows us to model and simulate the consequences of our actions. The co-occurrence of imitation deficit, negative symptoms, and social deficits suggest that the connection between internal states and the external world may be severed or tenuous.

Imitation deficit may be most problematic when rapid learning of new skills is required because the greatest difficulty was found on the first presentation of the target gesture. However, improved imitation with repeated presentation suggests a potential strategy for remediation. Specifically, individuals could be familiarized with the action that is to be learned, which would reduce the burden that seems to be associated with generating and maintaining a novel internal representation.

One significant innovation in the present study is that our measurement method was objective and quantitative, which opens up possibilities of rigorous follow-up studies. However, there are some limitations to this study. First, the potential effects of antipsychotic drugs on imitation are not clearly understood. Although antipsychotic medication does not seem to cause deficits in imitation,¹³ sequence learning,³⁹ or WM,⁴⁰ all antipsychotic drugs affect the dopamine system and the frontostriatal circuitry. In our sample, there was no significant correlation between imitation ability and CPZ-equivalent dose. Furthermore, we are reasonably confident that the motor imitation deficit reported in the present study was

not caused by antipsychotic medication because we observed normal PMPT in SZ when there was no WM demand. However, potential drug effects must be directly addressed in the future. A second limitation is the modest sample size. However, the 2 groups were carefully matched, and our measures were objective and quantitative. Furthermore, the group difference in imitation ability appears to be robust, as indicated by large effect sizes. A third limitation is that the tasks in the present study proved to be easy, resulting in low overall error rates. Thus, we were unable to separately analyze the 3 types of spatial errors, which could have further clarified different sources of imitation errors in schizophrenia. Future studies should examine these different types of errors by increasing the sequence length and the WM demand to increase the overall error rates. Because our tasks were very easy, it is possible that the imitation deficit in schizophrenia may actually be more profound than reported here. Indeed, a previous study¹³ reported a larger imitation deficit even with less precise measurements.

In sum, we found that the imitation of novel gestures was impaired in schizophrenia, and a WM demand exacerbated this deficit. In addition, we observed an association of negative symptoms and imitation ability. Because imitation is an important means by which we learn new social and nonsocial skills, impaired imitation ability may play a significant role in functional outcome of schizophrenia. However, we also found that with repetition, imitation improved even in the patients. This finding underscores the importance of practice and repetition in the learning environment.

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