Prism Adaptation Deficits in Schizophrenia

Lisa A. Bartolomeo¹, Yong-Wook Shin^{*,2}, Hannah J. Block^{3,6}, Amanda R. Bolbecker¹, Alan F. Breier⁴, Brian O'Donnell¹, and William P. Hetrick¹

¹Department of Psychological and Brain Sciences and Program in Neuroscience, Indiana University, Bloomington, IN; ²Department of Psychiatry, Asan Medical Center, University of Ulsan College of Medicine, Seoul, South Korea; ³Department of Kinesiology and Program in Neuroscience, Indiana University, Bloomington, IN; ⁴Department of Psychiatry and Prevention and the Recovery Center for Early Psychosis, Indiana University School of Medicine, Indianapolis, IN

*To whom correspondence should be addressed; tel: +821037275375, fax: 02-485-8381, e-mail: shaman_korea@mac.com

Recent clinical and neurobehavioral evidence suggests cerebellar dysfunction in schizophrenia (SZ). We used the prism adaptation motor task (PAT) to probe specific cerebellar circuits in the disorder. PAT requires cerebellum-dependent motor adaptation, perceptual remapping, and strategic control. A failure to engage in early corrective processes may indicate impairment within either the cerebellum or regions contributing to strategic components, such as the parietal lobe, while an inability to develop and retain a visuomotor shift with time strongly suggests cerebellar impairment. Thirty-one individuals with SZ and 31 individuals without a history of psychological disorders completed PAT. Subjects reached to a target before, during, and following prism exposure, while their movements were recorded using motion-sensing technology. The SZ group performed worse on conditions consisting of adaptation, post-adaptation, aftereffects, and reorientation, thereby demonstrating a failure to adapt to the same degree as healthy controls. SZ performance remained impaired even with visual feedback and did not differ from controls at baseline, suggesting the observed deficit is specific to adaptation. Results indicate that sensorimotor adaptation is impaired in SZ and implicate disturbances in cerebellar circuits.

Key words: schizophrenia/prism adaptation/motor adaptation/cerebellum

Introduction

Psychomotor disturbances are core features of schizophrenia (SZ) that can occur independently of medication and illness-related effects. Cerebellar dysfunction in SZ is corroborated by behavioral, anatomical, and neurophysiological approaches.^{1–3} A recent meta-analysis of neuroimaging results in first-episode, drug naïve individuals with SZ found consistent and widespread cerebellar

structural and functional abnormalities in regions associated with motor, affective, and cognitive processes.⁴ The cerebellum is therefore a region of particular interest when studying the pathophysiology of SZ.

Motor adaptation, a process of adjusting motor commands to reduce systematic movement error, depends on the cerebellum.^{5,6} The prism adaptation motor task (PAT) requires motor adaptation and may therefore be well-suited for testing hypotheses about cerebellar dysfunction in SZ. PAT is a visuomotor task that elicits several adaptive processes, including (1) perceptual remapping, a change in the spatial relationship between vision and proprioception⁷; (2) strategic control, involving changes in feedforward planning or cognitive components⁸; and (3) motor adaptation, a trial-and-error feedback-based modification of internal models relating motor commands to movement outcomes.^{8,9} PAT requires participants to point to a target while wearing prismatic goggles that alter the angle at which light enters the eye, thereby displacing the visual field. This visual perturbation subsequently displaces pointing behavior to a degree and direction, that is proportional to the visual displacement.⁸ Participants adapt to the displacement in order to regain pointing accuracy when the pointing limb is exposed to their view. With concurrent exposure, participants are provided a distal view of their endpoint location, which permits the integration of proprioceptive and visual information.⁸ After multiple trials with concurrent exposure, participants rapidly reduce pointing errors and regain target-pointing accuracy. If concurrent exposure is discontinued while the prism is present, adaptation continues, albeit at a slower rate. Removal of the prism leads to errors in the opposite direction, a phenomenon known as negative aftereffect.⁸ Negative aftereffect is a sign of motor adaptation, signifying that the brain has successfully updated and retained internal models relating motor commands

to movement outcomes. With time, participants adapt again to regain pointing accuracy.

The adaptive processes elicited by PAT are therefore thought to comprise 2 distinct stages: an early stage characterized by rapid error reduction, but also rapid forgetting when the perturbation is removed, and a later stage, involving more gradual error reduction coupled with gradual forgetting of the adaptation when the perturbation is removed.^{8–11} The later stage is thought to be more cerebellum-dependent than the early stage, indicated by fMRI findings of increasing cerebellar activation during prism exposure^{12,13}; however, the cerebellar BOLD-signal measured in these studies might largely reflect neural changes outside of the cerebellum.¹⁴ Notably, prism adaptation is impaired in nonhuman primates¹⁵ and human patients with cerebellar diseases or lesions,^{16–21} but a lack of imaging studies in these populations poses challenges for clarifying the cerebellum's role in early and late adaptive processes.

The few studies of prism adaptation in SZ have demonstrated performance deficits relative to healthy controls. Ebner et al²² found that individuals with SZ had significantly less “adaptive shift” after prism exposure, which was replicated in a later study.²³

More recently, Bigelow et al²⁴ found that individuals with SZ had reduced target-pointing accuracy relative to healthy controls during early adaptation, and demonstrated greater persistence of aftereffects during reorientation, indicating difficulty adjusting after the prism was removed. These findings were interpreted as a procedural learning deficit involving abnormalities within brain regions responsible for visuomotor processing, including the frontal cortex, basal ganglia, and cerebellum. Taken together, there is evidence for the potential utility of PAT in studying the cerebellum in SZ, but details of which adaptive processes are affected, and on what timescale, are unclear.

Here, we extend previous findings of abnormal PAT performance in SZ to determine whether SZ-related deficits are associated with the later adaptive processes that are thought to be more cerebellum-dependent. We hypothesized that individuals with SZ would adapt to a lesser extent than healthy controls during prism exposure, and that this difference would also be present during late adaptation. Associations with measures of involuntary movement and ataxia were also examined, with the expectation that more severe adaptation deficits would be correlated with more severe motor abnormalities in individuals with SZ.

Method

Participants

Thirty-one individuals between the ages 18 and 55 meeting DSM-IV criteria for SZ ($n = 20$) or schizoaffective disorder ($n = 11$; SZ group) and 31 individuals without a

history of psychopathology (healthy controls; HC) were included in the study. Three participants in the SZ group were first-episode patients. Participants were recruited through word of mouth, flyers posted in the community and local mental health clinics, newspaper advertisements, and the research network of Indiana University's Clinical and Translational Sciences Institute. Exclusionary criteria included a history of head injury with loss of consciousness exceeding 5 min, alcohol or drug dependence within the past 6 months, cardiovascular problems, neurological problems, IQ lower than 70, and lack of grade school education. The study procedures were approved by the Indiana University Institutional Review Board and the study was conducted in accordance with the Declaration of Helsinki (Edinburgh amendments). Written informed consent was obtained from all participants.

During the initial screening visit, trained research staff administered either the patient or nonpatient edition of the Structured Clinical Interview for the DSM-IV Axis I Disorders (SCID^{25,26}) and the anxiety portion of the Mini-International Neuropsychiatric Interview (MINI²⁷). The SCID-I psychotic and mood modules were used as diagnostic tools to determine whether participants had SZ or schizoaffective disorder (SZ group) or were free from psychotic and mood disorders (HC group). The MINI was used to screen for the presence of anxiety disorders, an exclusionary criterion for HC. HC were also interviewed using the SCID-II to assess for the presence of DSM-IV Axis II personality disorders. IQ was assessed using the Wechsler Abbreviated Scale of Intelligence Version II (WASI-II²⁸). All participants completed a urinary drug screen as part of the study, except for 2 SZ and 3 HC. Nine SZ (marijuana $n = 8$; cocaine $n = 1$) and 3 HC tested positive (marijuana $n = 2$; amphetamine $n = 1$).

Assessments

Gross motor function was measured using the Abnormal Involuntary Movement Scale (AIMS²⁹) to assess involuntary movements resulting from neuroleptic medications, and the International Cooperative Ataxia Rating Scale (ICARS) to assess ataxias associated with neurological conditions and medication side effects.³⁰ The Positive and Negative Syndrome Scale (PANSS³¹) was used to assess symptom severity in the SZ group.

Materials

The PAT apparatus, modeled after Bigelow et al,²⁴ was modified to include motionsensing devices to continuously track participants' movements and measure end-point accuracy.

As depicted in [figure 1](#), the plywood structure was placed upon a desk so that participants could comfortably reach from the origin to the target within the enclosure. The origin was located on the base of the apparatus,

Fig. 1. Prism adaptation apparatus and pointing behavior during prismatic exposure. *Note:* (A) Top-down view of a participant who has pointed to the right (positive axis) of the target (black circle), due to a rightward shift in visual field caused by the prism goggles (colored wedge). Gray shaded area was not visible to the subject. (B) A participant is shown where he pointed in relation to the target and is surprised by the displacement (as denoted by !). This exposure leads to rapid error detection and correction. (C) Photo of the PAT apparatus, similar to A, in which the participant's terminal endpoint location is to the right of the target.

36 cm behind and 16.5 cm to the right of the target. To ensure a uniform start point, a round (1.5 cm diameter) touchpad sensor was affixed to the origin that illuminated a small light bulb atop the target when touched with the right index finger. The target was a rod centered behind a piece of plexiglass spanning the width of the apparatus. An occlusion board blocked the participant's view of his/her reach and location relative to the target, but could be removed to provide terminal visual feedback on appropriate trials. A chin rest ensured a uniform stature. A motion sensor was attached to the dorsal surface of the participant's right index finger. Ascension Motion Sensing Technology 3DGuidance trakSTAR and *Cubes* Software measured participants' position in the front hemisphere of the transmitter, or along the x -axis (ie, as participants reached to left or right of the target in front of the transmitter), at a rate of 80 measurements per second.³² The target plane ran parallel to the y -axis within the transmitter's performance window (ie, the area in which the transmitter can detect movement); therefore, the y -coordinate (in cm) of the participant's terminal reach location was obtained. The apparatus was placed at least 5 ft away from any metal in the testing room to avoid distorting the magnetic field.³² Twenty diopter prismatic lenses inserted into Fisher scientific goggles were worn on appropriate trials to induce the visual displacement. The experimenter inspected the placement of goggles on each participant to ensure the prism covered the entire visual field. Participants wearing corrective lenses were able to keep them on under the prismatic

goggles. The prism induced a displacement of approximately 12 degrees.

Procedure

Procedures were also modeled after Bigelow et al.²⁴ Participants sat in a chair at the open end of the apparatus, positioned on the chin rest and with their finger on the origin touchpad (figure 1). At the beginning of each condition, participants were instructed to complete a set number of trials, where one trial consisted of reaching from the origin to the target and back again. Consistent with past studies, the number of trials varied across conditions and ranged from 6 to 20, which is sufficient for eliciting the adaptive processes of interest^{12,33,34} (table 3). Participants were told to reach back and forth at their own pace using their right index finger, waiting for the light atop the target to illuminate before removing their finger from the origin. The experimenter counted the number of trials aloud, while also monitoring the participants' performance to ensure they remained engaged and attentive throughout the task. On conditions when concurrent visual feedback was provided, the experimenter moved the occlusion board when the participant arrived at the target to briefly display his/her finger. Before returning to the origin, participants were instructed to hold their finger at the target until the occlusion board was completely back in place. The task lasted approximately 20 min, and there were no gaps between trials.

Table 1. Demographic, Clinical, and Medication Information

	SZ (<i>n</i> = 31)	HC (<i>n</i> = 31)	Two-Sample <i>t</i> -Test Test Statistic (<i>P</i>)
Age (years)	38.23 ± 11.24	36.45 ± 11.54	−0.79 (.43)
Years of education	13.60	16.58	6.61 (<.0001)*
Handedness (R:L)	27:4	27:4	
Sex (F:M)	16:15	14:17	
Psychotropic medication			
No medication	2	31	
Atypical antipsychotic	27	0	
Typical antipsychotic	5	0	
Antipsychotic/mood stabilizer	7	0	
Antidepressant	10	0	
Anxiolytic	5	0	
Anticholinergic	3	0	
Duration of illness	18.68 ± 12.58	—	
CPZ dose equivalent (mg)	525.51 ± 363.64	—	
AIMS total	0.23 ± 0.57	0.03 ± 0.18	−1.84 (.07)
ICARS total	6.55 ± 3.97	3.03 ± 2.27	−4.23 (<.001)*
FSIQ	92.55 ± 13.53	111 ± 11.5	5.78 (<.001)*
PANSS			
Positive symptoms	15.57 ± 5.04	—	
Negative symptoms	14.79 ± 4.79	—	
Disorganized symptoms	2.36 ± 1.25	—	
General symptoms	29.36 ± 7.28	—	
Total score	59.71 ± 12.67	—	

Note: Means ± SD unless otherwise indicated. Asterisk denotes significant group difference where $P < .05$. One participant in the SZ group did not disclose educational information. One participant in the HC group did not complete the cerebellar assessments. Two participants in the SZ group did not complete the PANSS. SZ, schizophrenia; HC, healthy control; CPZ, chlorpromazine; AIMS, Abnormal Involuntary Movement Scale; ICARS, International Cooperative Ataxia Rating Scale; FSIQ, WASI Fullscale IQ; PANSS, Positive and Negative Syndrome Scale.

Table 2. Prism Adaptation Trial Conditions

Condition	Number of Trials	Prism	Visual Feedback
Practice	2	Absent	Present
Baseline	6	Absent	Absent
Preadaptation	10	Present	Absent
Adaptation	20	Present	Present
Post-adaptation	20	Present	Absent
Aftereffect	10	Absent	Absent
Reorientation	6	Absent	Present

Note: Trial procedures for the prism adaptation paradigm.

To orient participants to the task, 2 practice trials were completed with visual feedback. In order to assess baseline behavior before prism exposure, participants then completed 6 reaches without visual feedback. The prismatic goggles were then introduced for the preadaptation condition, during which participants completed 10 reaches without visual feedback in order to assess the initial perturbation induced by the prism. Next, during the adaptation condition, participants completed 20 reaches with visual feedback, which allowed them to adapt to the displacement. This condition captures the early stage of adaptation characterized by rapid learning. During post-adaptation, participants completed 20 reaches without visual feedback in order to assess the later, slower stage

of adaptation. Next, participants were instructed to remove the prismatic goggles for the aftereffect condition, where they completed 10 reaches without visual feedback to measure negative aftereffect. Finally, during reorientation, participants completed 6 reaches with visual feedback, which measured their ability to adjust to displacement incurred by removing the prism.

Statistical Analyses

All analyses were conducted using SPSS Version 24.0.³⁵ Consistent with Bigelow et al.,²⁴ one-way ANOVAs were used to examine group differences in PAT performance within baseline, preadaptation, aftereffect, and reorientation conditions. The adaptation and post-adaptation conditions were grouped into blocks of 5 trials and repeated measures ANOVAs were used to examine group differences. Although there were a significant group difference and significant correlations between ICARS total and kinetic scores and aftereffect magnitude, these variables were not entered as covariates because they were found to be nonsignificant in the models for ANOVAs assessing PAT performance. Greenhouse–Geisser corrections were used for repeated measures analyses with more than one degree of freedom. In these cases, the uncorrected degrees of freedom and the corrected P values are reported. ANOVA tests were considered statistically significant at the $P < 0.05$ level (2-tailed).

Table 3. Prism adaptation test performance

Conditions	SZ (<i>n</i> = 31)		HC (<i>n</i> = 31)	
	Mean	SD	Mean	SD
Baseline	-20.64	20.21	-13.77	18.62
Pre-adaptation	-96.60	26.91	-89.95	25.29
Adaptation* Trials 1–5*	-68.06	33.54	-52.92	23.13
Trials 6–10*	-43.00	29.02	-26.71	22.87
Trials 11–15	-37.35	32.90	-25.13	26.05
Trials 16–20*	-37.61	28.25	-24.05	23.12
Post-adaptation* Trials 1–5*	-48.50	34.33	-24.05	22.97
Trials 6–10*	-45.60	37.08	-20.20	24.42
Trials 11–15*	-47.15	36.72	-18.87	26.20
Trials 16–20*	-47.41	35.41	-16.45	27.62
Aftereffect*	7.93	24.79	25.00	21.43
Reorientation*	-14.00	22.67	0.67	18.06

Note: Asterisk denotes significance where $P < .05$. Means and SD for reach endpoint location across trial conditions. SZ, schizophrenia; HC, healthy control.

Two separate difference scores were calculated to index adaptation magnitude (average of the last 5 post-adaptation trials – average of the last 5 pre-adaptation trials) and aftereffect magnitude (average of the first 5 aftereffect trials – average of the last 5 baseline trials), which are standard measures used to gauge performance on visuomotor adaptation tasks.²⁰ In SZ, Pearson correlations were conducted to assess the relationship between adaptation and aftereffect magnitude with AIMS and ICARS total scores, as well as with chlorpromazine (CPZ) equivalents to assess medication effects. See [supplementary material](#) for methods and results regarding correlations with cognitive, clinical, and additional motor variables. No correction for multiple comparisons was performed.

Results

Participants

The HC group completed significantly more education and had significantly higher IQ than the SZ group ($P < .001$). The SZ group scored significantly higher on the ICARS ($P < 0.05$), suggesting a higher prevalence of cerebellar ataxias; however, there was no difference in AIMS scores between groups. Demographic information is summarized in [table 1](#), and clinical results are displayed in [table 2](#).

Prism Adaptation Performance Across Trials

Group differences in PAT performance are displayed in [table 3](#) and depicted in [figure 2](#).

Baseline. Groups did not differ significantly in endpoint location during baseline trials [$F(1, 61) = 1.92, P = .17$], suggesting that SZ and HC performed comparably before the visual displacement induced by prism exposure.

Preadaptation. Both groups demonstrated rightward errors when the prismatic goggles were introduced in

the absence of visual feedback and did not significantly differ in endpoint location during preadaptation [$F(1, 61) = 1.01, P = .32$].

Adaptation. When provided terminal visual feedback while still wearing the prismatic goggles, the HC group corrected rightward errors more effectively, as indicated by a significant main effect of group [$F(1, 60) = 4.62, P = .04$]. There was also a significant effect of trial [$F(19, 1140) = 48.69, P < .01$], indicating a shift toward the target with successive trials and nonsignificant group-by-trial interaction [$F(19, 1140) = 0.98, P = .45$]. Repeated measures ANOVA revealed a significant main effect of group for the first, second, and fourth trial blocks: block 1 [$F(1, 60) = 4.28, P = .04$], block 2 [$F(1, 60) = 5.75, P = .02$], block 3 [$F(1, 60) = 2.63, P = .11$], block 4 [$F(1, 60) = 4.30, P = .04$].

Post-adaptation. There was a significant main effect of group on endpoint location during post-adaptation following removal of visual feedback [$F(1, 60) = 12.99, P < .001$], indicating that correction of rightward errors was impaired in SZ. There was also a significant effect of trial [$F(19, 1140) = 2.78, P < .01$] and a nonsignificant group-by-trial interaction [$F(19, 1140) = 1.09, P = .37$]. Repeated measures ANOVA revealed a significant main effect of group for all trial blocks: block 1 [$F(1, 60) = 10.87, P < .01$], block 2 [$F(1, 60) = 10.15, P < .01$], block 3 [$F(1, 60) = 12.18, P < .01$], block 4 [$F(1, 60) = 14.74, P < .001$].

Aftereffect. Upon removal of the prismatic goggles, both groups demonstrated leftward errors relative to the target. Aftereffect was significantly reduced in SZ relative to HC [$F(1, 61) = 8.42, P = .01$], indicating deficient motor adaptation.

Reorientation. Reorientation was significantly reduced in the SZ group relative to HC [$F(1, 61) = 7.94, P = .01$]. This suggests that, even with visual cues and normative viewing conditions restored, individuals with SZ remained significantly further from the target compared with HC.

Fig. 2. Individual and mean terminal endpoint location across trial conditions. *Note:* Distance from the target in millimeters is on the y -axis, and trial number is on the x -axis. The positive y -axis represents rightward endpoint errors and the negative y -axis represents leftward endpoint errors. The value for the mean finger position was shifted to have zero at the mean y -axis coordinates at the baseline for both groups. Both groups rapidly reduced their rightward errors during adaptation, but main effects of group ($P < .05$) were present in adaptation and each subsequent block of trials, indicating that SZ adapted less than HC.

Correlations

In SZ, there was a significant correlation between aftereffect magnitude and ICARS total score ($n = 31$, $r = .38$, $P = .04$), such that higher ICARS scores were associated with greater aftereffect magnitude. A significant correlation between ICARS kinetic domain scores and aftereffect magnitude ($n = 31$, $r = .39$, $P = .03$) suggests this association is driven by limb ataxia. There was a nonsignificant correlation between ICARS total score and adaptation magnitude ($n = 31$, $r = .32$, $P = .08$). Neither adaptation nor aftereffect magnitude were associated with total AIMS scores ($n = 30$; for adaptation $r = .13$, $P = .50$; for aftereffect $r = .23$, $P = .23$) nor CPZ equivalent dose ($n = 26$; for adaptation $r = -.30$, $P = .13$; for aftereffect $r = -.12$, $P = .55$), suggesting that prism adaptation performance was not associated with extrapyramidal and medication effects.

Discussion

In the present study, we examined cerebellar-dependent adaptive processes by comparing prism adaptation in individuals with SZ and HC. During prism exposure, SZ adapted less than HC both in the presence and absence of visual feedback (ie, during the adaptation and post-adaptation conditions, respectively). When the prism was removed, both groups demonstrated retention of motor learning and achieved significant negative aftereffect, which was again reduced in SZ. Consistent with past studies, these findings point to deficits during early adaptation in SZ. Importantly, we extended past findings by also showing novel evidence for impairment during late adaptation. These findings suggest that individuals with

SZ rely more on visual feedback and cannot integrate proprioceptive information as well as controls. Greater aftereffect magnitude was associated with more severe ataxia in SZ, particularly limb ataxia, but was not determined to be significant as a covariate; thus, the predominant deficits related to prism appear to involve perceptual remapping and proprioception rather than motor difficulties like ataxia and involuntary movements.

The current findings add to abundant behavioral evidence for deficits in early adaptation in SZ.^{22–24} In contrast to past studies, our methodological approach may have permitted us to also detect impairment during late adaptation. Specifically, we measured location using motion sensing technology, whereas previous research relied on visual observation by the experimenters.

Bigelow et al²⁴ reported measurement precision within 0.5 cm, experimenters measured endpoint location using a flashlight in the dark in the study by Pitblado et al.,²³ and Ebner et al.²² did not elaborate on measurement procedures. The methodology in the present study likely enhanced measurement precision, leading to more sensitive detection of group differences. The current study also included a patient sample with more diverse psychosis pathology than past studies. Bigelow et al²⁴ included participants with SZ or schizophreniform disorder, whereas our sample included individuals with SZ or schizoaffective disorder. It is possible that affective dysregulation in schizoaffective disorder in the present sample could affect performance in ways that are not yet determined; likewise, it could be that some of the participants in the schizophreniform group in the study by Bigelow et al²⁴ did not transition to a SZ diagnosis.

Neuroimaging studies in healthy controls have examined the neural circuits subserving early and late adaptive processes operating during prism adaptation, which may shed light on neuropathological abnormalities in SZ. Early stages of adaptation likely involve basal ganglia connections to frontal and parietal cortices^{36,37}; moreover, cerebellar interactions with parietal cortices appear to be critical in early adaptation, with cerebellar activation occurring in advance of and likely modulating subsequent parietal cortex activity as evidenced in patients with spatial neglect following frontal lesions.³⁸ This cerebellar influence on parietal activity is likely effected via cerebellar deep nuclei, namely the ventro-caudal dentate projection to parietal (and prefrontal) cortex,⁶ an inference supported by an fMRI study of prism adaptation by Küper et al.³³ that found that posterior cerebellum (lobules VIII and IX) and ventro-caudal dentate were activated in the early stage of prism adaptation, where fast strategic motor control processes involved in explicit learning are required and cognitive processes are more prominent. Posterior parietal cortex has been shown to be critically involved in early-stage prism adaptation.³⁶ During late-stage PAT, when spatial realignment processes which are implicitly learned occur, it is generally agreed that lobules IV–VI are involved.^{33,39} Importantly, Luarte et al.¹² reported increasing activation in lobules VI–V as spatial realignment progressed. These findings of cerebellar involvement in early and late stages of prism adaptation are consistent with reports from other forms of procedural learning, including Penhune and Steele,⁴⁰ who reported lateral cerebellum fMRI activation presumably providing input to the frontal–parietal–striatal circuit involved in accuracy, whereas late stage adaptation recruited midline cerebellum which is involved in motor control. Thus, our findings of SZ impaired early and late adaptation during prism adaptation may point to fundamental deficits within the cortico-cerebellar-thalamiccortical circuit, which has been suggested to underlie diverse symptoms of the disorder including motor dysfunction.

To our knowledge, similar neuroimaging studies have not yet been conducted in individuals with SZ, which are necessary for determining the neural mechanisms underlying impaired prism adaptation. Such work has the potential to improve understanding of pathophysiological processes implicated in the etiology of SZ and inform novel targets for intervention. As just one example, stimulating the cerebellum with transcranial direct current stimulation (tDCS) has been shown to alter prism adaptation in healthy participants. Anodal stimulation appears to enhance adaptation speed,⁴¹ while cathodal stimulation results in larger and more persistent displacement of reach endpoint in healthy individuals.⁴² tDCS may be useful for modulating cerebellar abnormalities in SZ, which could be assessed using a behavioral paradigm like PAT.

Analyzing endpoint accuracy, as we did here, is useful for making inferences about prism adaptation; however,

other behavioral parameters may provide further insight into observed differences between individuals with SZ and controls. In particular, movement speed,⁴³ starting posture, and reach angle⁴⁴ may also account for variance in performance. We were able to rule out influences from speed by measuring the speed of target-directed finger movement, which showed no group differences in all 6 PAT conditions (all $P > .05$); however, we were unable to assess posture and reach angle, which are important considerations for future studies. Another limitation of the study was the inclusion of participants with positive drug screens, which is another consideration for future research.

Conclusion

The results of the present study provide evidence for impaired prism adaptation in SZ. Findings converged across PAT trial conditions to reflect deficits in the fast and slowly developing adaptive processes that underlie motor adaptation. Considering results from lesion, neuroimaging, and tDCS studies implicating the cerebellum in motor adaptation, the current findings add to accumulating evidence for cerebellar dysfunction in SZ and suggest that PAT has utility for further probing the nature of this dysfunction.

Supplementary Material

Supplementary data are available at *Schizophrenia Bulletin* online.

Funding

This work was supported in part by the National Institute of Mental Health (R01 MH074983 to W.P.H.).

Acknowledgments

The authors wish to thank the patients and their families for participation as well as support from the staff and administration of Larue D. Carter Memorial Hospital. All authors declare that they have no conflicts of interest.

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