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RESEARCH ARTICLE

Control of Movement

Disruption of somatosensory cortex impairs motor learning and retention

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Abstract

This study tests for a function of the somatosensory cortex, that, in addition to its role in processing somatic afferent information, somatosensory cortex contributes both to motor learning and the stabilization of motor memory. Continuous theta-burst magnetic stimulation (cTBS) was applied, before force-field training to disrupt activity in either the primary somatosensory cortex, primary motor cortex, or a control zone over the occipital lobe. Tests for retention and relearning were conducted after a 24 h delay. Analysis of movement kinematic measures and force-channel trials found that cTBS to somatosensory cortex disrupted both learning and subsequent retention, whereas cTBS to motor cortex had little effect on learning but possibly impaired retention. Basic movement variables are unaffected by cTBS suggesting that the stimulation does not interfere with movement but instead disrupts changes in the cortex that are necessary for learning. In all experimental conditions, relearning in an abruptly introduced force field, which followed retention testing, showed extensive savings, which is consistent with previous work suggesting that more cognitive aspects of learning and retention are not dependent on either of the cortical zones under test. Taken together, the findings are consistent with the idea that motor learning is dependent on learning-related activity in the somatosensory cortex.

NEW & NOTEWORTHY This study uses noninvasive transcranial magnetic stimulation to test the contribution of somatosensory and motor cortex to human motor learning and retention. Continuous theta-burst stimulation is applied before learning; participants return 24 h later to assess retention. Disruption of the somatosensory cortex is found to impair both learning and retention, whereas disruption of the motor cortex has no effect on learning. The findings are consistent with the idea that motor learning is dependent upon learning-related plasticity in somatosensory cortex.

motor cortex; motor learning; retention; somatosensory cortex

INTRODUCTION

The brain areas that are active during human motor learning have been identified in neuroimaging studies, using reinforcement, sequence learning, and adaptation procedures. Extensive involvement of the frontal and parietal cortex, basal ganglia, and cerebellum has been observed (for reviews, see Refs. 1–8). These studies, although successful in identifying the aggregate motor learning network, share a common limitation, namely, uncertainty about whether individual areas are active because of their causal contribution to learning or, instead, because they communicate with one of these critical zones. In the present study, we test for causal involvement in learning by applying continuous theta-burst magnetic stimulation (cTBS) before a force-field adaptation task to the motor or somatosensory cortex or a control zone over the occipital lobe. If the motor or somatosensory cortex contributes directly to learning, the disruption of their activity should adversely affect learning or retention or possibly both.

There is considerable evidence for the involvement of somatosensory cortex, and parietal cortex more generally, in the control of movement and motor learning. In electrophysiological studies with non-human primates, activity in the somatosensory cortex is recorded well before the onset of movement, and well before the initiation of muscle contraction (9–11). Movements can be elicited from stimulation throughout the somatosensory cortex and parts of the posterior parietal lobe (12, 13). In neuroimaging studies with humans, planned movements can be decoded from activity in the somatosensory cortex as effectively as from cortical

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motor areas (14, 15). Other recent work points to a central role of the somatosensory cortex in learning-related plasticity. Changes in cortical excitability during motor learning occur first in the somatosensory cortex and predict subsequent learning (16). In other work, it is seen that somatosensory training and plasticity facilitate motor learning (17–19). Somatosensory plasticity also supports motor learning by observation (20). In the work on learning and motor memory stabilization, disruption of the somatosensory cortex blocks motor learning and the retention of newly learned movements (21, 22). Together these studies point to the involvement of the somatosensory cortex in the cortical control of movement and to somatic plasticity as central to motor learning and retention.

We directly assess somatosensory and motor cortex participation in learning using a force-field adaptation task. cTBS is applied, following baseline movements, to one of these areas or a control zone. Participants subsequently train to make arm movements to a visually presented target while a gradually introduced velocity-dependent force is applied to the limb. Tests of retention and relearning are conducted following adaptation, after a 24-h delay. Disruption of the somatosensory cortex is found to interfere both with learning and subsequent retention. As reported previously, motor cortex disruption has little effect on learning. However, a retention decrement relative to control participants may be present 24 h later. The present findings taken in combination with work documenting the involvement of somatosensory cortex in the retention of newly learned movements (21), provide support for the idea that learning-related plasticity in somatosensory cortex is central to motor learning and that somatosensory and possibly motor cortex are together responsible for retention.

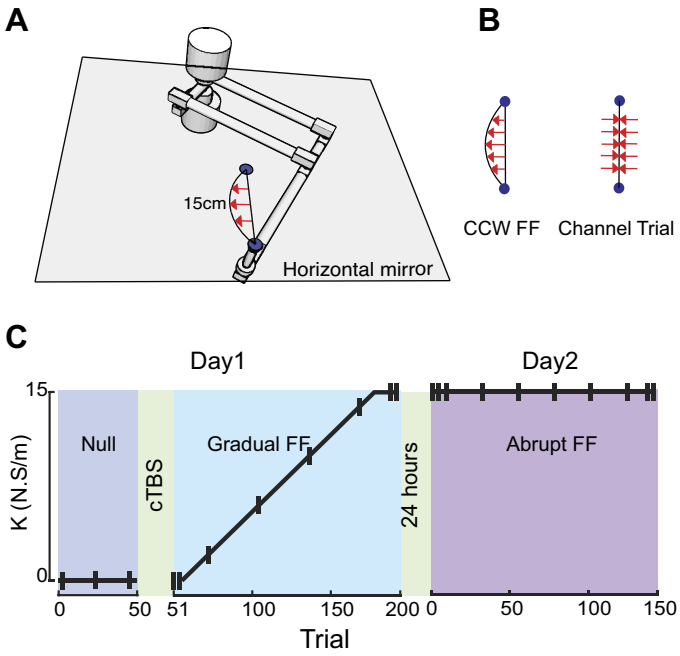

Figure 1. Experimental setup and design. *A*: participants held the handle of a robot arm and made center-out movements in a velocity-dependent force field. *B*: learning and retention are evaluated using perpendicular deviation (PD) at maximum hand velocity (*left*) and force applied to the channel walls during error-clamp trials (*right*). *C*: the experiment is run over the course of 2 days. On the first day, baseline movements (null) are followed by cTBS to one of the motor or somatosensory cortex or to a control zone over the occipital lobe. This is followed by training movements in a gradually introduced force field, with error-clamp trials interspersed. Retention and relearning in an abruptly introduced force field are tested 24 h later. CC, counterclockwise; cTBS, continuous theta-burst magnetic stimulation; FF, force field. *K* indicates the strength of the applied force as in *Eq. 1*.

MATERIALS AND METHODS

Participants

Sixty right-handed adults (39 females), between the ages of 18 and 31 (21.8 ± 2.4) yr, participated in the study. Participants reported no history of sensory or motor disorder and were naïve regarding the experimental procedures. The study protocol was approved by the McGill University Faculty of Medicine Research Ethics Board and written informed consent was obtained.

Experimental Setup

During the behavioral task, participants were seated in front of a two-degree-of-freedom planar robotic arm (InMotion2, Interactive Motion Technologies Inc.) and grasped the handle of the robot with their right hand (Fig. 1*A*). For each participant, the position of the seat relative to the robot was adjusted to yield a shoulder angle of 45° (relative to the frontal plane) and an elbow angle of 90° (relative to the upper arm). This posture corresponds to the middle of the workspace of the robot and defines the start position of all reaching movements. The seat height was adjusted to have 80° of shoulder abduction. The participant's arm was supported against gravity by an air sled and a harness restrained the subject's shoulder and upper body. Hand position was measured using two 16-digit optical encoders (Gurley Precision Instruments) located in the robot arm. The force applied to the robot handle by the participant was measured using a force-torque sensor (ATI Industrial Automation) that was mounted above the manipulandum handle. Position and force data were sampled at 400 Hz. A semisilvered mirror was positioned halfway between a horizontal monitor and the robot handle, immediately below eye level. In this way, participants' vision of their arm and the robot handle was blocked. Participants held the robot handle and performed point-to-point reaching movements. Start and target positions were indicated with 20-mm diameter white circles, which were projected onto the mirror along the body midline of the participant. The target position was 15 cm in front of the start position. The position of the participant's hand while holding the robot was displayed as a 5-mm diameter yellow circle.

At the beginning of each trial, the robotic arm brought the participant's hand to the start position. The participant was required to hold the handle in the start position for a random delay (500 ± 500 ms). When the cursor turned green, participants were asked to move to the target in one smooth movement. They were told that this was not a reaction time task, but they must finish their reaching movement in 700 ms (700 ± 50 ms) after they started. At the end of each movement, participants received color-coded visual feedback based on their movement duration. The feedback was used only to encourage subjects to move at the desired duration, but no trials

were dropped from the analysis due to not satisfying this criterion. At the end of each trial, the robot moved the participant's hand back to the start position. The visual cursor was turned off during the return movement.

In total, each participant performed 350 reaching movements over two consecutive days (Fig. 1*C*). *Day 1* (training day) started with 50 trials under null-field conditions (baseline phase). These trials were followed by continuous theta-burst stimulation (cTBS). Participants were then asked to perform 150 more reaching movements, which involved the gradual introduction of a counterclockwise curl field (training phase). The curl field was applied according to *Eq. 1*:

$$\begin{bmatrix} f_x \\ f_y \end{bmatrix} = \begin{bmatrix} 0 & -k \\ k & 0 \end{bmatrix} \begin{bmatrix} v_x \\ v_y \end{bmatrix}. \quad (1)$$

In this equation, x and y are lateral and sagittal directions (Fig. 1*A*), f_x and f_y are applied force to the hand due to the robot in N, and v_x and v_y are hand velocities in m/s. The strength of the applied force depended on k (N·s/m). On *day 1*, k increased linearly from 0 to 15 over the first 135 trials and was then kept at 15 for the last 15 trials. On *day 2* (retention phase), k was set to 15 throughout, which resulted in an abrupt introduction of load. Channel trials (error-clamp trials) were interspersed throughout each experimental block. The position of channel trials was held constant for all participants (vertical lines in Fig. 1*C*). In these trials, the lateral deviation of the hand was restricted by the robot while the participant could move freely in the sagittal direction. This was done with the help of a stiff force channel, with a stiffness of 4,000 N/m and viscosity of 40 N·s/m.

There is little lateral force to the channel walls during baseline movements. However, during the gradual introduction of load, participants learn to compensate for the counterclockwise force field by applying forces in the opposite direction. The applied force to the channel walls serves as a measure of the amount of force field that is compensated for due to training. Moreover, channel trials at the start of the second day (retention phase) provide a measure of how much of the original learning is retained after 24 h.

Continuous Theta-Burst Stimulation

cTBS has been shown to disrupt neural activity (21, 23–25) and is used here to investigate the role of three candidate regions in motor learning and the retention of motor memory. cTBS was applied on *day 1*, right after participants finished reaching movements under baseline conditions. A Magstim Super Rapid^2 TMS system (Magstim, Whitland, UK) with a 70-mm figure eight coil was used for stimulation. The Brainsight neuronavigation system (Rogue Research, Montreal, Canada) was used for coil positioning and tracking. A single-pulse transcranial magnetic stimulation (TMS) unit (Magstim200) was used to elicit motor-evoked potentials (MEPs) from the biceps brachii. At the beginning of the cTBS session, participants were randomly assigned to one of three experimental conditions: S1, M1, or stimulation to a control zone over the occipital cortex (20 participants in each condition). For the M1 condition, we applied cTBS to the position on the participant's scalp at which the biceps were most excitable (hot spot). The hot spot was identified by having participants hold their forearms at 90°, parallel to the floor and against gravity. In pilot work, we found that this results in ~5% of maximum voluntary contraction (MVC) of the biceps. In the S1 condition, we used the MNI coordinates of the biceps' representation in the primary somatosensory cortex as reported previously (26). Two trains of cTBS pulses (10 min apart) were applied to the targeted area. Each cTBS block comprised 600 pulses in total, in which three pulses at 50 Hz were repeated five times a second. Based on a previous study (21) and our own pilot participants, we used 40% of the maximum Magstim output as the intensity of cTBS stimulation for all participants.

Data Analysis

Hand position and forces applied to the robot handle were sampled at 400 Hz. The resulting signals were low pass filtered at 40 Hz with a zero-phase lag Butterworth filter. Position signals were numerically differentiated to provide velocity values. On each trial, movement start was scored at 5% of peak hand velocity. Movement end was defined as the time at which tangential velocity dropped below 5% of its maximum and stayed there for at least 100 ms. The perpendicular deviation of the hand (PD) from a straight-line connecting movement start and end positions was calculated as a dependent measure of learning. We used ANOVA to compare, between experimental conditions, the average PD over the last 50 trials of the learning phase relative to the 50 trials of the baseline. In tests of savings at the beginning of the relearning trials, we tested for differences in PD using the mean of trials 5 to 10. This was done so as not to consider transient effects following the abrupt reintroduction of load. Following a significant ANOVA, we used the Holm–Bonferroni correction in combination with one-tailed post hoc comparisons.

During channel trials, lateral forces applied to the channel walls provide a measure of the extent to which the force field is compensated for by the participant. Channel trials were interspersed randomly during baseline, motor learning, and retention blocks. For each channel trial, we calculated the time-varying force applied to the channel walls and the force profile that would be needed to fully compensate for the action of the robot (ideal force). We calculated the slope of linear regression between the ideal and actual lateral force, up to the point of maximum velocity. This was done to minimize the contribution of possible online corrections to this measure. A slope of one corresponds to full compensation for the force field while a slope of zero indicates no compensation at all. Differences in the regression coefficient between conditions were assessed using ANOVA followed by Holm–Bonferroni corrected one-tailed post hoc comparisons. For statistical tests involving channel trials, all three channel trials during the baseline phase were used. For late learning, we used the two last channel trials, which were in the plateau phase of force application. For retention, we used the two-channel trials at the beginning of *day 2*, before relearning.

RESULTS

We sought to determine whether disruption of the primary motor cortex or primary somatosensory cortex using cTBS impairs human motor learning or retention. cTBS was

applied immediately after participants finished baseline null field movements and before the gradual introduction of a velocity-dependent force field (Fig. 1*C*). A third set of participants served as a control group. In this condition, stimulation was delivered over the occipital lobe. Figure 2*A* shows the average perpendicular deviation (PD) of the participants' hand from a straight line connecting the start and target points during baseline movements and gradual force-field adaption. As can be seen, reaching movements in all three experimental conditions were roughly straight during baseline movements (values near zero). Ten minutes after the end of the cTBS procedure, all participants made movements in a gradually introduced force field (learning phase). The force-field strength increased from zero to a maximum value over the course of the first 135 trials and was then kept at the maximum force for 15 further trials. It can be seen that early in the force-field block all participants were able to fully compensate for the force field. However, starting approximately halfway through the force-field sequence, S1 participants' performance grew progressively worse than that of the other two experimental conditions, as indicated by less complete compensation for the load (greater deviation values). Figure 2*B* shows the average change in PD relative to the baseline trials for all three experimental conditions. Average PD is seen to be greater in the S1 condition than in the other experimental groups. A one-way independent samples ANOVA found a significant difference between conditions [$F(2,57) = 3.53$, $P = 0.03$, $\omega^2 = 0.08$]. Post hoc comparisons indicated a significant difference in performance at the end of the learning phase between the S1 and control conditions ($P < 0.05$) and also between the S1 and M1 conditions ($P < 0.03$).

cTBS had little effect on participants' ability to perform the movements themselves (Supplemental Fig. S1). We measured maximum hand velocity, the path length of the movement, and movement duration both during the 50 trials of the baseline phase and during the last 50 trials of the learning phase of the experiment. Participants moved faster during the learning phase (0.383 m/s ± 0.015) than in the baseline (0.379 m/s ± 0.014) [$F(1,57) = 4.89$, $P = 0.03$, $\omega^2 = 0.02$]. Movement path lengths were also longer during learning (15.9 ± 0.7 cm vs. 15.4 ± 0.3 cm) [$F(1,57) = 66.2$, $P < 0.001$, $\omega^2 = 0.2$] and movement duration was greater (790 ± 60 ms vs. 740 ± 35 ms [$F(1,57) = 68.2$, $P < 0.001$, $\omega^2 = 0.24$]. However, there was no difference between experimental conditions, and none of these differences from baseline during learning depended upon the site of stimulation, that is, there was no interaction with experimental conditions ($P > 0.05$ in all cases). The differences observed between baseline and training movements are presumably due to the presence of a force field in each of the experimental conditions and are not a consequence of interference due to cTBS with the movements themselves.

Participants' learning and retention of the motor task were also assessed using force-channel trials. Figure 3*A* shows the average lateral force applied to the channel wall (green) along with the ideal force (blue) during baseline, late in learning, and during retention tests for all three conditions. Note that participants' reaching movements were in all cases in the anterior-posterior direction. As can be seen, little force is applied to the channel walls during the baseline phase. In contrast, toward the end of learning, participants in all conditions substantially compensate for the applied force. In tests of retention of learning at the beginning of the second day (first two-channel trials), participants in the control condition are seen to have retained more of the previous learning than those in the other two conditions. We calculated the slope relating to the force profiles (the regression of applied force on ideal force); a regression coefficient of 1 shows full compensation of the force field, whereas a regression coefficient of 0 indicates the absence of compensation at all. Figure 3*B* shows the average regression coefficient for the three phases of the experiment. The pattern mirrors that of Fig. 3*A*; low force under baseline conditions; substantial compensation at the end of learning; and differential retention of learning 24 h later, with the S1 and possibly M1 conditions showing less retention than participants in the control condition.

A two-way repeated-measures ANOVA found that retention on *day 2* was less than learning at the end of *day 1* [$F(1,57) = 141.6$, $P < 0.001$, $\omega^2 = 0.45$). There was also a significant difference between experimental conditions in the amount of force-field compensation [$F(2,57) = 3.49$, $P = 0.04$, $\omega^2 = 0.03$). Holm–Bonferroni-corrected multiple comparisons found impaired performance in S1 relative control participants ($P < 0.05$). It was also found that the patterning of applied force in the different experimental conditions differed for the first and second sessions [an interaction between experimental conditions and sessions, $F(2,57) = 3.7$, $P = 0.03$, $\omega^2 = 0.02$]. A simple effects analysis on the first day's data found no differences between experimental conditions [$F(2,57) = 0.5$, $P = 0.61$, $\omega^2 = 0.000$], whereas there were significant differences during the retention session [$F(2,57) = 5.09$, $P = 0.009$, $\omega^2 = 0.12$]. In the retention tests, Holm–Bonferroni-corrected multiple comparisons found a significant difference between S1 and control participants ($P < 0.01$) and also

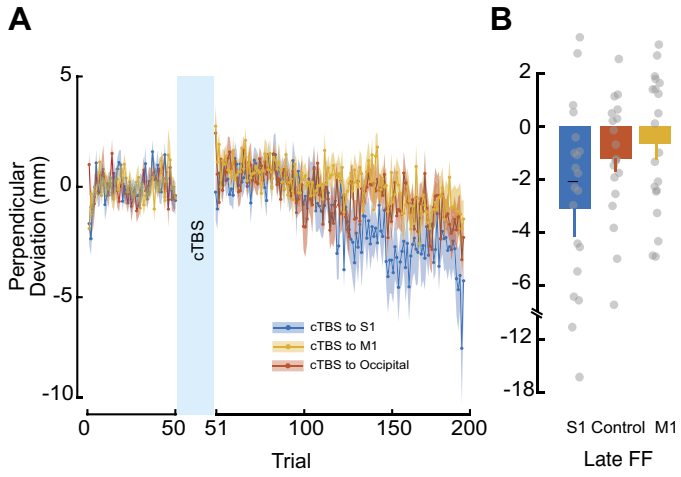

Figure 2. cTBS to somatosensory cortex disrupts motor learning. *A*: kinematic measures of learning (PD ± standard error). Perfect adaptation would be reflected in straight movements throughout (deviations close to zero). cTBS to M1 shows little disruption of learning in comparison with cTBS to the control zone. The gradual increase in PD reflects incomplete compensation for the force field. cTBS to S1 results in a progressive impairment in adaptation. *B*: mean deviation (± standard error), relative to baseline, late in force-field adaptation (trials 150 to 200 relative to trials 1 to 50). Dots show individual subjects. cTBS to S1 results in significantly less adaptation. cTBS, continuous theta-burst magnetic stimulation; PD, perpendicular deviation.

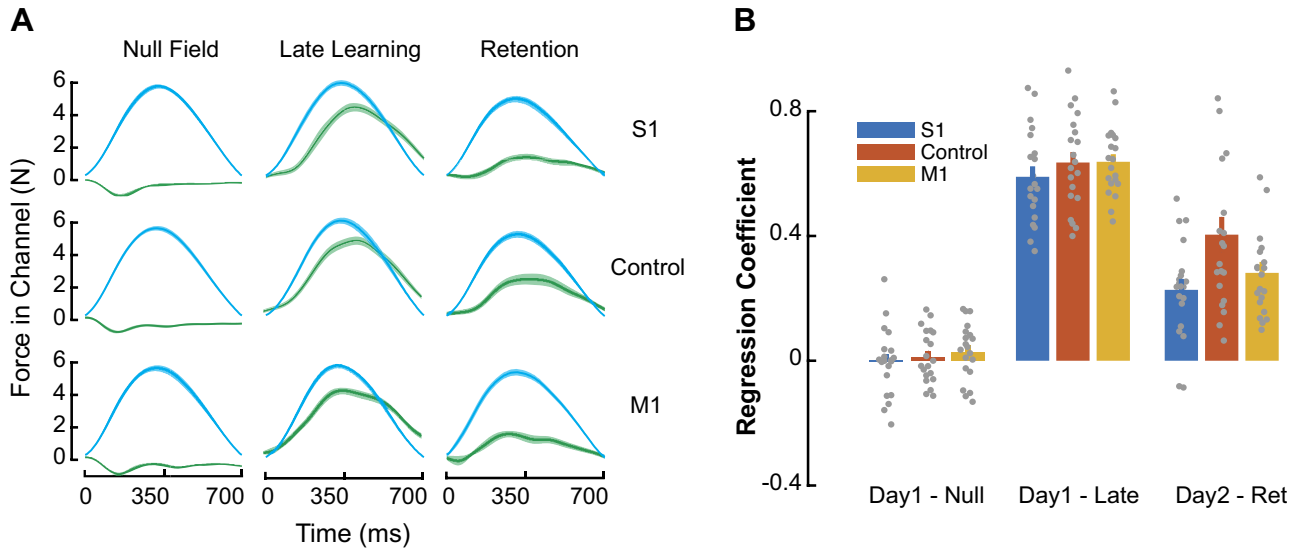

Figure 3. Force-channel trials indicate less retention 24 h after initial learning, following cTBS to S1. *A*: force channel trials showing ideal (blue) and actual (green) force profiles (means ± standard error). *B*: average regression coefficient values in channel trials during baseline (*day 1* Null, last two channel trials), at the very end of *day 1* training (*day 1* Late, last two channel trials), and during retention tests at the start of *day 2* (*day 2* Ret, first two channel trials). Retention on *day 2* is less when cTBS is applied to S1 prior to *day 1* training. cTBS, continuous theta-burst magnetic stimulation.

between M1 and control participants ($P < 0.02$). There was no evidence of a difference between the M1 and S1 conditions ($P > 0.05$). The pairwise comparisons were also analyzed using Bayesian statistics. In these analyses, there was substantial evidence for a difference between the S1 and control conditions ($BF_{10} = 6.23$), whereas, for the other two comparisons, M1 versus control, and M1 versus S1, the evidence was inconclusive ($BF_{10} = 1.55$ and 0.50, respectively).

On the second day of the experiment, following the first two-channel trials, savings of initial learning was tested; participants performed a block of reaching movements in the presence of full force-field strength (abrupt introduction of load). Figure 4 shows the reaching movements' PD during this phase of the experiment. It can be seen that apart from the first few movements (PDs for the first 5 movements are shown in Fig. 4*A*), there is extensive relearning in all experimental conditions (Fig. 4*B*). A one-way independent samples ANOVA on first three movements found a marginally significant difference between experimental conditions [$F(2,57) = 2.94$, $P = 0.06$, $\omega^2 = 0.06$], attributable by post hoc comparisons with a difference between the S1 and control conditions ($P = 0.06$). The same analysis was repeated for trials 6–10 where no evidence of a difference between conditions was obtained [$F(2,57) = 1.91$, $P = 0.16$, $\omega^2 = 0.03$]. In addition, we conducted a two-way repeated-measures ANOVA to compare trials 6–55 with trials 101–150. We found a small but reliable change in PD such that deviation was greater (−3.27 ± 0.51 mm vs. −2.87 ± 0.47 mm) at the end of relearning [$F(1,57) = 4.64$, $P = 0.035$ $\omega^2 = 0.002$]. There was no indication that the pattern differed for the three conditions [$F(2,57) = 0.34$, $P = 0.71$, $\omega^2 < 0.001$]. To summarize, there is exceptionally rapid and near-complete relearning under conditions of abrupt reintroduction of load, apart from a possible transient difference at the start of the relearning block.

Correlation analyses were conducted to assess possible relationships between the different measures that were obtained on the two days of the experiment. Recall that on *day 1* an initial kinematic measure of learning was followed by channel trials at the very end of the learning, where the force field at this point was at maximum strength. On *day 2*, initial channel trials, with no other force inputs, were followed by tests of relearning in which the force field was reintroduced abruptly. It was found that kinematic measures of learning as assessed using differences in PD from early (first 5 trials) to late in learning (last 5 trials) on *day 1* were correlated with measures of retention as measured in channel trials at the start of *day 2* ($r_{58} = -0.25$, $P = 0.05$, Fig. 4*C*). No other correlations between *day 1* and *day 2* performance were statistically significant, nor were the first and second measures on each day correlated ($P > 0.05$ in all cases).

DISCUSSION

The effects on motor learning and retention of disruption of either primary motor cortex or primary somatosensory cortex were tested using a force-field adaptation task. The load was introduced gradually on an initial training day to minimize awareness of the perturbation and then 24 h later, after tests of retention, the force field was reintroduced abruptly to evaluate relearning. It was observed that for participants who received cTBS to the somatosensory cortex, there was significantly less compensation for the applied force field during initial training, in comparison with subjects tested following cTBS to the motor cortex or to a control zone over the occipital lobe. This progressive impairment in performance was present for much of the somatosensory condition and thus provides evidence that the somatosensory cortex contributes directly to motor learning. The kinematic differences between conditions also serve to dissociate the roles of the somatosensory and motor cortex in learning, and likewise argue against the possibility that somatosensory cortex stimulation indirectly affects the motor cortex.

Retention and relearning were assessed 24 h after initial training. Retention testing was conducted at the start of the

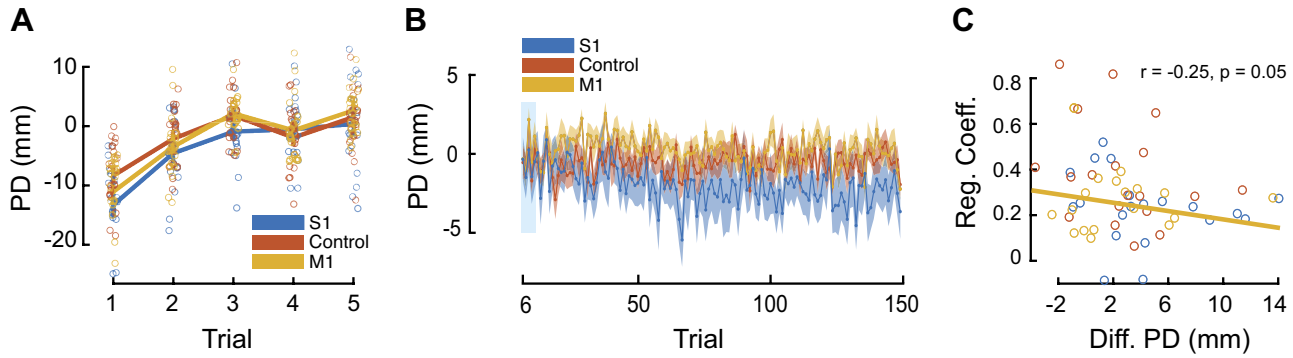

Figure 4. There are no differences in kinematic measures of relearning in an abruptly introduced force field, apart from possible transient differences over the first relearning trials. *A*: S1 participants show less retention than controls over the first three trials. Dots show individual subjects. *B*: overlay of trials 6 to 150. Lateral deviation (means ± standard error) during relearning is close to zero in each condition (full compensation). The highlighted band shows trials 6 to 10. *C*: differences in PD from early to late in learning on *day 1* (trials 196 to 200 vs. trials 51 to 55) are correlated with measures of retention as measured in channel trials at the start of *day 2*. Dots show individual subjects, color-coded by experimental condition. PD, perpendicular deviation.

second session and was based on forces applied during force-channel trials without any other force application. Relearning was tested afterward, using abruptly introduced loads with the goal of evaluating both implicit learning and any cognitive strategies that may have been engaged. Retention tests revealed a substantial loss of information when cTBS was applied to the somatosensory cortex and a smaller loss of information following cTBS to the motor cortex. Following cTBS to the somatosensory cortex, retention was ~50% of that in the control condition, whereas, following cTBS to the motor cortex, retention averaged 65% relative to controls. The drop in retention for the somatosensory cortex presumably reflects both the initial impairment in learning and possibly an additional loss of information related to motor memory stabilization. But, in the case of motor cortex, any effects seem wholly related to retention and could indicate motor cortex participation, as has been previously demonstrated for somatosensory cortex (21).

In tests of relearning, differences between conditions were observed over the first few trials, with participants in the somatosensory cortex condition showing less retention in comparison with those in the control condition. However, these differences were transient; afterward, and throughout the remainder of the relearning test, participants in all groups showed almost complete savings of prior learning. The extremely rapid relearning is striking. A tentative explanation is that the abruptly introduced loads, which were used to test for relearning, serve to engage cognitive contributions or strategies that come into play almost immediately after the introduction of the load. The similarity of the pattern of relearning to that in the control condition suggests that these rapid relearning effects (savings) are not associated with either cortical motor or somatosensory areas.

The data from the present study accord well with the idea that plasticity in the somatosensory cortex contributes to motor learning. This idea is supported by previous work in which cTBS was applied to the somatosensory cortex following learning, rather than before learning as in the present study, with the finding that motor memory consolidation was disturbed (21). This indicates that there were learning-related changes to the somatosensory cortex whose stabilization was blocked. The fact that the disruption of somatosensory and possibly motor cortex impairs the retention of learning would be consistent with the idea that movements are jointly encoded in both cortical structures. The finding that upcoming movements can be decoded in the somatosensory cortex at essentially the same time as in the motor cortex (27) is consistent with the view that somatosensory and motor cortex jointly control movement, as is neuroimaging work with humans, in which upcoming movements can be decoded as effectively from activity in the somatosensory cortex as from that in the motor cortex (14, 15).

Somatosensory cortex participation in movement might take the form of inputs to the motor cortex (28–33), which provide intended somatic states or targets. It is also possible that the somatosensory cortex may contribute directly to the efferent control of movement. There is both neuroanatomical and electrophysiological evidence in support of this latter possibility. In non-human primates, ~60% of the corticospinal tract originates in Brodmann areas 3a, 3b, 1, 2, 5, and the second somatosensory cortex (34) with the densest terminations in lamina IV to VII in the intermediate zone of the spinal cord (35). Corticospinal outputs from posterior area 2 and area 5 have been shown to terminate on last-order interneurons in this region, which, in turn, project monosynaptically onto α motor neurons involved in the movement of the fingers and hands (13). In addition, some postcentral neurons change activity well in advance of movement, early enough to rule out the possibility that they are active in response to peripheral muscle activity (10, 11). In rodents, long-train intracortical stimulation of the somatosensory cortex results in the production of coordinated movements, which differ from those produced when comparable stimuli are delivered to the motor cortex suggesting that both structures participate in the control of movement (36). Moreover, in non-human primates, movements can be elicited throughout the somatosensory and posterior parietal cortex using this kind of stimulation (12).

Learning and retention were each assessed using kinematic measures (lateral deviation at maximum velocity) and error-clamp trials (force applied to the channel walls). The data suggest that the tests at the beginning and end of each day tap into different aspects of learning. On *day 1*, testing with kinematics occurred first followed by testing with

channel trials, whereas on *day 2*, the channel trials were done first. The initial tests each day provide measures of implicit learning; the latter tests engage cognitive and strategic aspects of force-field performance. Kinematic measures on *day 1*, in which the force field was introduced gradually, show a progressive impairment in performance following cTBS to the somatosensory cortex. Correspondingly, the channel trials that were done at the beginning of *day 2*, in which no load was applied, mirror this pattern, with impaired performance in retention tests. The finding that kinematic performance at the end of *day 1* correlated with retention measured in channel trials on *day 2* is consistent with both measures tapping into similar underlying processes. In contrast, the tests at the end of each day show a different pattern in which there is extensive compensation in all experimental conditions. In the relearning trials on *day 2*, the force field is at full strength throughout. The exceptionally rapid compensation in these trials is more likely dependent on strategic intervention than learning. The fact that patterns of force and kinematics seen in these trials are unaltered by cTBS to motor or somatosensory cortex is consistent with previous work suggesting that explicit contributions to learning are not dependent upon either of these cortical structures (21).

The effects of cTBS on learning and retention in the present study are consistent with other reports of the effects of magnetic brain stimulation on sensorimotor adaptation. In the present study, disruption of the somatosensory cortex impaired both learning and motor memory retention, which is consistent with previous work on force-field learning with rodents (22) and motor skill learning and consolidation of force-field adaptation in humans (21, 37). It was also observed that cTBS to the motor cortex had no observable effect on learning but may have disrupted subsequent consolidation. This latter result would be consistent with other work on adaptation learning in which single-pulse TMS to the primary motor cortex had no effect on learning with gradually introduced visuomotor (38, 39) or force field perturbations (40) but disrupted consolidation as measured 24 h later (39). A similar pattern of normal learning and impaired retention the next day was observed following repetitive TMS (rTMS) to the primary motor cortex, in the context of force-field adaptation (41). In one conflicting result, no effects on the retention of force-field adaptation were observed when rTMS was applied following learning to the motor cortex (42). However, in this case, tests of retention were conducted without an intervening delay.

It would be informative to follow the effects of stimulation over time. Based on the present results, it is expected that, at intermediate delays, following disruption of S1, retention would be little different than that observed at 24 h. This is because performance is impaired in the present data regardless of the timing of testing in relation to S1 stimulation. M1 could be more informative in that there are no effects of M1 disruption on learning but, after 24 h, retention may be impaired. Previous work suggests that 4 h is sufficient for consolidation (43) so a test at an intermediate delay might be another way to assess this estimate. For relearning measures, it is probable that intermediate delays would be a little different than what is presently seen. The reason here is that there is rapid relearning even following the disruption of a control zone, which suggests that relearning is mediated by regions other than S1 or M1.

In conclusion, the present study provides evidence that the somatosensory cortex contributes to motor learning. When the somatosensory cortex is disrupted, learning is impaired. Following stimulation, the movements themselves remain normal, indicating that the effects are specific to learning. Effects on retention, measured after a 24-h delay, are observed following disruption of somatosensory and possibly motor cortex as well. This latter finding would be consistent with the idea that newly learned movements are jointly encoded and presumably jointly controlled by both cortical structures.

DATA AVAILABILITY

Data will be made available upon reasonable request.

SUPPLEMENTAL DATA

Supplemental Fig. S1: https://doi.org/10.6084/m9.figshare.24324778.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

M.D. and D.J.O. conceived and designed research; M.D. and T.F.M. performed experiments; M.D. and T.F.M. analyzed data; M.D. and D.J.O. interpreted results of experiments; M.D. prepared figures; M.D. and D.J.O. drafted manuscript; M.D. and D.J.O. edited and revised manuscript; T.F.M. and D.J.O. approved final version of manuscript.

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