Effects of TMS over Premotor and Superior Temporal Cortices on Biological Motion Perception

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Abstract

■ Using MRI-guided off-line TMS, we targeted two areas implicated in biological motion processing: ventral premotor cortex (PMC) and posterior STS (pSTS), plus a control site (vertex). Participants performed a detection task on noise-masked point-light displays of human animations and scrambled versions of the same stimuli. Perceptual thresholds were determined individually. Performance was measured before and after 20 sec of continuous theta burst stimulation of PMC, pSTS, and control (each tested on different days). A matched nonbiological object motion task (detecting point-light displays of translating polygons) served as a further control. Data were analyzed within the signal detection framework. Sensitivity (d') significantly decreased after TMS of PMC. There was a marginally significant decline in d' after TMS of pSTS but not of control site. Criterion (response bias) was also significantly affected by TMS over PMC. Specifically, subjects made significantly more false alarms post-TMS of PMC. These effects were specific to biological motion and not found for the nonbiological control task. To summarize, we report that TMS over PMC reduces sensitivity to biological motion perception. Furthermore, pSTS and PMC may have distinct roles in biological motion processing as behavioral performance differs following TMS in each area. Only TMS over PMC led to a significant increase in false alarms, which was not found for other brain areas or for the control task. TMS of PMC may have interfered with refining judgments about biological motion perception, possibly because access to the perceiver's own motor representations was compromised. ■

INTRODUCTION

The perception of others' body movements is important for many tasks of biological significance. Despite intense interest in how the brain supports this ability, there are many unknowns about the underlying perceptual processes and neural systems. Studies have revealed a network of brain areas involved in biological motion perception (e.g., Saygin, in press; Grosbras, Beaton, & Eickhoff, 2012; Pelphrey, Morris, Michelich, Allison, & McCarthy, 2005; Peuskens, Vanrie, Verfaillie, & Orban, 2005; Saygin, Wilson, Hagler, Bates, & Sereno, 2004; Vaina, Solomon, Chowdhury, Sinha, & Belliveau, 2001; Grossman et al., 2000). The posterior STS (pSTS) was proposed to be the key area in several neuroimaging and neurophysiological studies of biological motion (Wyk, Hudac, Carter, Sobel, & Pelphrey, 2009; Puce & Perrett, 2003; Oram & Perrett, 1996). Although vision researchers have mostly focused on posterior areas, there is a related body of literature that has put emphasis on premotor cortex (PMC). In the macaque monkey, the ventral PMC contains mirror neurons, which fire during action execution as well as observation (Rizzolatti & Craighero, 2004; Gallese, Fadiga, Fogassi, & Rizzolatti, 1996). The network of brain areas that support action and biological motion perception in the human brain (the pSTS, PMC, and the anatomical link between the two: the inferior parietal lobe; Matelli & Luppino, 2001) is often called the mirror neuron system. Because our interest is not limited to mirror neurons, here, we use the more neutral term "action perception system" (APS) to refer to this network.

Body movements can be represented with just a few markers (point-lights) attached to the limbs of a person (Johansson, 1973). When in motion, these sparse point-light displays (PLDs) can vividly depict actions as well as information such as gender, identity, and emotions (Pollick, Paterson, Bruderlin, & Sanford, 2001; Cutting & Kozlowski, 1977; Kozlowski & Cutting, 1977). Texture and form cues per se are absent in PLDs, so these stimuli are well suited to study the contribution of motion signals to body movement perception. PLDs have an established history in vision science (Blake & Shiffrar, 2007), and there are well-characterized control stimuli to use in experiments (such as "scrambled" PLDs; see Methods).

Given that PLDs can evoke action percepts, are they also processed in the PMC? Or are motion signals alone insufficient to drive neural responses in this area? Using fMRI, we previously reported that ventral PMC was as selective for biological motion as the pSTS (Saygin, Wilson, Hagler, et al., 2004). However, it is difficult to

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infer causal links between brain and behavior from fMRI studies. There is a small literature with neuropsychological patients on the perception of biological motion perception, but lesion-deficit relationships are highly heterogeneous (e.g., Saygin, 2007, in press; Sokolov, Gharabaghi, Tatagiba, & Pavlova, 2010; Saygin, Wilson, Dronkers, & Bates, 2004; Battelli, Cavanagh, & Thornton, 2003; Schenk & Zihl, 1997). To make reliable lesion-deficit inferences, patient studies require large sample sizes (Bates et al., 2003); in a study with 60 stroke patients, lesion sites most strongly associated with deficits in biological motion perception included the pSTS and the PMC (Saygin, 2007).

TMS can aid in making causal links between brain and behavior (Walsh & Cowey, 2000). TMS allows targeting brain regions more specifically than neuropsychological studies. To our knowledge, there is only one TMS study of biological motion perception, which reported that TMS over pSTS (but not over MT+/V5) reduces sensitivity to PLDs of biological motion perception (Grossman, Battelli, & Pascual-Leone, 2005). It is unknown whether TMS over PMC affects biological motion perception, although it can impact other aspects of action perception (e.g., Chouinard & Paus, 2010; Candidi, Urgesi, Ionta, & Aglioti, 2008; Urgesi, Candidi, Ionta, & Aglioti, 2007; Pobric & Hamilton, 2006).

In Experiment 1 (which consisted of three sessions), we used PLDs, established psychophysical paradigms, and targeted pSTS, PMC, and a control site (vertex) with continuous theta burst (cTBS) TMS to test whether biological motion processing is dependent on these regions and, more generally, to explore functional properties of these nodes of the APS. In Experiment 2, we investigated whether effects of TMS over PMC were specific to biological motion or might generalize to nonbiological object motion.

METHODS

Participants

Subjects were right-handed adults aged 19–29 years (mean = 22.6 years). Twelve adults completed all three TMS sessions (pSTS, PMC, and vertex). Fifteen participants started Experiment 1. One participant discontinued after the practice session because of discomfort from the TMS; two subjects did not come to their third session for unspecified reasons. Each site was stimulated on a separate day, and the order of sessions was varied across subjects. Nine additional subjects participated in Experiment 2. The study was approved by the local ethics board. All subjects were checked against TMS exclusion criteria (Wassermann, 1998) and gave written informed consent.

Stimuli

Biological motion stimuli were created by videotaping an actor performing several full body actions and encoding the joint positions on the digitized videos (Ahlstrom, Blake, & Ahlstrom, 1997). Stimuli were 11 PLDs depicting walking, jogging, stepping up, stepping aside, low kicking, side kicking, high kicking, high throwing, middle throwing, underarm throwing (bowling), and skipping. An example frame (from a walking motion) is shown in Figure 1. The joints were represented with 12 small white dots against a black background. PLDs subtended approximately 5.5° × 7.7° of visual angle when viewed from 52 cm.

Scrambled PLDs were used for target-absent trials (see below), which were created by randomizing the starting positions of the points while keeping the same motion trajectories. They contained the same local motions but did not have the global form and action percept as the biological motion animations (e.g., Saygin, 2007; Saygin, Wilson, Hagler, et al., 2004; Grossman et al., 2000). The area occupied by the scrambled PLDs was kept of the same size as that of the intact PLDs. Eleven scrambled animations matched to each action were used consistently.

For the nonbiological control study (Experiment 2), we used point-light shapes that were composed of 12 white dots of the same size as those used on the biological motion PLDs. An example shape (a diamond) is shown in Figure 1. Nonbiological motion stimuli translated at a fixed speed (see Procedure). The nonbiological stimuli were also presented scrambled, where the same number of points translated with the same motion trajectory as the target animations but with the positions of the points scrambled such that the points did not comprise a recognizable polygon shape.

In each trial, the PLDs were presented with "noise" dots, with the number determined as described below (Figure 1B, C, E, and F). The more noise dots are present, the more difficult the task becomes. In each trial, each noise dot had the same trajectory as one of the dots from the PLD. The area in which the PLDs and the noise dots occupied together subtended approximately 8° × 12° of visual angle.

Stimuli were presented on a Color Graphic Monitor (Silicon Graphics GDM-4011P) at 60 Hz and 1024 × 768 pixels resolution using Matlab (Mathworks, Natick, MA) and the Psychophysics Toolbox (Brainard, 1997; Pelli, 1997).

Procedure

We used previously established stimuli and paradigms to test sensitivity to biological motion. Each trial started with a fixation cross, followed by a PLD of biological motion or its scrambled counterpart, presented with a variable number of similarly moving noise dots of the same shape, size, and color (Saygin, in press; Gilaie-Dotan, Bentin, Harel, Rees, & Saygin, 2011; Saygin, Cook, & Blakemore, 2010; Hiris, 2007; Bertenthal & Pinto, 1994). The observers' task was to determine whether a person was present. Feedback was provided via the color of the fixation cross, which turned green (correct) or red (incorrect) for 750 msec before the start of the next trial. On

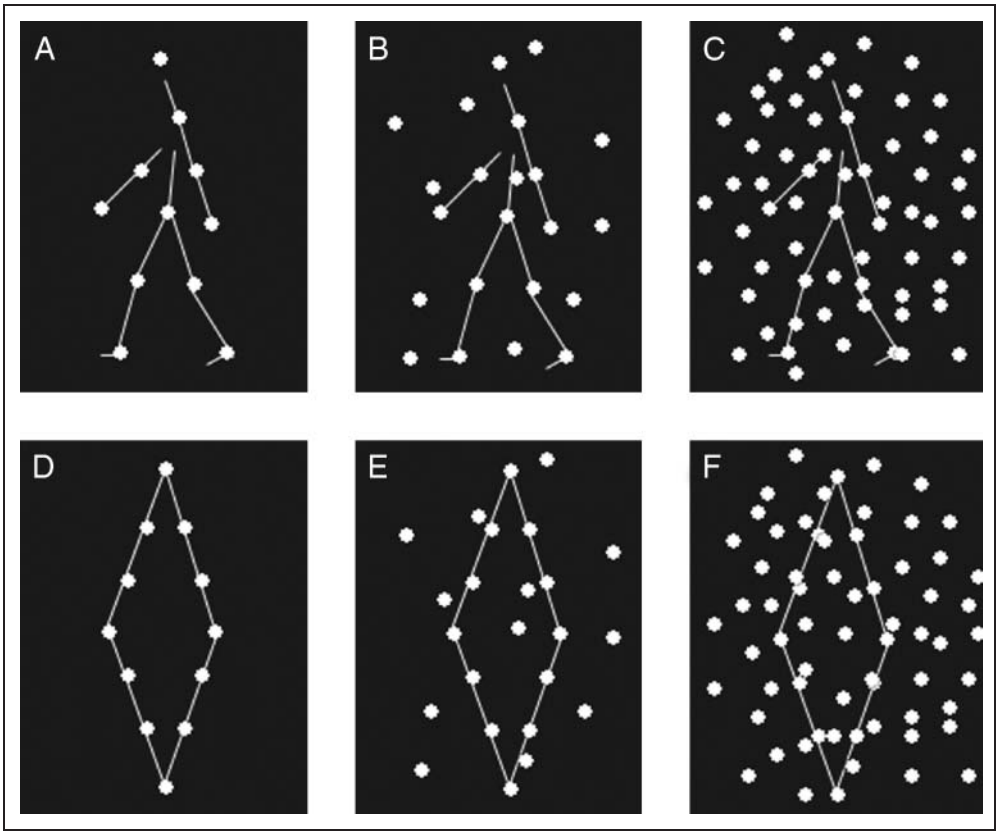

Figure 1. Schematic of the stimuli. Depicted are still images from a biological motion animation (a point-light walker) with no noise (A) and two different levels of noise (B, C) and a nonbiological stimulus from Experiment 2 without (a diamond, D) and with noise (E, F). The connecting lines were added as a visual aid and were not presented in the studies. Noise dots moved in trajectories that were the same as the target animations.

each trial, the position of the PLD was spatially jittered randomly within a 2.2° radius from the center to prevent a response strategy based on purely local motion information. There was a fixation cross before and after the PLD, but fixation was not compulsory, and eye movements were not recorded. Each animation lasted 583 msec (35 frames). Participants responded by pressing one of two adjacent keys on the keyboard. If no response was given within 2 sec, an incorrect response was assumed in the adaptive thresholding algorithm (for the thresholding stage), or the trial was excluded from the signal detection analyses (pre- and post-TMS sessions).

Of course, what is primarily of interest here is the change in behavioral measures after TMS and not raw measurements per se. Even so, we attempted to bring the subjects' performance to a similar range to decrease variability. Before each testing session, we measured individual thresholds and then tested subjects' sensitivity and response bias at those levels because intersubject variability in biological motion perception is high (Gilaie-Dotan, Kanai, Bahrami, Rees, & Saygin, 2011). Furthermore, we measured thresholds in each session because, even within subjects, thresholds can vary from session to session (Saygin, 2007). At the beginning of each session, the observers were shown all the PLDs that were used in the experiment and completed a 12-trial practice block. We then estimated a noise dot threshold individually for each session using a Bayesian adaptive procedure, QUEST. During adaptive thresholding, subjects completed two runs of 68 trials each, and we estimated the number of noise dots at which they were at 75% accuracy using the mean of the posterior probability density function (Gilaie-Dotan, Bentin, et al., 2011; Gilaie-Dotan, Kanai, et al., 2011; Saygin et al., 2010; Watson & Pelli, 1983). The larger of the two thresholds was used as the number of noise dots to be used in the pre- and post-TMS measurements for that session.

After a threshold was estimated for the session, subjects completed three pre-TMS blocks of 60 trials each, administered at the number of noise dots determined by the thresholding procedure. After cTBS was administered and a delay of 5 min, subjects completed three 60-trial post-TMS blocks. Dependent measures from these pre- and post-TMS runs were evaluated statistically.

Off-line TBS was used instead of standard repetitive TMS (rTMS) because TMS over frontal areas such as PMC can induce eye blinks and muscle twitches that can interfere with perceptual processing, complicating the interpretation of results. Theta-burst TMS (Huang, Edwards, Rounis, Bhatia, & Rothwell, 2005) was delivered using a MagStim Rapid2 stimulator (MagStim, Whitland, United Kingdom) and a figure-eight coil (diameter = 70 mm). A train of rTMS pulses, three pulses at 50 Hz delivered every 200 msec, was delivered at 40% of maximum stimulator output over the site being tested in each session. Each session included a 20-sec train of such pulses, which should lead to an effect on the region stimulated for at least 15–20 min, likely longer (Allen, Pasley, Duong, & Freeman, 2007; Huang et al., 2005).

We used subjects' structural MRI scans and Brainsight (Rogue Research, Montreal, Canada) to localize the stimulation sites (Figure 2). Three sites were stimulated on different days, 3–7 days apart: PMC (near the junction of the inferior frontal and precentral sulci, Montreal Neurological Institute coordinates: −38 12 24.5), pSTS (Montreal Neurological Institute coordinates: −49 −62 18), or vertex (halfway between inion and nasion and halfway between the intertragal notches), which served as the control site. The coordinates for PMC and STS were based on previous work (Saygin, 2007). Because the lesion analysis in the latter study was only possible in the left hemisphere, we stimulated these sites in the left hemisphere. Because of individual variability in anatomy, to ascertain that the stimulated site was in the intended locations, we moved the Brainsight probe if needed, by no more than 5 mm, around targeted coordinates. For pSTS, we targeted the sulcus and not the adjacent gyri; for PMC, we targeted the inferior frontal sulcus or slightly posterior to it (and not the middle frontal gyrus).

Control Experiment (Experiment 2)

The results of Experiment 1 indicated that TMS over PMC affected the perception of biological motion. In a control experiment, we investigated whether this effect was specific to biological motion perception or might generalize to other nonbiological stimuli as well.

We generated 11 geometric shapes (four-sided polygons) composed of 12 point-lights of the same size and color as those used in the biological motion animations (Figure 1). In each trial, either a coherent point-light shape (e.g., a rectangle or a diamond) or a scrambled set of dots that did not comprise a shape translated upward or downward, along with translating noise dots (Gilaie-Dotan, Bentin, et al., 2011; Saygin et al., 2010). The task, as in the main experiment, was to determine whether a coherent shape was present. All experimental procedures were identical to the main experiment.

Data Analysis

Descriptive statistics (mean and standard deviation) for the signal detection measures as well as accuracy and RT are reported in Table 1 for both experiments.

The experimental data were analyzed within the signal processing framework. Trials in which no response was recorded were removed from the analyses. The proportion of such trials was low, ranging between 0.08% and 0.6%, but did not significantly vary between conditions. We computed sensitivity (d') and response bias (Green & Swets, 1966), which allowed for comparison with previous work (Grossman et al., 2005). After observing a significant effect of TMS on response bias, we ran post hoc tests using hit and false alarm rates. RTs were recorded and reported in Table 1 along with accuracy but were not focused on because, in TMS experiments, they can be difficult to interpret (Chouinard & Paus, 2010; Terao et al., 1997). Our hypotheses (that TMS would affect biological motion processing for PMC and pSTS but not for control) were tested using paired-samples t tests performed between pre- and post-TMS measurements because the full ANOVA does not represent our null hypothesis. Sphericity assumptions were verified and corrected for if needed. p Values were corrected for multiple comparisons.

RESULTS

Experiment 1

Average sensitivity was 1.49 (SD = 0.27), and average response bias was 0.005 (SD = 0.09). Mean accuracy was 0.76 (SD = 0.037), and mean RT was 0.929 sec (SD = 0.1). Descriptive statistics for pre- and post-TMS sessions are provided in Table 1.

Given large interindividual and intersession variability in biological motion tasks (Saygin, 2007), we adaptively measured thresholds (see Methods) at the beginning of

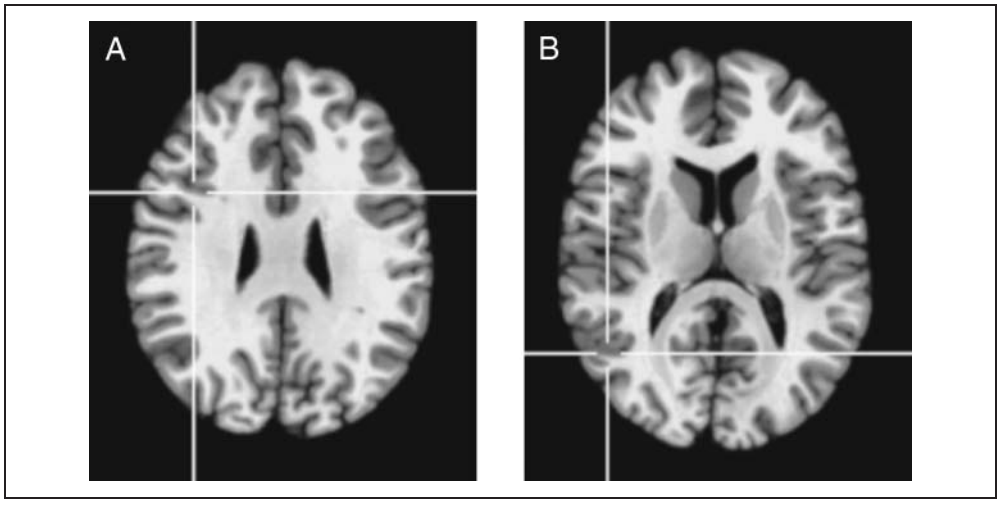

Figure 2. Stimulation sites. PMC (A) and pSTS (B) conditions, shown on axial slices of the Montreal Neurological Institute template brain.

Table 1. Descriptive Statistics for Behavioral Data for All TMS Sites (PMC, pSTS, and Control), Including the Control Experiment (Experiment 2, PMC)

	PMC Pre-TMS	*PMC Post-TMS*	*pSTS Pre-TMS*	*pSTS Post-TMS*	*Control Pre-TMS*	*Control Post-TMS*	*PMC (Exp 2) Pre-TMS*	*PMC (Exp 2) Post-TMS*
Sensitivity (d')	**1.693 (0.52)**	**1.521 (0.65)**	1.442 (0.48)	1.316 (0.51)	1.475 (0.38)	1.540 (0.43)	1.795 (0.63)	1.788 (0.74)
Response bias (criterion)	**0.069 (0.09)**	**−0.130 (0.19)**	0.041 (0.17)	0.004 (0.21)	0.049 (0.19)	0.022 (0.24)	0.271 (0.29)	0.131 (0.26)
Hit rate	0.775 (0.07)	0.799 (0.08)	0.741 (0.09)	0.733 (0.11)	0.748 (0.07)	0.760 (0.07)	0.74 (0.05)	0.76 (0.12)
False alarm rate	**0.191 (0.08)**	**0.280 (0.12)**	0.230 (0.07)	0.260 (0.11)	0.231 (0.09)	0.233 (0.11)	0.153 (0.10)	0.17 (0.11)
Accuracy	**0.791 (0.07)**	**0.760 (0.08)**	0.745 (0.07)	0.727 (0.08)	0.760 (0.06)	0.767 (0.06)	0.793 (0.02)	0.793 (0.08)
RT	0.943 (0.10)	0.908 (0.11)	**0.958 (0.11)**	**0.896 (0.08)**	0.931 (0.13)	0.911 (0.15)	0.710 (0.10)	0.704 (0.13)

The mean values for sensitivity (d'), response bias (criterion), hit rate, false alarm rate, accuracy, and RT (in seconds) for pre- and post-TMS sessions are shown, along with the standard deviations for each data point (in parentheses). The data in **bold** font are those where significant pre-TMS versus post-TMS differences were observed (see Results for inferential statistics). Exp = experiment.

each experimental session (PMC, pSTS, control). This procedure estimates the number of noise dots at which a subject is expected to perform at 75% accuracy. This threshold corresponded to 18.36 noise dots on average (SD = 5.094). In each session, the measured threshold (rounded to the nearest integer) was used to administer the pre- and post-TMS trials. Subjects tended to improve over the three sessions ($p < .05$), indicating that it is important to acquire thresholds in each session (mean threshold for first session: 12.6, SD = 7.51; for second session: 18.54, SD = 6.59; for third session: 25.54, SD = 9.50). Despite our attempts at counterbalancing session order and separate adaptive thresholding for each session, pre-TMS performance still varied between sessions (though the differences were not significant, all ps > .01 uncorrected), highlighting the importance of using individually determined thresholds as was done here.

The results of the experiment are reported in Figure 3, depicting sensitivity (d', A) and response bias (criterion, B) for each condition in pre- and post-TMS. Planned paired-samples t tests revealed that sensitivity decreased significantly after TMS of PMC (t = 2.673, p = .029), nearly significantly for pSTS (t = 1.674, p = .060), but did not change significantly after TMS of vertex (t = −0.758, p = .231). There was a significant decrease in criterion after TMS of PMC (t = 3.917, p = .002) but not after TMS of pSTS or vertex (t = 0.547, p = .581 and t = 0.565, p = .594, respectively).

A lower criterion indicates that participants were more likely to say "yes," which could mean they made more hits, more false alarms, or both. Although TMS did not significantly affect hit rate for any condition, participants made significantly more false alarms after TMS of PMC (t = −3.734, p = .001). False alarm rates were unaffected for the pSTS and vertex conditions (t = −1.2, p = .13 and t = −0.099, p = .45, respectively). The change in false alarms after TMS of PMC corresponded to a mean of 55% increase.

Thus, TMS of PMC affected participants' response bias to biological motion stimuli in a specific way, namely, by increasing the tendency to respond that biological motion was present when it was not. Importantly, this was not a generalized response tendency: No significant increase in false alarms was found in the control experiment featuring the same task with nonbiological object stimuli (Experiment 2).

Although the effects of TMS on RTs tend to be nonspecific and unlikely to be informative about biological motion perception per se, for completeness, we report RT data. RT decreased after TMS for all conditions (main effect: $F(1, 11) = 9.598, p = .010$); the difference reached significance for STS (t = 4.044, p = .002) but not for PMC and control (t = 1.547, p = .14 and t = 1.041, p = .316, respectively). Exploring the relationship between the signal detection measures and changes in RT, we only found a relationship with false alarm rate for PMC (r = .52, p < .05). However, this was not a speed–accuracy trade-off; instead, longer RTs were associated with higher false alarm rates.

Experiment 2

Average sensitivity was 1.79 (SD = 0.23), and average response bias was 0.2 (SD = 0.09). Mean accuracy was 0.79 (SD = 0.025), and mean RT was 0.71 sec (SD = 0.04). Only RT was significantly different from Experiment 1 ($p < .001$), although response bias also approached significance (p = .06). Descriptive statistics (pre- and post-TMS) are provided in Table 1.

None of the TMS effects reported for Experiment 1 approached significance for TMS of PMC for nonbiological structure from motion detection (Table 1; all p values > .1). This shows that the effects of TMS over the PMC found in the main experiment were, at least to some degree, specific to biological motion perception

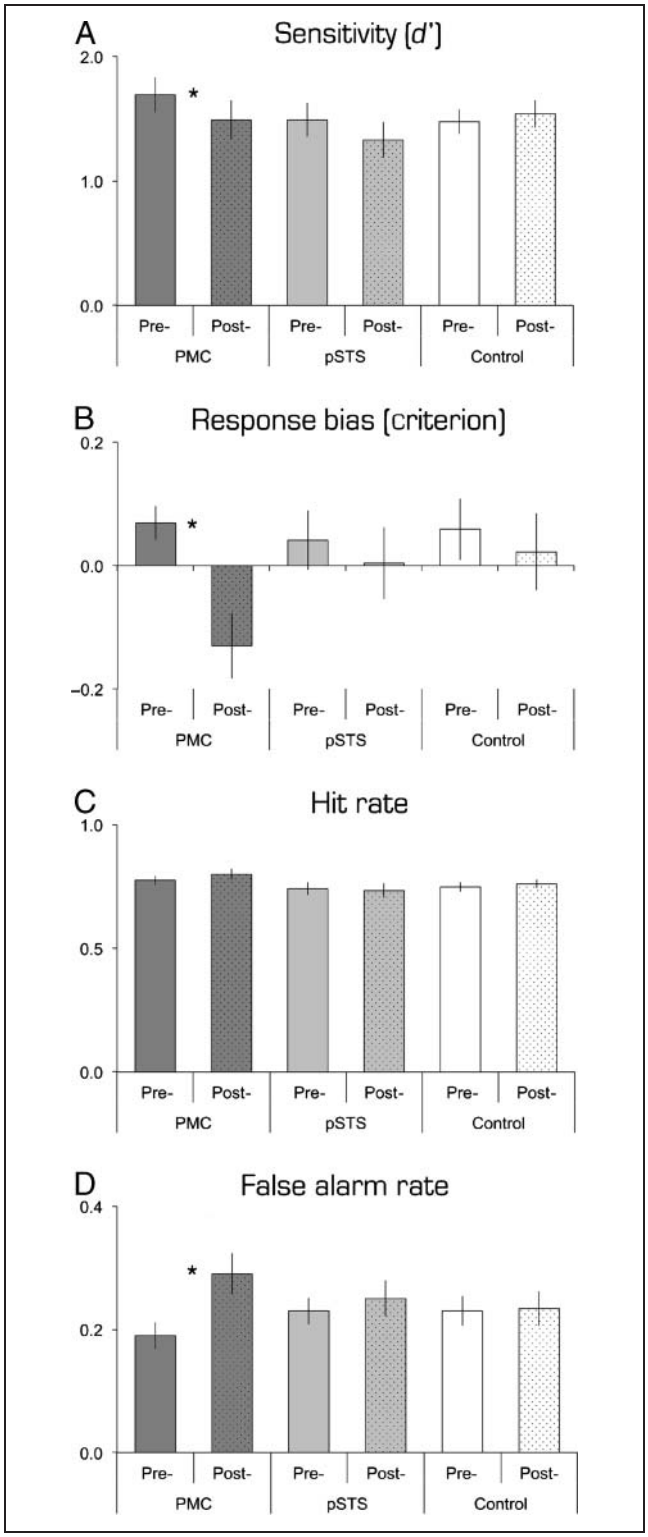

Figure 3. Results of Experiment 1. Sensitivity (A), response bias (B), hit rate (C), and false alarm rate (D) data from pre- and post-TMS sessions are shown. The dark gray bars depict the data for PMC; the medium gray bars, for the pSTS; and the light gray bars, for the control site (vertex). * indicates significant effects (see Results). Error bars are *SEM*. (A) Sensitivity (d') decreased significantly after TMS of PMC and approached significance after TMS of pSTS. (B) Response bias (criterion) significantly decreased after TMS of PMC. (C) Hit rate did not significantly change after TMS of any site. (D) False alarm rates were significantly increased after TMS of PMC.

and not general response patterns for our (detection in noise) task.

DISCUSSION

In many biologically relevant situations, from tracking prey and detecting predators to learning a new skill from others and inferring social norms, organisms must observe their conspecifics and understand their movements and actions. The processing of biological motion signals is critical for achieving these important and ubiquitous tasks (Blake & Shiffrar, 2007; Puce & Perrett, 2003). Neuroimaging and neurophysiological studies have highlighted the pSTS as a key brain area for biological motion perception (Gilaie-Dotan, Kanai, et al., 2011; Wyk et al., 2009; Saygin, Wilson, Hagler, et al., 2004; Grossman et al., 2000; Oram & Perrett, 1996). To support action and biological motion perception, pSTS works within a larger network of regions including the PMC, here referred to as the APS (Saygin, in press; Grafton & Hamilton, 2007; Rizzolatti & Craighero, 2004).

Although the "virtual lesion" depiction of this technique is too simplistic, and the precise physiological effects need further specification, TMS has great potential in cognitive neuroscience by allowing reversible perturbations of processing in selected brain areas in healthy individuals (Miniussi, Ruzzoli, & Walsh, 2010; Silvanto, Muggleton, & Walsh, 2008; Allen et al., 2007). TMS over pSTS has been shown to decrease sensitivity to biological motion (Grossman et al., 2005), and TMS of PMC affects other aspects of action perception (e.g., Chouinard & Paus, 2010; Candidi et al., 2008; Urgesi et al., 2007; Pobric & Hamilton, 2006). The specific role of biological motion had not been tested for PMC. Furthermore, it was unclear what distinct contributions pSTS and PMC might make to computations underlying biological motion processing. To address these gaps in knowledge, we used TMS over both pSTS and PMC, along with well-established stimuli and paradigms from vision science (Blake & Shiffrar, 2007), and explored causal links between the APS and biological motion. Off-line cTBS TMS was used to avoid potential confounds from eye blinks and muscle twitches that can occur with stimulation over some frontal areas.

To summarize, we found that TMS of PMC led to a significant decrease in sensitivity (d') and response bias (criterion) for PLDs of biological motion. Subjects made significantly more false alarms post-TMS of PMC. We also found a marginally significant decrease in sensitivity following TMS of the pSTS. None of these effects were found for TMS of the control site or for the control task.

These findings significantly extend previous work on the effects of TMS on biological motion perception. A reduction in sensitivity to biological motion following rTMS over pSTS was reported previously by Grossman and colleagues (2005). Although their study had targeted the right pSTS, we targeted the left pSTS selecting our

stimulation coordinates from prior work with comparable stimuli and tasks (based on left-hemisphere lesion-behavior maps; Saygin, 2007; Bates et al., 2003). It is possible that TMS effects would have been stronger over the right pSTS, consistent with a right hemisphere dominance for biological motion processing found in some fMRI studies (e.g., Pelphrey et al., 2005; Grossman et al., 2000). On the other hand, although the difference did not reach significance ($p = .06$), TMS of pSTS did reduce sensitivity to biological motion in the left hemisphere. Furthermore, neuroimaging and neuropsychological studies have revealed significant links between the left pSTS and biological motion (Gilaie-Dotan, Kanai, et al., 2011; Saygin, 2007; Saygin, Wilson, Hagler, et al., 2004), indicating that laterality effects in biological motion processing are likely to be relatively subtle and/or dependent on the specifics of the stimuli and task.

A novel finding is that TMS of PMC significantly reduces sensitivity and response bias in biological motion perception. Furthermore, we found that these effects were driven by a specific increase in false alarms post-TMS of PMC. Note that, when biological motion was not present, there was still scrambled motion presented. Following TMS of PMC, participants tended to not reject these stimuli, but instead, they perceived them as biological motion (a person is present). Given the distinct response profiles obtained after TMS of PMC and pSTS, our data show that these regions may make different functional contributions to biological motion perception. We suggest a modulatory role for PMC on biological motion processing. In an alternative way of thinking about these data, TMS of PMC may have affected decision-making criteria regarding action perception, leading to increased false alarm rates (which were associated with longer RTs).

PMC is theorized to be a region in which visual signals are compared with or supplemented by embodied representations of the body (Chouinard & Paus, 2010; Rizzolatti & Craighero, 2004; Giese & Poggio, 2003). If processing in PMC is disrupted, and the match-to-body process is impacted, subjects could exhibit reduced sensitivity for biological motion. It is possible that, in processing these stimuli, the pSTS broadly categorizes movements as biological (Grossman, Jardine, & Pyles, 2011) and works in concert with PMC to further refine the computations, perhaps via a template matching strategy (Lange, Georg, & Lappe, 2006).

Why would TMS lead to only increases in false alarms and not a decrease in hits? Although our discussion is necessarily speculative given the small literature on TMS and biological motion, our interpretations may be partially constrained by the ways in which the effects of TMS can be conceptualized. TMS can be viewed as temporarily disabling neural function, impacting processing of information (signal), with studies suggesting that it can be thought of a reduction in the strength of the perceptual signal (Harris, Clifford, & Miniussi, 2008). Alternatively, TMS could also affect perception by the induction of unrelated neural activity, which effectively increases neural noise in the stimulated area (Ruzzoli, Marzi, & Miniussi, 2010). Both reduction of signal and increased noise could lead to decreased sensitivity following TMS. In terms of the effects of TMS on PMC, we speculate that increased neural noise is more likely than reduced signal strength to explain our findings, given the selective increase in false alarms. Note that, in trials where biological motion was absent, subjects were presented with scrambled versions of the same animations, which contain the same local motion signals but not the coherent body form. It is possible that, when a coherent form is present (i.e., the trial is a hit if correct), the match-to-body is easier to detect, perhaps via body form information also transmitted by pSTS (Thompson, Clarke, Stewart, & Puce, 2005), and there is no effect of TMS of PMC on performance. When the coherent form is absent (i.e., the trial is a false alarm if incorrect), the PMC is still primed by the local biological motion information to perform the match-to-body process, but this computation is disrupted by TMS. No increase in false alarms was found for the nonbiological motion task, indicating that local biological motion information may trigger specific neural computations and/or populations and may even selectively engage the match-to-body process. Studies in which signal and noise are manipulated independently could help test these possibilities (Ruzzoli et al., 2011). Eye tracking can be used to test whether TMS affects how observers scan the noise-masked displays. More generally, future experiments with stimuli that manipulate biological motion and form as well as degree of match to the observers' body can be useful in further specifying the functional properties of the APS (Saygin, Chaminade, Ishiguro, Driver, & Frith, 2011; Calvo-Merino, Grezes, Glaser, Passingham, & Haggard, 2006; Casile & Giese, 2006). Single-pulse TMS, EEG, and magnetoencephalography would be additional methods with which to investigate the dynamics of body motion processing in the APS.

Conclusions

Our study was the first to use cTBS to explore the neural basis of action perception. We studied the effects of TMS on point-light biological motion processing using established psychophysical methods, adaptive thresholding, and signal detection analyses. TMS over PMC led to a decrease in sensitivity and response bias, the latter because of an increase in false alarms post-TMS. These effects were specific to biological motion and did not generalize to the same task performed with nonbiological object motion. Combining these data with other findings in the literature, we suggest that TMS of PMC may have interfered with processing biological motion because access to the body's own motor representations was compromised. It is possible that PMC provides a modulatory influence to help refine the computations of posterior areas during biological motion perception and/or in decision-making regarding biological motion.

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