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Posttraumatic stress disorder and the social brain: Affect-related disruption of the default and mirror networks

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Abstract

Background: Social cognitive impairments, specifically in mentalizing and emotion recognition, are common and debilitating symptoms of posttraumatic stress disorder (PTSD). Despite this, little is known about the neurobiology of these impairments, as there are currently no published neuroimaging investigations of social inference in PTSD.

Methods: Trauma-exposed veterans with and without PTSD (n = 20 each) performed the Why/How social inference task during functional magnetic resonance imaging (fMRI). Patients with PTSD had two fMRI sessions, between which they underwent affect labeling training. We probed the primary networks of the "social brain"—the default mode network (DMN) and mirror neuron system (MNS)—by examining neural activity evoked by mentalizing and action identification prompts, which were paired with emotional and nonemotional targets.

Results: Hyperactivation to emotional stimuli differentiated PTSD patients from controls, correlated with symptom severity, and predicted training outcomes. Critically, these effects were nonsignificant or marginal for nonemotional stimuli. Results were generally consistent throughout DMN and MNS. Unexpectedly, effects were nonsignificant in core affect regions, but robust in regions that overlap with the dorsal attention, ventral attention, and frontoparietal control networks.

Conclusions: The array of social cognitive processes subserved by DMN and MNS appear to be inordinately selective for emotional stimuli in PTSD. However, core affective processes do not appear to be the primary instigators of such selectivity. Instead, we propose that affective attentional biases may instigate widespread affect-selectivity throughout the social brain. Affect labeling training may inhibit such biases. These accounts align with numerous reports of affect-biased attentional processes in PTSD.

KEYWORDS
default mode network, emotion recognition, mentalizing, mirror neuron, PTSD, social cognition, theory of mind

1 | INTRODUCTION

Posttraumatic stress disorder (PTSD) is characterized by intrusive trauma-related cognition (e.g., thoughts, dreams, and flashbacks), exaggerated affective responses (e.g., chronic fear, anxiety, and hyperarousal), and—conversely—affective blunting (e.g., anhedonia and emotional numbing; American Psychiatric Association, 2013). Though PTSD is commonly associated with affective dysfunction, social cognitive impairments are ubiquitous and often debilitating in PTSD (Brewin, Andrews, & Valentine, 2000; Nietlisbach & Maercker, 2009; Sharp, Fonagy, & Allen, 2012). A strong body of behavioral evidence links PTSD with deficits in emotion recognition (Fonzo et al., 2010; Knežević & Jovančević, 2004;

Mazza et al., 2012; Poljac, Montagne, & de Haan, 2011; Schmidt & Zachariae, 2009; Shin et al., 2005) and mentalizing (Allen & Fonagy, 2006; Mazza et al., 2012; Nazarov et al., 2014; Nietlisbach, Maercker, Rösler, & Haker, 2010; Parlar et al., 2014). This pattern of social cognitive impairment is distinct from other anxiety disorders (Plana, Lavoie, Battaglia, & Achim, 2014). Here, emotion recognition refers to perceiving and identifying others' emotions, while mentalizing refers to reasoning about others' mental states and traits (e.g., beliefs, desires, and intentions). Emotion recognition and mentalizing are both considered to be facets of social inference and theory of mind (Mitchell & Phillips, 2015; Schaafsma, Pfaff, Spunt, & Adolphs, 2015). Taken together, there is converging behavioral evidence that social inference impairments are common and debilitating symptoms of PTSD. However, little is known about the neural underpinnings of these impairments—such knowledge would add a social dimension to our existing understanding of PTSD's neurocognitive mechanisms, perhaps inspiring novel avenues in the diagnosis and treatment of PTSD.

In healthy populations, neuroimaging investigations have revealed that social cognition is primarily subserved by two dissociable large-scale neural networks—the mirror neuron system (MNS) and default mode network (DMN)—which are associated with action identification and mentalizing, respectively (Spunt, Falk, & Lieberman, 2010; Spunt, Kemmerer, & Adolphs, 2016; Spunt, Satpute, & Lieberman, 2011). During social inference, MNS may represent observable sensorimotor actions (e.g., grasping for food) that are used by DMN to infer unobservable mental states (e.g., hunger; Waytz & Mitchell, 2011). Mirror neurons, first discovered in macaque frontoparietal cortex, fire when specific actions are performed and observed (di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992). In humans, similar sensorimotor "mirroring" responses may occur in posterior inferior frontal gyrus (pIFG), dorsal premotor cortex (dPMC), intraparietal sulcus (IPS), and lateral occipitotemporal cortex (LOTC; Oosterhof, Tipper, & Downing, 2013). These regions collectively referred to as MNS, appear to encode facial expressions (Buccino et al., 2001), body language (de Gelder et al., 2010), and other biological actions (Rizzolatti & Sinigaglia, 2010). DMN is strongly implicated in abstract mental state reasoning, such as mentalizing/theory of mind (Frith & Frith, 2006), emotion recognition (Spunt & Lieberman, 2012a), empathy (Zaki & Ochsner, 2012), morality (Reniers et al., 2012), and introspection (Davey, Pujol, & Harrison, 2016). These functions consistently recruit the core DMN hubs of the medial prefrontal cortex (mPFC), posterior cingulate cortex (PCC), and temporoparietal junction (TPJ; Amft et al., 2015). Outside of social cognition, DMN is broadly associated with spontaneous and internally-oriented cognition (Andrews-Hanna, 2012). However, most DMN activity occurs during rest, as DMN activation and connectivity are quickly engaged during the absence of goal-directed cognition (Fox, Foster, Kucyi, Daitch, & Parvizi, 2018).

Unlike in healthy populations, there are currently no published neuroimaging investigations of social inference in PTSD. Thus, little is known about the neurobiology of PTSD-related social inference impairments. However, PTSD-related alterations in DMN activity have been found in other social tasks (Lanius, Frewen, Nazarov, & McKinnon, 2014), such as script-driven social-emotional imagery (Frewen et al., 2010, 2012, 2008), self-reference (Bluhm et al., 2012; Frewen et al., 2011), self-other reference (Frewen, Thornley, Rabellino, & Lanius, 2017), and face perception (Cisler, Scott Steele, Smitherman, Lenow, & Kilts, 2013; Rabellino et al., 2015). Moreover, PTSD-related effects on DMN connectivity are consistently reported in resting-state studies (DiGangi et al., 2016; Sripada et al., 2012; Tursich et al., 2015; Zhang et al., 2015). We are unaware of any reports of PTSD-related effects in regions explicitly defined as MNS. However, MNS appears to overlap substantially with the dorsal attention (DAN), ventral attention (VAN), and frontoparietal control networks (FPCN; Barrett & Satpute, 2013), which are strongly implicated in PTSD-related attentional biases (reviewed in Block & Liberzon, 2016).

We conducted the first neuroimaging investigation of social inference in PTSD to help uncover the neurobiology of PTSD-related social cognitive impairments. To this end, we used functional magnetic resonance imaging (fMRI) to record brain activity during the Why/How social inference task (Spunt & Adolphs, 2014) in trauma-exposed veterans with and without PTSD. The Why/How task contains mentalizing (*Why*) and action identification (*How*) prompts (Figure 1) that dissociate DMN and MNS activity (Figure 2). We explored whether DMN and MNS responses could differentiate patients with PTSD from controls, correlate with symptom severity, and predict outcomes from affect labeling training (Burklund et al., Under review).

2 | MATERIALS AND METHODS

2.1 | Participants and procedure

Forty trauma-exposed military veterans were recruited from the Los Angeles area. All participants were exposed to combat trauma, mostly in Iraq and Afghanistan. Twenty participants met DSM-5 criteria for PTSD or other trauma-related disorder, while twenty healthy controls had no current or lifetime psychiatric diagnoses. Diagnostic status was determined by the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995), which was administered by certified research staff. Participants were 18–45 years old, English-speaking, right-handed, and were excluded for serious medical conditions, moderate-to-severe substance abuse, recent changes to medication/psychotherapy, chronic childhood abuse/neglect, and standard fMRI contraindications. Participants provided informed consent, and the study was approved by the University of California, Los Angeles (UCLA) institutional review board.

All participants performed baseline pretraining assessments involving a clinical interview, questionnaires, and an fMRI scan. Only the PTSD group continued with 3 weeks of twice-weekly affect labeling training, followed by posttraining assessments similar to the pretraining assessments. Affect labeling training involved repeated practice with several computer-based tasks that were designed to strengthen inhibitory capacity (Lieberman et al., 2007; Torre & Lieberman, 2018). This training regimen was investigated as a

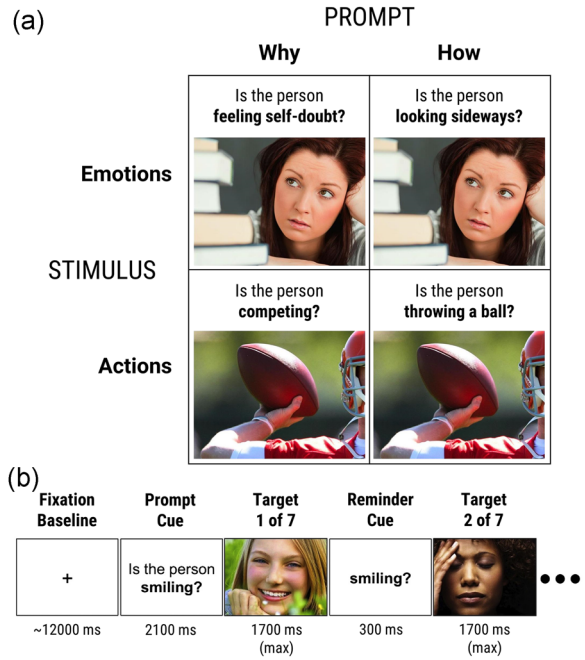

FIGURE 1 Summary of the standardized Why/How social inference task used to evoke activity in the social brain (study 3 of Spunt & Adolphs, 2014). (a) Diagram of the task's 2 × 2 design across Prompt and Stimulus. Each stimulus was shown twice once with a mentalizing prompt (Why) and once with an action identification prompt (How). There were two stimulus types of emotional facial expressions (Emotions) and intentional hand actions (Actions). Thus, there were four conditions WhyEmotions, WhyActions, HowEmotions, and HowActions. (b) The sequence of events in a task block. Each block began with a prompt followed by seven target stimuli paired with that prompt. During the presentation of target stimuli, participants were instructed to judge whether the prompt was true or false for the target as quickly and accurately as possible. Target stimuli were presented for 1,700 ms or until a response was made. A reminder prompt was shown for 300 ms between target stimuli, and each block was preceded by a fixation baseline period. There were 16 blocks in total, 4 per condition, presented in random order. ms, millisecond

proof-of-concept for a novel, brief computerized intervention for PTSD; full methods and data for affect labeling training will be presented in a separate manuscript (Burklund et al., Under review).

In the baseline session, only data from 18 PTSD and 17 control participants were used due to lack of task data (n = 3), a previous brain tumor (n = 1), and noncompliance with fMRI instructions (n = 1). Due to participant dropout, only 13 PTSD participants completed the posttraining session. Posttraining fMRI data from only 11 PTSD participants were used due to lack of task data (n = 1), a previous brain tumor (n = 1), and suspected cannabis intoxication (n = 1).

2.2 | Why/How social inference task

Participants completed the "fast" version of the standardized Why/How task (http://www.bobspunt.com/whyhowlocalizer), which corresponds to study 3 in Spunt and Adolphs (2014). The task features a 2 × 2 design across $\text{Prompt}_{[\text{Why, How}]}$ and $\text{Stimulus}_{[\text{Emotions, Actions}]}$,

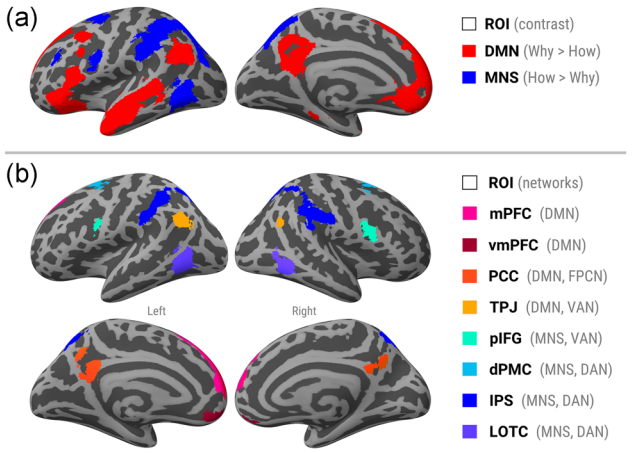

FIGURE 2 *A priori* bilateral ROI masks defined by the Why/How contrast in an independent dataset (Spunt & Adolphs, 2014). All ROI masks are bilateral. The Why/How contrast dissociates DMN and MNS regions that are recruited during mentalizing and action identification, respectively. (a) Whole-network DMN and MNS masks. (b) Within-network ROIs that are thought to be key nodes of the DMN or MNS. Some within network ROIs are also considered to be nodes of the attentional networks. DAN, dorsal attention network; DMN, default mode network; dPMC, dorsal premotor cortex; FPCN, frontoparietal control network; IPS, intraparietal sulcus; LOTC, lateral occipitotemporal cortex; MNS, mirror neuron system; mPFC, medial prefrontal cortex; PCC, posterior cingulate cortex; pIFG, posterior inferior frontal gyrus; ROI, region of interest; TPJ, temporoparietal junction; VAN, ventral attention network; vmPFC, ventromedial prefrontal cortex

making four conditions: *WhyEmotions*, *WhyActions*, *HowEmotions*, and *HowActions*. The task was implemented in Psychophysics Toolbox 3 (Kleiner et al., 2007) on MATLAB 2007a (Mathworks, 2007) displayed via virtual reality goggles at 800 × 600 resolution (Resonance Technology, 2012), with responses recorded by a hand-held button box (Figure 1).

2.3 | fMRI acquisition and preprocessing

fMRI data were acquired at UCLA Staglin Center for Cognitive Neuroscience using a Siemens TIM Trio (3 Tesla) MRI scanner. fMRI data were preprocessed via SPM12 (Friston, 2007) and the DARTEL pipeline (Ashburner, 2007). See Supporting Information Materials for further details.

2.4 | Single-subject/session fMRI analyses

To estimate neural responses to the Why/How task within each participant and session, task timings were specified in SPM12's general linear model and convolved with the canonical double-gamma hemodynamic response function. Realignment parameters were used as covariates to account for remaining motion artifacts. Data were high-pass filtered at 1/128 Hz to correct for signal drift. Parameter estimates from Why/How contrasts for both stimulus

types (*WhyEmotions/HowEmotions*, *WhyActions/HowActions*) were used for subsequent group-level fMRI analyses.

2.5 | Group-level analyses

Unless otherwise noted, group-level statistical analyses were performed via Matlab 2016b Statistics and Machine Learning Toolbox (Mathworks, 2016), with linear mixed-effects models (LMEMs) used for hypothesis testing. LMEMs were specified with the maximal random (within Subject) effects structure justified by each analysis, which is thought to be ideal for hypothesis testing (Barr, 2013; Barr, Levy, Scheepers, & Tily, 2013). Post hoc simple effects tests were performed for LMEMs with significant interaction effects. To obtain canonical "main effects," effects coding was used in multifactor models with at least one categorical factor, otherwise, dummy coding was used (Hardy, 1993).

2.5.1 | Behavioral analyses

In the Why/How task, pretraining group differences in response time and accuracy were analyzed through separate LMEMs. Both LMEMs featured a full-factorial design between $\text{Group}_{[\text{PTSD, Control}]}$, $\text{Prompt}_{[\text{Why, How}]}$, and $\text{Stimulus}_{[\text{Emotions, Actions}]}$. The intercept, Prompt, Stimulus, and Prompt × Stimulus were nested within Subject.

2.5.2 | Region of interest fMRI analyses

To interrogate brain regions that subserve social inference, masks of a priori region of interests (ROIs; Figure 2) were functionally defined by the Why/How contrast in an independent dataset featuring healthy participants ($N = 50$; studies 1 and 3 in Spunt & Adolphs, 2014). A two-tailed t test was used to define DMN (*Why* > *How*) and MNS (*How* > *Why*) masks. Whole-network DMN and MNS masks were defined with a threshold of $p < .001$. A more stringent threshold of $p < 1 \times 10^{-6}$ was used to define within-network ROIs that are considered to be key nodes of DMN and MNS (Andrews-Hanna, Reidler, Sepulcre, Poulin, & Buckner, 2010; Molenberghs, Cunnington, & Mattingley, 2012).

The masks obtained above were used to extract ROI parameter estimates (mean of all voxels within a mask) from single-subject/session Why/How contrasts in our sample. Multiple comparisons across ROIs were accounted for by controlling the false discovery rate (FDR) under 0.05, and p values were adjusted (p_{FDR}) accordingly (Yekutieli & Benjamini, 1999). Pretraining group differences in neural response were analyzed in LMEMs with Group, Stimulus, and their interaction as effects; the intercept and Stimulus were nested within Subject. In patients with PTSD, the relationship between symptom severity (CAPS) and neural responses were examined in LMEMs with CAPS and CAPS × Session × Stimulus as effects; the intercept, Session, Stimulus, and Session × Stimulus were nested within Subject. In patients with PTSD who completed affect labeling training, the relationship between training outcomes (Post-pre CAPS score difference; $\text{CAPS}_{\text{diff}}$) and neural responses was analyzed in LMEMs with $\text{CAPS}_{\text{diff}}$ and $\text{CAPS}_{\text{diff}}$ × Stimulus as effects; the intercept and Stimulus were nested within Subject.

2.5.3 | Whole-brain fMRI analyses

Whole-brain fMRI analyses were performed to complement the primary ROI analyses. Pretraining group differences were examined using Aaron Schultz's MR Tools (Shultz, 2018). We specified a general linear model with Group, Stimulus, and their interaction as effects, with the intercept and Stimulus, nested within Subject. Residuals were used in AFNI's 3dFWHMx and 3dClustSim to estimate a cluster extent (k) that controls familywise error rate (FWER) under 0.05 (Cox, Chen, Glen, Reynolds, & Taylor, 2017). Post hoc Group simple effects tests were conducted in clusters with significant interaction effects ($p < .005$, $k > 120$ voxels).

3 | RESULTS

3.1 | Behavioral results

The PTSD group exhibited greater symptom severity (CAPS) than controls, and symptom severity was reduced after affect labeling training. Full clinical results will be presented in a separate manuscript (Burklund et al., Under review). Unexpectedly, pretraining Why/How task performance did not differ significantly between PTSD and controls. For both response time and accuracy, the main effect of Group and all Group-related interaction effects were nonsignificant (see Table S1).

3.2 | Pretraining neural responses in the PTSD and control groups

Overall, the Why/How contrasts produced activations in DMN ROIs (Figure 3a), deactivations in MNS ROIs (Figure 3b), aligning with previous studies (Spunt & Adolphs, 2014). Unexpectedly, the main effects of Group and Stimulus were nonsignificant in all ROIs. Instead, Group × Stimulus was significant in 4/5 DMN ROIs and 4/5 MNS ROIs. Within these ROIs, post hoc tests revealed that significant Group differences were elicited only by emotional facial expressions (Emotions), not intentional hand actions (Actions). In DMN ROIs, Emotions-evoked activations in the PTSD group, while controls showed negligible responses. In MNS ROIs, Emotions evoked negligible responses in the PTSD group, while controls showed deactivations. These results portray higher Emotions-evoked BOLD activation in the PTSD group relative to controls (Figure 3 and Table 1).

Mirroring the ROI results, the whole-brain analysis (Figure 3c) found nonsignificant Group and Stimulus main effects ($p < .005$, clusterwise FWER < 0.05). Instead, Group × Stimulus was significant in three clusters: left dorsolateral prefrontal cortex (dlPFC; peak = [−33, 28, 40], $F_{1,33} = 18.96$, $k = 503$), bilateral dorsal somatomotor cortices (dSMC; peak = [−6, −28, 60], $F_{1,33} = 16.73$, $k = 811$), and left TPJ (peak = [−51, −51, 39], $F_{1,33} = 16.40$, $k = 476$). In these clusters, post hoc tests revealed that only Emotions elicited significant Group

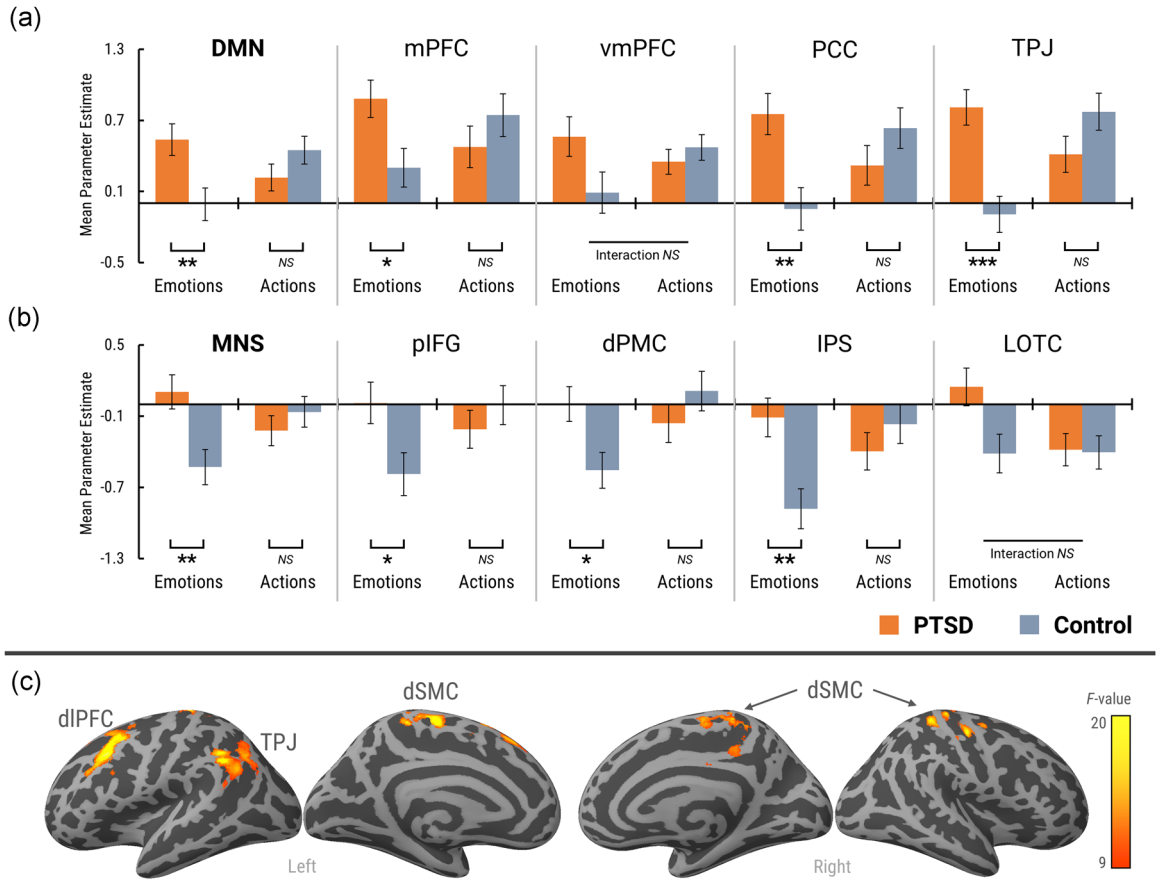

FIGURE 3 Pretraining Why/How neural responses across $Group_{[PTSD,\ Control]}$ and $Stimulus_{[Emotions,\ Actions]}$. These main effects were nonsignificant, instead, the Group × Stimulus interaction was robust. (a, b) Mean parameter estimates in all (a) DMN and (b) MNS ROIs. Error bars represent standard error of the mean. U-shaped brackets indicate the significance of Group simple effects. (c) Whole-brain analysis of Group × Stimulus ($p < .005$, clusterwise FWER < 0.05). dlPFC, dorsolateral prefrontal cortex; DMN, default mode network; dPMC, dorsal premotor cortex; dSMC, dorsal somatomotor cortices; FDR, false discovery rate; FWER, familywise error rate; IPS, intraparietal sulcus; LOTC, lateral occipitotemporal cortex; MNS, mirror neuron system; mPFC, medial prefrontal cortex; pIFG, posterior inferior frontal gyrus; PCC, posterior cingulate cortex; PTSD, posttraumatic stress disorder; ROI, region of interest; TPJ, temporoparietal junction; vmPFC, ventromedial prefrontal cortex. $^{*}p_{FDR} < .05$; $^{**}p_{FDR} < .01$; $^{***}p_{FDR} < .001$; NS $p_{FDR} > .05$

differences, specifically by evoking greater activation in PTSD relative to controls in left dlPFC (Emotions: $t_{33} = 3.60$, $p < .001$; Actions: $t_{33} = -1.89$, $p = .07$), dSMC (Emotions: $t_{33} = 3.35$, $p = .002$; Actions: $t_{33} = -1.94$, $p = .06$), and left TPJ (Emotions: $t_{33} = 4.04$, $p < .001$; Actions: $t_{33} = -1.98$, $p = .06$).

3.3 | Symptom severity and neural responses

In patients with PTSD, the main effect of CAPS on Why/How neural responses were nonsignificant in all ROIs. Instead, CAPS × Session × Stimulus was significant in 4/5 DMN ROIs and 4/5 MNS ROI. Within these ROIs, post hoc CAPS simple effects tests revealed that directionality was flipped across Stimuli and Session. For Emotions, the CAPS correlation was positive during pretraining (significant in PCC and IPS), and negative during posttraining (significant in pIFG). For Actions, CAPS was negative during pretraining (nonsignificant), and positive during posttraining (significant in DMN, MNS, and IPS; Figure 4 and Table 2).

3.4 | Predicting training outcomes from pretraining neural responses

In patients with PTSD who completed affect labeling training, the main effect of $CAPS_{diff}$ (Post-pre CAPS score difference) on pretraining Why/How neural responses was nonsignificant in all ROIs. Instead, $CAPS_{diff}$ × Stimulus was significant in 3/5 DMN ROIs and 5/5 MNS ROIs. Within these ROIs, post hoc CAPSdiff simple effects tests revealed that the $CAPS_{diff}$ correlation was negative for Emotions (significant in DMN, PCC, TPJ, MNS, IPS, and LOTC) and slightly positive for Actions (nonsignificant). In sum, greater pretraining reactivity to emotional stimuli predicted better training outcomes (Figure 5 and Table 3).

4 | DISCUSSION

We conducted the first neuroimaging investigation of social inference in PTSD to help uncover the etiology of PTSD-related social cognitive impairments. To this end, we examined neural

TABLE 1 Pretraining neural responses in the PTSD and control groups

	DMN ROIs					MNS ROIs				
Effect	**ROI**	***b***	***SE***	***t***	p_{FDR}	**ROI**	***b***	***SE***	***t***	p_{FDR}
$Group_{PTSD\text{-}Control}$	DMN	.079	.052	1.505	.238	MNS	.118	.062	1.897	.155
$Stimulus_{Emotions\text{-}Actions}$		−.034	.072	−0.476	.716		−.035	.074	−0.464	.716
Group × Stimulus		**.194**	.072	2.715	.014		**.198**	.074	2.656	.014
Group (Emotions)[a]		**.546**	.191	2.861	.011		**.633**	.207	3.054	.009
Group (Actions)[a]		−.232	.162	−1.426	.387		−.159	.181	−0.878	.387
$Group_{PTSD\text{-}Control}$	mPFC	.079	.098	0.808	.422	pIFG	.099	.076	1.297	.249
$Stimulus_{Emotions\text{-}Actions}$		−.010	.069	−0.138	.891		−.090	.093	−0.970	.671
Group × Stimulus		**.213**	.069	3.073	.010		**.200**	.093	2.162	.043
Group (Emotions)[a]		**.583**	.227	2.571	.017		**.599**	.251	2.383	.023
Group (Actions)[a]		−.268	.251	−1.065	.387		−.203	.228	−0.888	.387
$Group_{PTSD\text{-}Control}$	vmPFC	.088	.063	1.400	.238	dPMC	.071	.083	0.862	.422
$Stimulus_{Emotions\text{-}Actions}$		−.042	.078	−0.541	.716		−.126	.074	−1.716	.602
Group × Stimulus		.149	.078	1.899	.069		**.207**	.074	2.817	.014
Group (Emotions)[a]		Interaction NS					**.557**	.210	2.654	.016
Group (Actions)[a]		Interaction NS					−.272	.233	−1.168	.387
$Group_{PTSD\text{-}Control}$	PCC	.122	.087	1.407	.238	IPS	.135	.070	1.919	.155
$Stimulus_{Emotions\text{-}Actions}$		−.062	.087	−0.719	.716		−.107	.091	−1.183	.602
Group × Stimulus		**.279**	.087	3.221	.010		**.249**	.091	2.750	.014
Group (Emotions)[a]		**.801**	.249	3.211	.008		**.769**	.233	3.296	.008
Group (Actions)[a]		−.314	.240	−1.308	.387		−.228	.226	−1.010	.387
$Group_{PTSD\text{-}Control}$	TPJ	.136	.059	2.302	.148	LOTC	.146	.066	2.223	.148
$Stimulus_{Emotions\text{-}Actions}$		−.118	.090	−1.301	.602		.130	.083	1.554	.602
Group × Stimulus		**.315**	.090	3.488	.009		.134	.083	1.613	.111
Group (Emotions)[a]		**.902**	.213	4.238	.001		Interaction NS			
Group (Actions)[a]		−.359	.219	−1.638	.387		Interaction NS			

Note: Pretraining Why/How neural responses were analyzed in LMEMs with Group, Stimulus, and Group × Stimulus as effects; the intercept and Stimulus were nested within Subject. Post hoc Group simple effects tests were conducted for ROIs with significant Group × Stimulus interactions ($p_{FDR} < .05$). Bolded coefficients have corresponding P_{FDR} values < .05 (the exact significance values are shown in the P_{FDR} column in each row).

Abbreviations: *b*, regression coefficient; DMN, default mode network; dPMC, dorsal premotor cortex; IPS, intraparietal sulcus; LMEM, linear mixed-effects model; LOTC, lateral occipitotemporal cortex; MNS, mirror neuron system; mPFC, medial prefrontal cortex; NS, not significant ($p_{FDR} > .05$); PCC, posterior cingulate cortex; p_{FDR}, *p* value adjusted for false discovery rate (<.05); pIFG, posterior inferior frontal gyrus; PTSD, posttraumatic stress disorder; ROI, region of interest; *SE*, standard error of regression coefficient; TPJ, temporoparietal junction; vmPFC, ventromedial prefrontal cortex.

[a]Post hoc simple effect test.

activation evoked by the Why/How social inference task, which dissociates the primary networks of the social brain: the DMN and MNS (Spunt & Adolphs, 2014). We found that DMN and MNS responses differentiated patients with PTSD from controls, correlated with symptom severity, and predicted affect labeling training outcomes. Unexpectedly, these effects were driven primarily by hyperactivation to emotional stimuli. Our neuroimaging results were not corroborated by behavioral results from the Why/How task, despite numerous reports of impaired social inference performance in PTSD (Plana et al., 2014). This discrepancy may be attributable to the ease of the task. Taken together, these results suggest that the social brain may be inordinately selective for affective stimuli in PTSD, even in the absence of measurable behavioral impairments.

4.1 | Affect-related disruption of social cognitive processing in PTSD

In all three analyses, emotional expressions (Emotions) elicited robust PTSD-related effects, while intentional actions (Actions) elicited nonsignificant or marginal effects. Strikingly, the directionality of PTSD-related effects was flipped across Emotions and Actions, suggesting marked differences in the social cognitive processing of emotional and nonemotional stimuli in PTSD. These results were generally consistent throughout DMN and MNS, which is remarkable given the functional heterogeneity between and within these networks (Barrett & Satpute, 2013). Thus, the wide array of social cognitive processes subserved by DMN and MNS appear to be broadly selective for affective stimuli in PTSD. This aligns with numerous reports of affect-selective hyperactivation during other tasks in PTSD (Fani et al., 2012; Khanna et al., 2017; MacNamara, Post, Kennedy, Rabinak, & Phan, 2013; Pannu Hayes, Labar, Petty, McCarthy, & Morey, 2009; Rabellino et al., 2015; Thomaes et al., 2013).

Given the overarching role of affect in our results, it would be reasonable to expect the strongest effects in the ventromedial prefrontal cortex (vmPFC; Figure 2b), the hub of affective processing in the social brain (Lieberman, Straccia, Meyer, Du, & Tan, 2019). vmPFC has been shown to compute the affective properties of social and non-social stimuli (Chikazoe, Lee, Kriegeskorte, & Anderson, 2014; Grabenhorst & Rolls, 2011;

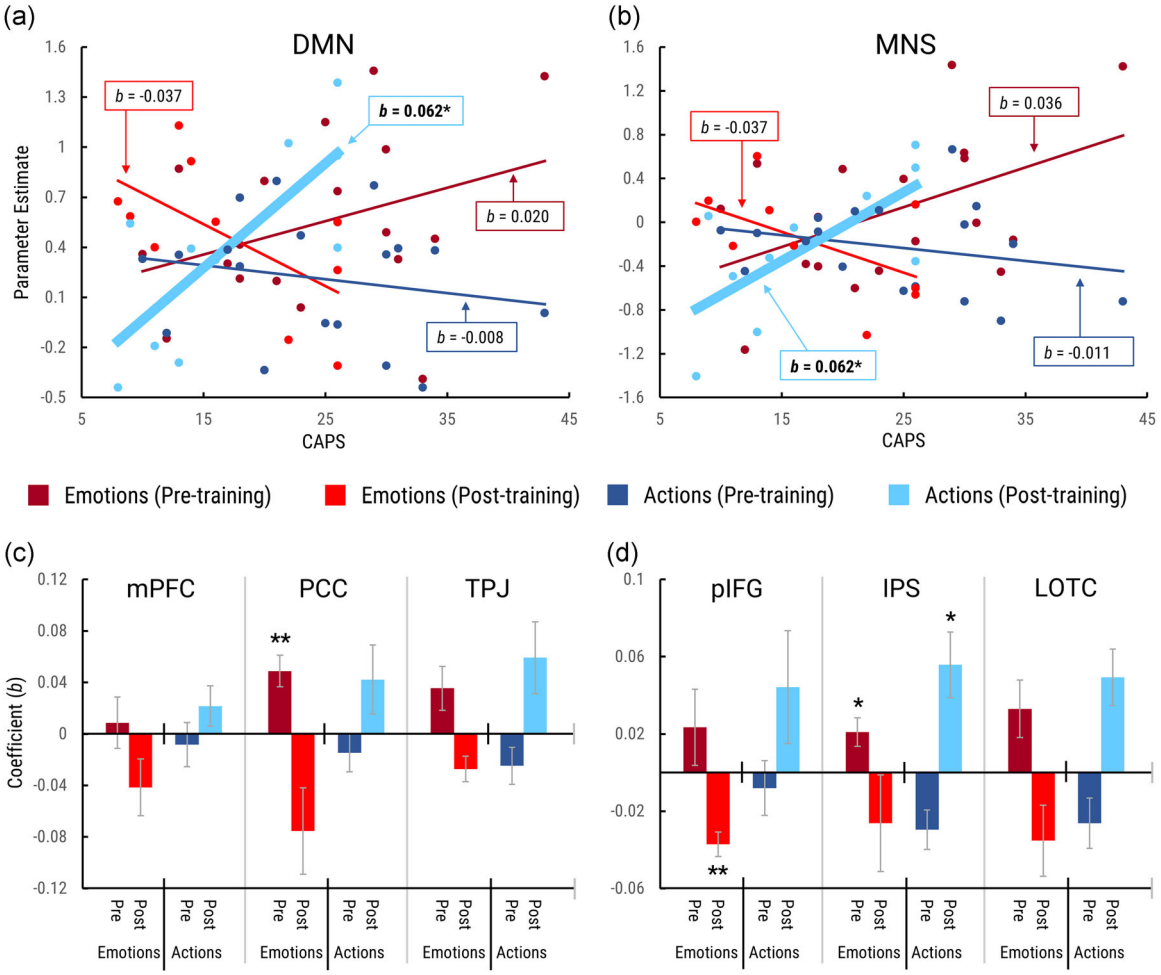

FIGURE 4 Relationship between symptom severity (CAPS score) and Why/How neural responses in patients with PTSD. Only ROIs with a significant CAPS × Session × Stimulus interaction are shown ($p_{FDR} < .05$). (a, b) Scatterplots of parameter estimates and CAPS scores in the (a) DMN and (b) MNS whole-network masks, with regression lines for CAPS simple effects. Thick lines represent significant regression coefficients, while thin lines represent nonsignificant regression coefficients. (c, d) Bar graphs of CAPS simple effects regression coefficients for ROIs within (c) DMN and (d) MNS. Error bars represent standard error of the regression coefficients. *b*, regression coefficient; CAPS, Clinician-Administered PTSD Scale; DMN, default mode network; FDR, false discovery rate; IPS, intraparietal sulcus; LOTC, lateral occipitotemporal cortex; MNS, mirror neuron system; mPFC, medial prefrontal cortex; PCC, posterior cingulate cortex; pIFG, posterior inferior frontal gyrus; PTSD, posttraumatic stress disorder; ROI, region of interest; TPJ, temporoparietal junction. $^{*}p_{FDR} < .05$; $^{**}p_{FDR} < .01$

Winecoff et al., 2013). During social inference, vmPFC can represent the emotions of others (Koster-Hale et al., 2017; Spunt & Lieberman, 2012a; Tamir, Thornton, Contreras, & Mitchell, 2016). Unexpectedly, we did not find significant PTSD-related effects in vmPFC, though it produced the same pattern of responses as other DMN ROIs. Moreover, whole-brain analyses failed to reveal significant PTSD-related effects in other core affective regions such as the amygdala (Phelps & LeDoux, 2005), orbitofrontal cortex (Rolls & Grabenhorst, 2008), and insula (Singer, Critchley, & Preuschoff, 2009). Though these null results may be attributable to a lack of statistical power given our small sample size, it is still apparent that stronger effects were found outside core affect regions, as all non-core affective ROIs produced significant PTSD-related effects. Thus, core affective processes may play a lesser role in disrupting social inference in PTSD.

4.2 | An attentional account of social cognitive dysfunction in PTSD

Outside of core affective processes, what neurocognitive mechanisms could instigate such broad affect-selectivity throughout the social brain? Putatively, attention may be one such mechanism, as attentional processes are often inordinately biased towards emotional stimuli in PTSD (Bardeen & Orcutt, 2011; Dalgleish et al., 2003; Iacoviello et al., 2014; Litz et al., 1996; MacLeod, Mathews, & Tata, 1986; Vythilingam et al., 2007). Concordantly, PTSD-related attentional biases have been linked with affect-evoked hyperactivation in DMN (Block & Liberzon, 2016; Morey, Petty, Cooper, LaBar, & McCarthy, 2008; Pannu Hayes et al., 2009). In general, DMN activation appears to correlate with attention during social tasks (Meyer, Spunt, Berkman, Taylor, & Lieberman, 2012; Spunt & Lieberman,

TABLE 2 Symptom severity and neural responses

	DMN ROIs					MNS ROIs				
Effect	**ROI**	***b***	***SE***	***t***	p_{FDR}	**ROI**	***b***	***SE***	***t***	p_{FDR}
CAPS	DMN	.000	.004	−0.099	.986	MNS	.001	.005	0.181	.986
CAPS × Session × Stimulus		**−.011**	.002	−5.683	<.001		**−.007**	.003	−2.823	.010
CAPS (Pre, Emotions)[a]		.020	.013	1.559	.185		.036	.016	2.250	.083
CAPS (Post, Emotions)[†]		−.008	.010	−0.837	.554		−.012	.010	−1.147	.530
CAPS (Pre, Actions)[a]		−.037	.015	−2.451	.106		−.037	.018	−2.126	.106
CAPS (Post, Actions)[a]		**.063**	.017	3.702	.045		**.063**	.021	2.986	.046
CAPS	mPFC	−.001	.009	−0.161	.986	pIFG	−.017	.006	−2.715	.089
CAPS × Session × Stimulus		**−.011**	.003	−3.529	.002		**−.005**	.002	−2.108	.050
CAPS (Pre, Emotions)[a]		.009	.020	0.435	.670		.023	.020	1.185	.289
CAPS (Post, Emotions)[a]		−.008	.017	−0.477	.640		−.008	.014	−0.569	.640
CAPS (Pre, Actions)[a]		−.042	.022	−1.877	.111		**−.037**	.006	−5.882	.003
CAPS (Post, Actions)[a]		.022	.016	1.375	.206		.044	.029	1.511	.193
CAPS	vmPFC	.003	.006	0.548	.986	dPMC	−.007	.007	−1.074	.719
CAPS × Session × Stimulus		−.004	.003	−1.495	.157		−.004	.003	−1.431	.158
CAPS (Pre, Emotions)[a]		Interaction NS					Interaction NS			
CAPS (Post, Emotions)[a]		Interaction NS					Interaction NS			
CAPS (Pre, Actions)[a]		Interaction NS					Interaction NS			
CAPS (Post, Actions)[a]		Interaction NS					Interaction NS			
CAPS	PCC	.013	.006	2.225	.152	IPS	.010	.005	1.796	.261
CAPS × Session × Stimulus		**−.011**	.003	−3.093	.006		**−.008**	.003	−3.041	.006
CAPS (Pre, Emotions)[a]		**.049**	.012	3.977	.009		**.021**	.007	2.847	.047
CAPS (Post, Emotions)[a]		−.015	.015	−1.002	.530		−.030	.010	−2.899	.084
CAPS (Pre, Actions)[a]		−.075	.034	−2.248	.106		−.026	.025	−1.051	.324
CAPS (Post, Actions)[a]		.042	.027	1.573	.193		**.056**	.017	3.277	.045
CAPS	TPJ	.000	.005	−0.018	.986	LOTC	−.003	.005	−0.488	.986
CAPS × Session × Stimulus		**−.010**	.002	−4.075	.001		**−.009**	.002	−4.520	<.001
CAPS (Pre, Emotions)[a]		.035	.017	2.091	.085		.033	.015	2.219	.083
CAPS (Post, Emotions)[a]		−.025	.014	−1.714	.282		−.026	.013	−2.014	.244
CAPS (Pre, Actions)[a]		−.027	.010	−2.761	.098		−.035	.018	−1.913	.111
CAPS (Post, Actions)[a]		.059	.028	2.131	.105		0.049	.020	2.492	.075

Note: In patients with PTSD, Why/How neural responses and were analyzed in LMEMs with CAPS and CAPS × Session × Stimulus as effects; the intercept, Session, Stimulus, and Session × Stimulus were nested within Subject. Post hoc CAPS simple effects tests were conducted for ROIs with significant CAPS × Session × Stimulus interactions (p_{FDR} < .05). Bolded coefficients have corresponding P_{FDR} values < .05 (the exact significance values are shown in the P_{FDR} column in each row).

Abbreviations: *b*, regression coefficient; CAPS, Clinician-Administered PTSD Scale; DMN, default mode network; dPMC, dorsal premotor cortex; IPS, intraparietal sulcus; LMEM, linear mixed-effects model; LOTC, lateral occipitotemporal cortex; MNS, mirror neuron system; mPFC, medial prefrontal cortex; NS, not significant (p_{FDR} > .05); PCC, posterior cingulate cortex; p_{FDR}, *p* value adjusted for false discovery rate (<.05); pIFG, posterior inferior frontal gyrus; ROI, region of interest; *SE*, standard error of regression coefficient; TPJ, temporoparietal junction; vmPFC, ventromedial prefrontal cortex.

[a]Post hoc simple effect test.

2012b, 2013; Wagner, Kelley, & Heatherton, 2011). Similarly, MNS activation can be modulated by top-down and bottom-up attention (Engel, Burke, Fiehler, Bien, & Rösler, 2008; Hesse, Sparing, & Fink, 2009; Kilner, Marchant, & Frith, 2006; Spunt & Lieberman, 2012b, 2013).

An attentional account is further supported by the anatomical overlap between the attentional networks and regions with significant PTSD-related effects in the present study. With the exception of mPFC, ROIs with significant effects appear to overlap with the DAN, VAN, and FPCN. DAN is involved in top-down attention and includes IPS, dPMC, and LOTC (Corbetta & Shulman, 2002; Vossel, Geng, & Fink, 2014). VAN is involved in bottom-up attention and includes TPJ and pIFG (Corbetta, Patel, & Shulman, 2008; Vossel et al., 2014). FPCN includes parts of PCC and is thought to facilitate attentional control by mediating activity between DMN, DAN, and other networks (Dixon et al., 2018; Leech, Braga, & Sharp, 2012). Moreover, whole-brain analyses revealed PTSD-related effects in one region wholly outside our a priori ROIs: the dlPFC, a central node of DAN and FPCN (Dixon et al., 2018). Our results align with numerous reports of affect-evoked hyperactivation in DAN, VAN, and FPCN in PTSD (Block & Liberzon, 2016; Fani et al., 2012; Morey et al., 2008; Pannu Hayes et al., 2009; White, Costanzo, Blair, & Roy, 2015). Taken together, affective attentional biases may drive widespread affect-selective hyperactivation throughout the social brain in PTSD.

4.3 | Affect labeling training

The PTSD group underwent affect labeling training, which involves labeling the emotional content of stimuli (Lieberman et al., 2007). Affect labeling is an emotional inhibitory regulation strategy that has

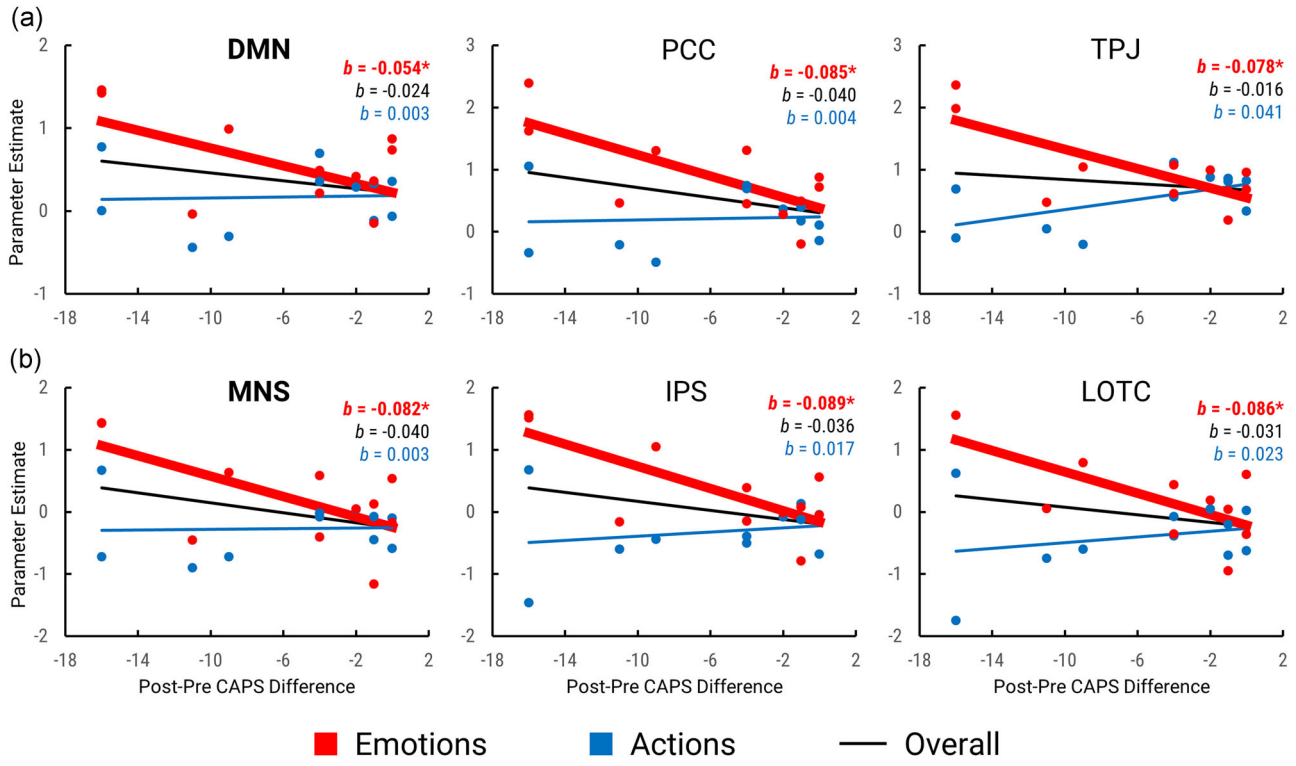

FIGURE 5 Prediction of training outcomes from pretraining neural responses in patients with PTSD who completed affect labeling training. The scatterplots show Why/How parameter estimates and post-pre CAPS score differences in (a) DMN and (b) MNS ROIs with significant predictive effects ($p_{FDR} < .05$), with regression lines, plotted for main (overall) and simple (Stimulus-specific) effects. Bolded lines represent significant regression coefficients, while thin lines represent nonsignificant regression coefficients. *b*, regression coefficient; CAPS, Clinician-Administered PTSD Scale; DMN, default mode network; FDR, false discovery rate; IPS, intraparietal sulcus; LOTC, lateral occipitotemporal cortex; MNS, mirror neuron system; PCC, posterior cingulate cortex; PTSD, posttraumatic stress disorder; ROI, region of interest; TPJ, temporoparietal junction. $^{*}p_{FDR} < .05$

been found to downregulate amygdala responses via right ventrolateral prefrontal cortex (vlPFC) in healthy subjects (Burklund, Creswell, Irwin, & Lieberman, 2014; Torrisi, Lieberman, Bookheimer, & Altshuler, 2013). Though we did not find PTSD-related effects in amygdala or vlPFC, affect labeling training was found to reduce symptom severity (Burklund et al., Under review). Affect labeling may inhibit affective biases in social inference processing, as reactivity to emotional stimuli became negatively correlated with symptom severity after training, a reversal of the positive correlation found pretraining (Figure 4). Moreover, posttraining symptom improvement was predicted by higher pretraining activation to emotional stimuli (Figure 5), suggesting that engagement with emotional stimuli enhances the efficacy of affect labeling training. These results are consistent with studies on related PTSD interventions (Badura-Brack et al., 2015; Barry, Sewart, Arch, & Craske, 2015; Craske et al., 2008; Jaycox, Foa, & Morral, 1998; Niles, Mesri, Burklund, Lieberman, & Craske, 2013). Taken together, affect labeling may be best suited for patients with greater affective biases in social cognition.

4.4 | Limitations and future directions

The interpretation of these results should be tempered by the relatively small sample size of this study, especially in the posttraining analyses. Additionally, generalizability may be limited by our selective recruitment of American veterans exposed to combat trauma. Future studies should use larger and more diverse samples. It should be noted that the primary measure in this study, the Why/How BOLD contrast, collapses across the Why and How conditions, thus making it impossible to distinguish the specific contributions of either condition to the evoked responses. Future studies should attempt to disentangle the two conditions. A potential confound in this study are nonaffective stimulus differences between emotional facial expressions (Emotions) and intentional hand actions (Actions); our key finding of Emotions-selective hyperactivation may not be exclusively driven by affect. Future studies should better match emotional and nonemotional stimuli. Another caveat is the putative nature of the functional-anatomic overlap between our findings and the attention networks. This overlap was inferred using reverse inference from existing literature, a form of reasoning that can be tenuous (Aguirre & Feinberg, 2003; Poldrack, 2006). This functional-anatomic overlap could be more definitively investigated by including functional localizers for DAN, VAN, and FPCN in addition to DMN and MNS in future studies. More broadly, the use of connectivity analyses, machine learning techniques, and other neuroimaging modalities will be

TABLE 3 Predicting training outcomes from pretraining neural responses

	DMN ROIs					MNS ROIs				
Effect	**ROI**	***b***	***SE***	***t***	p_{FDR}	**ROI**	***b***	***SE***	***t***	p_{FDR}
$CAPS_{diff}$	DMN	−.024	.018	−1.307	.380	MNS	−.040	.022	−1.842	.270
$CAPS_{diff}$ × Stimulus		**−.030**	.009	−3.309	.005		**−.043**	.010	−4.272	.001
$CAPS_{diff}$ (Emotions)[a]		**−.054**	.022	−2.449	.049		**−.082**	.030	−2.753	.036
$CAPS_{diff}$ (Actions)[a]		.003	.019	0.153	.987		.003	.022	0.118	.987
$CAPS_{diff}$	mPFC	−.015	.035	−0.426	.727	pIFG	−.030	.033	−0.932	.454
$CAPS_{diff}$ × Stimulus		−.023	.014	−1.654	.127		**−.034**	.009	−3.778	.002
$CAPS_{diff}$ (Emotions)[a]		Interaction NS					−.064	.037	−1.732	.117
$CAPS_{diff}$ (Actions)[a]		Interaction NS					.003	.033	0.104	.987
$CAPS_{diff}$	vmPFC	−.005	.015	−0.354	.727	dPMC	−.039	.033	−1.170	.380
$CAPS_{diff}$ × Stimulus		−.010	.011	−0.942	.358		**−.039**	.014	−2.734	.016
$CAPS_{diff}$ (Emotions)[a]		Interaction NS					−.077	.039	−1.971	.092
$CAPS_{diff}$ (Actions)[a]		Interaction NS					−.001	.038	−0.017	.987
$CAPS_{diff}$	PCC	−.040	.016	−2.519	.209	IPS	−.036	.018	−1.990	.270
$CAPS_{diff}$ × Stimulus		**−.051**	.013	−3.954	.002		**−.056**	.013	−4.453	.001
$CAPS_{diff}$ (Emotions)[a]		**−.085**	.024	−3.503	.013		**−.089**	.024	−3.644	.013
$CAPS_{diff}$ (Actions)[a]		.005	.024	0.206	.987		.017	.026	0.643	.987
$CAPS_{diff}$	TPJ	−.017	.015	−1.146	.380	LOTC	−.031	.023	−1.341	.380
$CAPS_{diff}$ × Stimulus		**−.050**	.010	−5.273	<.001		**−.057**	.010	−5.908	<.001
$CAPS_{diff}$ (Emotions)[a]		**−.078**	.021	−3.704	.013		**−.086**	.024	−3.536	.013
$CAPS_{diff}$ (Actions)[a]		.041	.018	2.289	.383		.023	.029	0.798	.987

Note: In patients with PTSD who completed affect labeling training, pretraining Why/How neural responses were analyzed in LMEMs with $CAPS_{diff}$ and $CAPS_{diff}$ × Stimulus as effects; the intercept and Stimulus were nested within Subject. Post hoc $CAPS_{diff}$ simple effects tests were conducted for ROIs with significant $CAPS_{diff}$ × Stimulus interactions (p_{FDR} < .05). Bolded coefficients have corresponding P_{FDR} values < .05 (the exact significance values are shown in the P_{FDR} column in each row).

Abbreviations: *b*, regression coefficient; CAPS, Clinician-Administered PTSD Scale; CAPSdiff, post-pre CAPS score difference; DMN, default mode network; dPMC, dorsal premotor cortex; IPS, intraparietal sulcus; LMEM, linear mixed-effects model; LOTC, lateral occipitotemporal cortex; MNS, mirror neuron system; mPFC, medial prefrontal cortex; NS, not significant (p_{FDR} > .05); PCC, posterior cingulate cortex; p_{FDR}, *p* value adjusted for false discovery rate (<.05); pIFG, posterior inferior frontal gyrus; ROI, region of interest; *SE*, standard error of regression coefficient; TPJ, temporoparietal junction; vmPFC, ventromedial prefrontal cortex.

[a]Post hoc simple effect test.

critical in incisively characterizing the neurobiology of social inference impairments in PTSD.

5 | CONCLUSION

In the first neuroimaging investigation of social inference in PTSD, the social brain was found to be broadly selective towards emotional stimuli in PTSD. Affect-selective hyperactivation throughout DMN, MNS, and beyond differentiated patients with PTSD from controls, correlated with symptom severity, and predicted training outcomes. Despite this, PTSD-related effects were not significant in core affective regions. Instead, our data putatively highlight the role of attentional processes in disrupting social cognitive processing in PTSD. These results strongly warrant further study of social inference processing in PTSD, specifically in disentangling the roles of effect and attention, and for developing more targeted PTSD interventions.

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CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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