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Decreased somatosensory activity to non-threatening touch in combat veterans with posttraumatic stress disorder

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ABSTRACT

Posttraumatic stress disorder (PTSD) is a severe psychiatric disorder prevalent in combat veterans. Previous neuroimaging studies have demonstrated that patients with PTSD exhibit abnormal responses to non-threatening visual and auditory stimuli, but have not examined somatosensory processing. Thirty male combat veterans, 16 with PTSD and 14 without, completed a tactile stimulation task during a 306-sensor magnetoencephalography (MEG) recording. Significant oscillatory neural responses were imaged using a beamforming approach. Participants also completed clinical assessments of PTSD, combat exposure, and depression. We found that veterans with PTSD exhibited significantly reduced activity during early (0–125 ms) tactile processing compared with combat controls. Specifically, veterans with PTSD had weaker activity in the left postcentral gyrus, left superior parietal area, and right prefrontal cortex in response to nonthreatening tactile stimulation relative to veterans without PTSD. The magnitude of activity in these brain regions was inversely correlated with symptom severity, indicating that those with the most severe PTSD had the most abnormal neural responses. Our findings are consistent with a resource allocation view of perceptual processing in PTSD, which directs attention away from non-threatening sensory information.

1. Introduction

Posttraumatic stress disorder (American Psychiatric Association, 2000, 2013) is a significant psychiatric disorder, which may occur in the aftermath of combat exposure. The symptom picture in PTSD is complex, including re-experiencing, avoidance, mood, and hyperarousal symptoms (American Psychiatric Association, 2000, 2013). The lifetime incidence of PTSD is roughly 7–9% of the US population (Kessler et al., 1995, 2005; American Psychiatric Association, 2013), but PTSD is reported in 13–22% of recent veterans (Seal et al., 2007, 2009).

Neuroimaging studies in PTSD demonstrate the clear importance of brain structures implicated in fear processing including the amygdalae, hippocampi, anterior cingulate, and insula, (Rabinak et al., 2010; Morey et al., 2012; Pitman et al., 2012; Sripada et al., 2012) as would be expected in a disorder rooted in traumatic, fear-provoking events. Imaging studies have also highlighted executive functioning deficits in patients with PTSD (Polak et al., 2012) and noted widespread alterations in parietal, frontal, and occipital areas (Eckart et al., 2011; Liu et al., 2012; Qi et al., 2013; Gong et al., 2014) consistent with cognitive models of PTSD emphasizing disrupted attentional and perceptual processes (Ehlers and Clark, 2000).

Electrophysiological studies have examined auditory and visual sensory processing in patients with PTSD. Most of these studies have recorded event-related potentials (ERP) using trauma-eliciting stimuli and shown significant increases in the P300 response (for a review see Javanbakht et al. (2011)), although Bae et al. (2011) found reduced P300 current-source density in patients with PTSD compared to healthy controls in response to non-threatening

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auditory stimuli. These seemingly disparate results are consistent with a meta-analysis of ERP studies in PTSD, which found enhanced responses for trauma-related stimuli and reduced responses for neutral stimuli, particularly in the parietal cortex (Karl et al., 2006a). Since the P300 is thought to reflect top-down information processing, these findings may suggest reduced allocation of cognitive resources in response to stimuli evaluated as non-threatening (Karl et al., 2006a).

To understand the bottom-up somatosensory alterations associated with PTSD, several studies have focused on evoked responses earlier in the time-course. For example, neural responses from stimuli onset (0 ms) to roughly 150 ms are thought to reflect pre-attentive automatic functions such as stimulus registration and sensory filtering, Many studies of the preattentive time-course have assessed sensory gating by presenting paired stimuli so closely together in time that the normal response to the second stimulus is sharply reduced (i.e., gated). Gating studies in PTSD have consistently shown impaired early gating (Javanbakht et al., 2011) including reduced habituation to the second tone in PTSD patients compared to trauma and no-trauma controls (Karl et al., 2006b). This failure to suppress or habituate to repeating stimuli in both auditory (Neylan et al., 1999; Ghisolfi et al., 2004; Holstein et al., 2010; Gjini et al., 2013) and visual (Gjini et al., 2013) modalities suggests that PTSD patients have difficulty filtering out irrelevant sensory input.

Interestingly, studies focusing on evoked responses to neutral stimuli, have provided perhaps the most critical evidence for basic somatosensory alterations in PTSD. For example, a recent study showed that survivors of torture with PTSD have significantly smaller primary auditory and visual responses to neutral stimuli compared to controls, reflecting a decreased early response after simple stimuli presentation (Gjini et al., 2013). Likewise, a MEG study by Hunter et al. (2011) found attenuated source strength in response to neutral stimuli in the right auditory area of the PTSD group compared to healthy controls. Similar deficits in early visual processing of neutral pictures have been described (Felmingham et al., 2011; Mueller-Pfeiffer et al., 2013), and a recent fMRI study identified diminished activity in the ventral visual stream, and dorsal and ventral attention systems in PTSD patients compared to controls (Mueller-Pfeiffer et al., 2013). Thus, processing deficits early in the time-course may be associated with impaired attentional processes consistent with automaticity of PTSD symptoms. From a clinical perspective, individuals with PTSD self-report greater sensory filtering disruption in perceptual modulation, including heightened stimulus sensitivity and sensory flooding, as well as general distractibility compared with trauma exposed and no trauma comparison groups (Stewart and White, 2008). Therefore, processing impairments are perceptible in the daily lives of PTSD patients.

The current study aimed to investigate oscillatory activity in response to non-threatening somatosensory stimuli (i.e., light airpuffs) in recent combat veterans with and without PTSD. Cortical oscillations, like evoked-potentials, are reflective of information processing in the brain and are a very sensitive measure of neuronal coding and communication both intra- and inter-regionally (Uhlhaas et al., 2009; Lisman and Jensen, 2013; Friston et al., 2015). Recent studies have connected information processing deficits in psychiatric and neurological conditions to aberrant cortical oscillatory activity, including Parkinson's disease (Heinrichs-Graham et al., 2014a, 2014b), cognitive impairment (Wilson et al., 2013a, 2013b, 2015), autism (Wilson et al., 2007; Rojas et al., 2011; Rojas and Wilson, 2014), attention-deficit/hyperactivity disorder (Wilson et al., 2012, 2013c, 2013d; Franzen et al., 2013), and other disorders (Uhlhaas and Singer, 2010, 2012). However, oscillations have rarely been studied in PTSD and have yet to be examined in somatosensory processing, which involves strong oscillations in controls (Gaetz and Cheyne, 2006). Our primary aim was to examine potential differences in somatosensory tactile processing without attempting any manipulation of the stimuli to increase threat perception. Given the literature indicating reduced primary auditory and visual responses to neutral stimuli in PTSD, we hypothesized that combat veterans with PTSD would have reduced cortical activity early in the time course in the contralateral primary somatosensory cortex and parietal lobe compared to healthy, demographically matched combat veterans without PTSD.

2. Methods and materials

2.1. Subject selection

We evaluated a community sample of 30 Operation Iraqi Freedom and Operation Enduring Freedom (OIF/OEF) combat veterans. All participants were male and right-handed. Sixteen of the veterans were diagnosed with PTSD according to DSM-IV criteria (2) using the Clinical Administered PTSD Scale (CAPS) (Blake et al., 1995) and the F1/I2 rule (Weathers et al., 1999). The remaining 14 combat veterans were age-matched to the patient group and did not have PTSD or any other psychiatric diagnosis, as validated by the CAPS and the Mini International Neuropsychiatric Interview (M.I.N.I.; Sheehan et al., 1998). All participants also completed the Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) to assess depression, and a measure of combat exposure (Vogt et al., 2008) to assess trauma severity. Other exclusionary criteria included any known central nervous system disease, neoplasm, or lesion; history of significant head injury, or ferromagnetic implants/shrapnel. The Creighton University Institutional Review Board approved the study protocol, and all participants provided their written informed consent to participate in this study.

2.2. Experimental paradigm

Participants were seated in a custom chair within the magnetically-shielded room with their head positioned inside the helmet-shaped MEG sensor array. They were instructed to remain still with both arms resting on a tray attached to the chair, while unilateral tactile stimulation was applied to the pad of the fifth digit of the right hand using a small airbladder (Fig. 1). For each participant, more than 160 trials were collected using an inter-stimulus interval that varied randomly between 2.5 and 4.0 s.

2.3. MEG data processing and statistics

2.3.1. MEG data acquisition and sMRI coregistration

All recordings were conducted in a one-layer magnetically-shielded room (MSR) with active shielding engaged. With an acquisition bandwidth of 0.1–330 Hz, neuromagnetic responses were sampled continuously at 1 kHz using an Elekta Neuromag system with 306 magnetic sensors, including 204 planar gradiometers and 102 magnetometers (Elekta, Helsinki, Finland). Using MaxFilter (v2.2; Elekta), MEG data from each participant were individually corrected for head motion and subjected to noise reduction using the signal space separation method with a temporal extension (tSSS; Taulu et al., 2005; Taulu and Simola, 2006). Prior to MEG measurement, four coils were attached to the participant's head and the locations of these coils, together with the three fiducial points and scalp surface, were determined with a 3-D digitizer (Fastrak 3SF0002, Polhemus Navigator Sciences, Colchester, VT, USA). Once the participant was positioned for MEG recording, an electric current with a unique frequency label (e.g., 322 Hz) was fed to each of the coils. This induced a measurable field and allowed each coil to be localized in reference to the sensors throughout the recording session. Since coil locations were also known in head coordinates, all MEG measurements could be transformed into a common coordinate system. With this coordinate system (including the scalp surface points), each participant's MEG data was coregistered with T1-weighted structural magnetic resonance imaging (sMRI) data for source space analyses. sMRI data were aligned parallel to the anterior and posterior commissures and were transformed into standard space after beamforming using BESA MRI (Version 2.0; BESA GmbH, Gräfelfing, Germany).

2.3.2. MEG preprocessing, time–frequency transformation, and statistics

Cardio-artifacts were removed from the data using signal-space projection (SSP) and the projection operator was accounted for during source reconstruction (Uusitalo and Ilmoniemi, 1997). Artifact rejection was based on a fixed threshold method, supplemented with visual inspection. Epochs were of 1.1 s duration (–0.4 to 0.7 s), with 0.0 s defined as stimulation onset and the baseline being the –0.4 to 0.0 s window. For each participant, at least 115 artifact-free epochs remained for further analysis.

Artifact-free epochs were transformed into the time–frequency domain using