Exp Brain Res (2015) 233:2013–2020
DOI 10.1007/s00221-015-4272-2

RESEARCH ARTICLE

Neural communication in posttraumatic growth

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Received: 9 January 2015 / Accepted: 2 April 2015 / Published online: 17 April 2015

Abstract Posttraumatic growth (PTG), or positive psychological changes following exposure to traumatic events, is commonly reported among trauma survivors. In the present study, we examined neural correlates of PTG in 106 veterans with PTSD and 193 veteran controls using task-free magnetoencephalography (MEG), diagnostic interviews and measures of PTG, and traumatic event exposure. Global synchronous neural interactions (SNIs) were significantly modulated downward with increasing PTG scores in controls ($p = .005$), but not in veterans with PTSD ($p = .601$). This effect was primarily characterized by negative slopes in local neural networks, was strongest in the medial prefrontal cortex, and was much stronger and more extensive in the control than the PTSD group. The present study complements previous research highlighting the role of neural adaptation in healthy functioning.

Keywords Posttraumatic growth · Neuroimaging · Magnetoencephalography

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Introduction

To understand the range of possible trauma sequelae, researchers have examined positive psychological changes following traumatic events, referred to as posttraumatic growth (PTG). PTG differs from resilience, or relatively good adaptation despite significant adversity (Curtis and Cicchetti 2007), by stressing improvements in areas of importance instead of the absence of impaired functioning. These perceived positive changes, including feelings of strength, becoming closer to loved ones, and appreciating life more, have been reported after a range of events such as brain injury (Collicutt McGrath and Linley 2006) and sexual assault (Frazier et al. 2001) and have been the focus of interventions (Garland et al. 2007).

The relationship between PTG and posttraumatic stress has been inconclusive. For instance, some research has found a moderate positive association between PTG and posttraumatic stress symptoms (Park et al. 1996; Schorr and Roemer 2002), whereas others find a large negative association between the two (Frazier et al. 2001), and many find no relationship (see Zoellner and Maercker 2006 for a review).

The inconsistencies in PTG research have raised important conceptual questions, including: (1) does self-reported growth correspond to actual positive behavioral change (Frazier et al. 2009)? (2) do survivors tend to derogate their past compared to present (McFarland and

Alvaro 2000)? (3) is PTG a process instead of an outcome (McMillan and Cook 2003)? and (4) are the most commonly used measures of growth valid (Frazier et al. 2009)? PTG research has relied solely on self-reported status to address these issues. Because technological advances allow neuroscience methods to measure non-self-report outcomes of psychological processes, interest in these methods has exploded. Researchers can record in-the-moment psychological processes that might otherwise be impossible to assess (Harmon-Jones and van Honk 2012).

Previous research on neural correlates of PTG

Although the neural correlates of PTSD and other negative consequences of trauma are well researched, and meta-analyses have identified both functional and structural differences in several areas of the brain (Eckart et al. 2011; Etkin and Wager 2007; Karl et al. 2006; Lanius et al. 2002), only one study has examined neural correlates of PTG (Rabe et al. 2006). Resting state EEG was assessed in motor vehicle accident survivors. Increased relative left fronto-central activation was significantly related to self-perceived PTG. The authors concluded that self-perceived PTG reflects approach-related motivational tendencies indexed by relative left frontal activity. Although this is an intriguing initial study in this area, additional research addressing some of the methodological limitations is warranted.

Previous research using MEG and PTSD

Magnetoencephalography (MEG) is a noninvasive neuroimaging technique that maps brain activity by recording magnetic fields that naturally occur in the brain (Hämäläinen and Hari 2002). MEG is less sensitive to conductivity of the head than EEG and thus provides more accurate localization of neural activation (Hämäläinen et al. 1993). Using synchronous neural interactions (SNI) biomarkers (Georgopoulos et al. 2007), MEG research has already identified a unique PTSD neural signature characterized by miscommunication of temporal and parietal and/or parieto-occipital right hemispheric areas (Engdahl et al. 2010; Georgopoulos et al. 2010). A recent study using task-free MEG found that global synchronous neural interactions (GSNI) decreased with increasing exposure to traumatic events in a resilient control veteran group; however, there was no significant relationship in a group of veterans with PTSD (James et al. 2013). This effect was significantly stronger for the right compared to the left hemisphere in the resilient controls, but that there was no statistical difference in the PTSD group. The researchers interpreted this as evidence of the brain's ability to adapt to trauma exposure; that is, neural network decorrelation is "a mechanism by which the network is 'freed' from the hold of a particular input (e.g., sensory stimulus or, in our case, trauma event) and becomes available for encoding new information (James et al. 2013, pg. 16)." That study did not measure growth directly, but instead used an absence of PTSD in the presence of trauma exposure as a proxy for resilience. The current paper extends these findings to examine the specific neural processes involved in PTG by measuring PTG directly.

Present study

We sought to detect neural markers of PTG using MEG in veterans with and without PTSD diagnoses with varying degrees of trauma exposure. Based on past research, we anticipated that more PTG would be associated with decorrelated SNI, particularly in the left hemisphere, and that this relationship would be independent of trauma history.

Materials and methods

Participants and procedures

Two hundred and ninety-nine U.S. veterans including 193 controls and 106 veterans diagnosed with PTSD according to the Diagnostic and Statistical Manual of Mental Disorders, fourth edition, text revision (DSM-IV-TR; American Psychiatric Association 2000) participated in the study as paid volunteers. In the control group, there were 182 men (age mean ± SD, 59.69 ± 13.36 years) and 11 women (41.38 ± 15.42 years); in the PTSD group, there were 96 men (52.84 ± 15.09 years) and 10 women (42.48 ± 11.51 years). Veterans without mental health concerns and those with a primary PTSD diagnosis according to medical chart were recruited. Exclusion criteria included active substance use disorders, serious chronic pain and other central nervous system disorders, a history of psychosis or bipolar disorder, moderate or severe TBI, and cardiac pacemakers or other imbedded ferrous metal (due to magnetic effects on MEG). Veterans who met eligibility criteria completed diagnostic interviews and the questionnaires described below and underwent a MEG scan. The study protocol was approved by the Institutional Review Board at the Minneapolis VA Medical Center, and participants provided written informed consent prior to the study.

Measures

These data are part of a larger study (e.g., see James et al. 2013 for a recent paper published using some of the same

data addressing a different research question); only measures relevant to the present analyses are described here.

PTSD symptoms

PTSD was assessed using the Clinician-Administered PTSD Scale for DSM-IV-TR (CAPS; Blake et al. 1995) or the Structured Clinical Interview for DSM-IV-TR (SCID; First et al. 2002). CAPS symptom scores were converted to dichotomous scores using the SCID Symptom Calibration method (SXCAL; Weathers et al. 1999) to categorize participants with regard to PTSD status. Criterion A2 was relaxed when diagnosing PTSD, consistent with Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-V; American Psychiatric Association 2014). PTSD diagnoses were associated with a variety of traumatic events, including combat, childhood abuse, and sexual assault. The SCID (First et al. 2002) and DSM-IV-TR criteria were used to assess lifetime history of non-PTSD axis I diagnoses. Individuals with current diagnoses other than PTSD were excluded.

Posttraumatic growth

PTG was assessed using the Posttraumatic Growth Inventory (PTGI; Tedeschi & Calhoun 1996). The PTGI is a 21-item instrument designed to measure positive outcomes of traumatic events. Participants first select from a list the most "upsetting, traumatic, or life-altering event" that has occurred in their life (e.g., loss of a loved one, job loss, combat). They then answered each item on a scale of 0 ("I did not experience this change as a result of the above event") to 5 ("I experienced this change to a very great degree as a result of the above event"); the PTG total was calculated by summing the items, so that scores ranged from 0 to 105. For the present sample, internal consistency was excellent (Cronbach's alpha = .931).

Traumatic events

The Deployment Risk and Resilience Inventory (DRRI; King et al. 2006) assessed lifetime trauma exposure. The DRRI is a collection of 14 scales that assess pre-deployment, deployment, and post-deployment-related factors implicated in risk for distress and resiliency. Three scales (pre-deployment stressors, combat experiences, and post-deployment stressors), totaling 47 dichotomously scored items, were included in the present study. The items were summed to provide an indicator of lifetime exposure to trauma. A corrected trauma score (items endorsed/items answered) was computed and used in all subsequent analyses to account for a small number of missing questions; thus, trauma scores ranged from 0 to 1.

Data Acquisition and Pre-processing

As described previously (Georgopoulos et al. 2007, 2010), participants lay supine within the electromagnetically shielded chamber and fixated their eyes on a spot 65 cm in front of them for 60 s. MEG data were acquired using a 248-channel axial gradiometer system (Magnes 3600WH, 4-D Neuroimaging, San Diego, CA), band filtered between 0.1 and 400 Hz, and sampled at 1017.25 Hz. Following acquisition, single trial MEG data from all sensors were prewhitened using a (50, 1, 1) AutoRegressive Moving Average (ARIMA) model (Box and Jenkins 1976; Priestley 1958) to remove trends and autocorrelations. Then, all possible pairwise zero-lag cross-correlations ($N = 30{,}628$, given 248 sensors) were computed between the prewhitened MEG time series. Finally, the partial, zero-lag cross-correlations PCC_{ij}^{0} between i and j sensors were computed for all sensor pairs (SNI); as such, for any given pair of sensors (from a total of 248), the effects of the remaining 246 were partialed out. The PCC_{ij}^{0} was transformed to z_{ij} using Fisher's (1958) transformation, and its absolute value was transformed again by taking the natural log to normalize the distribution (z'_{ij}).

Data analysis

Statistical analyses were conducted using the IBM SPSS statistical package (version 20 for Windows) and Intel Fortran. Following previous research showing modulation of SNI as a function of trauma (James et al. 2013), we examined the relations between PTG and SNI using similar analyses. Two levels of SNI were defined: global (GSNI) and local (LSNI = z'_{ij}). The strength of GSNI for each subject was estimated as the average z'_{ij} across all sensor pairs. To assess the relations between GSNI and PTG in each group, a multiple linear regression was performed with GSNI as the dependent variable and PTG, lifetime trauma, gender, and age as independent variables. The same analysis was performed for each hemisphere and for each pair of sensors in order to evaluate the effect of trauma on local synchrony (LSNI), i.e., on individual z'_{ij}.

Results

Posttraumatic growth and trauma scores

The frequency distributions of the PTG score for the PTSD and control groups are shown in Fig. 1. Although there are few PTG studies of U.S. veterans, these scores are similar to those obtained from another veteran population (Maguen et al. 2006). There were no statistically significant differences in the PTG score between the control

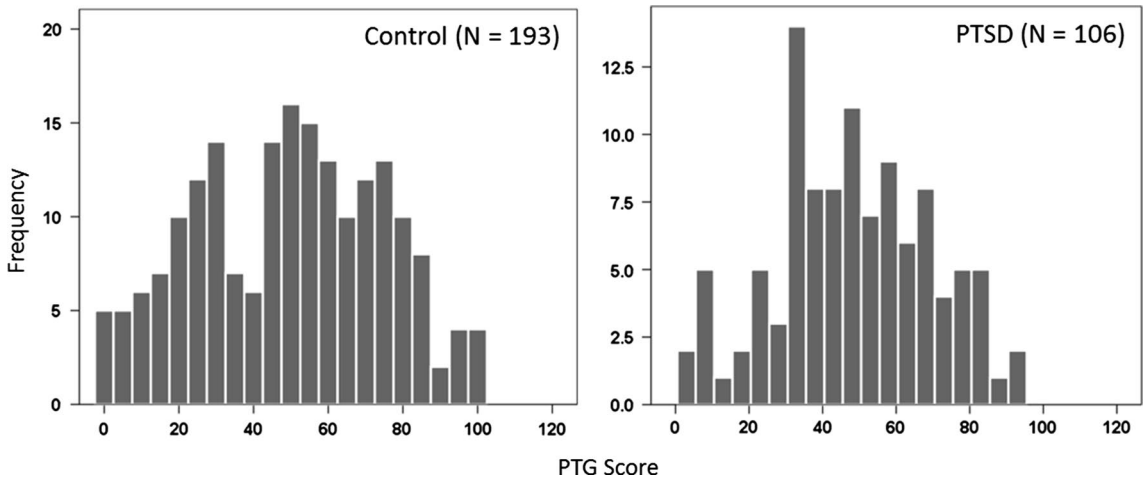

Fig. 1 Frequency distributions of PTG scores in the control and PTSD groups

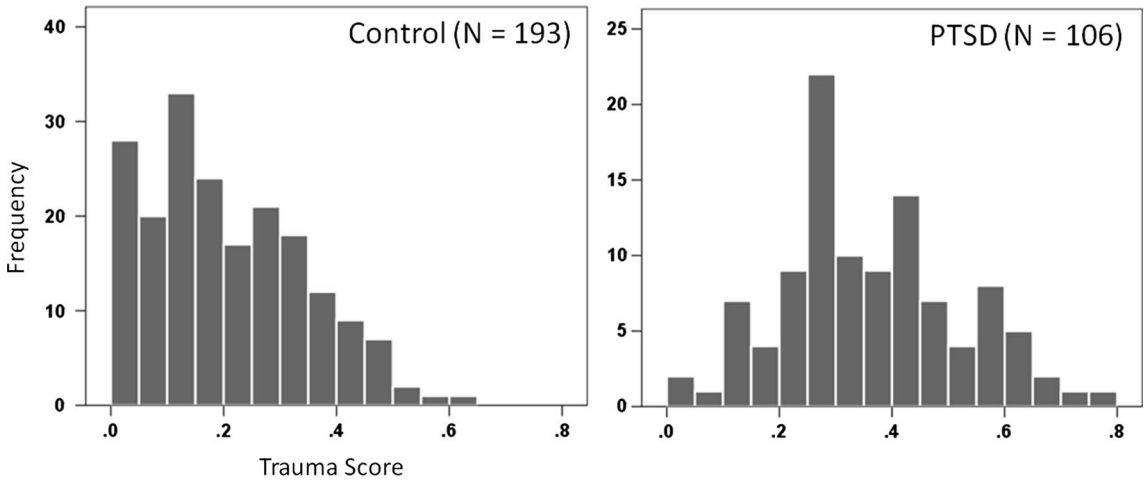

Fig. 2 Frequency distributions of trauma scores in the control and PTSD groups

(mean ± SD, 49.31 ± 25.01, $N = 193$) and PTSD group (50.18 ± 25.94, $N = 106$), $F(1, 294) = 0.26$, $p = .613$ (with age, gender, and lifetime trauma as covariates). In contrast, the trauma score differed significantly between the control (mean ± SD, 0.209 ± 0.139, $N = 193$) and the PTSD group (0.365 ± 0.165, $N = 106$), $F(1, 295) = 92.73$, $p < .001$ (with age and gender as covariates). Thus, we controlled for trauma score in the subsequent analyses. The frequency distribution of trauma score is shown in Fig. 2.

Since PTG is related to trauma, we computed the ratio of PTG per lifetime trauma as the target independent variable. The PTG/trauma ratio was available in all but three participants (all in the control group) for whom the trauma score was zero. The frequency distributions of this ratio differed significantly between the two groups (Kolmogorov–Smirnov test, $Z = 2.789$, $p < .001$; see Fig. 3); control subjects (mean ± SD, 466.11 ± 627.73, $N = 190$) reported more growth per trauma than did PTSD subjects (182.91 ± 261.62, $N = 106$), Wilcoxon $W = 11{,}936.50$, $Z = -5.389$, $p < .001$.

GSNI vs. PTG Score

In the control group, GSNI decreased significantly with increasing PTG score (multiple linear regression analysis, $\beta = -.207$, $p = .005$). No significant relation was found in the PTSD group ($\beta = .053$, $p = .601$). Age, gender, and lifetime trauma did not have a statistically significant effect in either group.

Separate analyses for each hemisphere revealed a significant negative effect of PTG score on GSNI in the control group for both hemispheres ($\beta = -.213$, $p = .004$ and $\beta = -.184$, $p = .013$ for the left and right hemisphere, respectively). No significant effect was observed

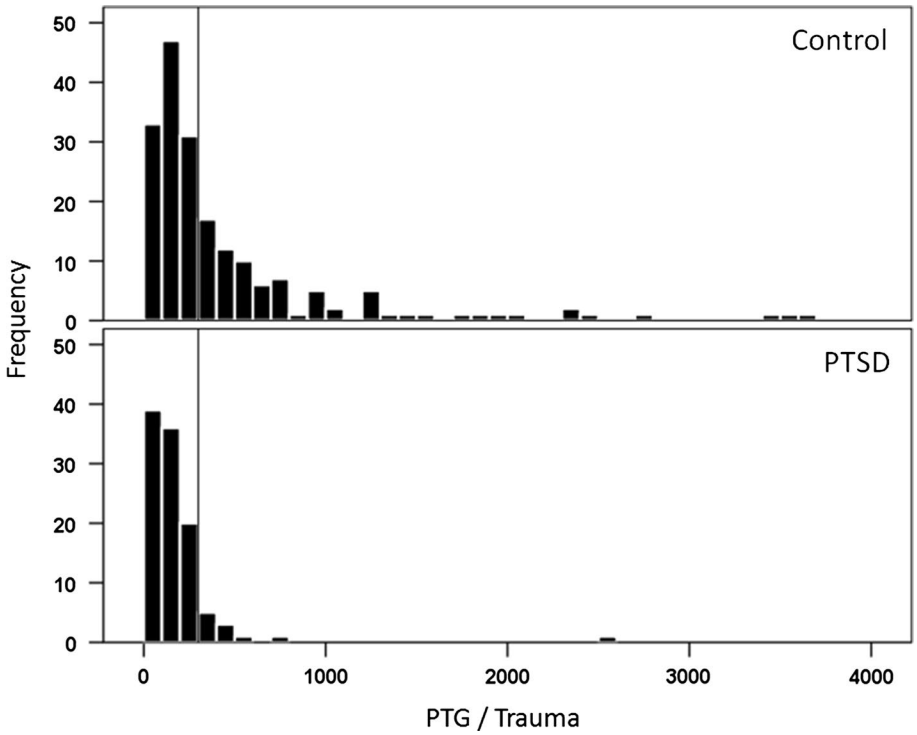

Fig. 3 Frequency distributions of PTG per trauma ratio scores in the control (*top panel*) and PTSD (*bottom panel*) groups

in the PTSD group in either the left or right hemisphere ($\beta = .038$, $p = .709$ and $\beta = .064$, $p = .528$, respectively). There was no significant effect of gender, age, or lifetime trauma in either hemisphere in either group.

LSNI versus PTG score

The partial regression coefficient ("slope" for short) of LSNI vs. PTG score is the key measure and indicates how the strength of the correlation between two neural signals recorded by a pair of sensors changes with the PTG score. The sign of the slope indicates the direction of change (e.g., whether the absolute value of the correlation increases or decreases as the PTG score changes), whereas the absolute value of the slope indicates the strength of that effect. The results are shown in Fig. 4 which contains plots for the negative and positive slopes. To create the plots, a nominal threshold of $p < .0025$ on the significance of the slope was applied to screen out weak effects. There were three striking results of this analysis.

First, modulations of SNI by PTG were much more numerous in the control ($N = 361$ significant SNI vs. PTG slopes exceeding the threshold above) than the PTSD group ($N = 20$). Second, most (94.4 %) of these modulations in the control group were negative slopes, corresponding to a decrease in SNI strength with increasing PTG score. The little neural modulation of PTG in the PTSD group was generally positive as opposed to negative (16/20 = 80 % positive slopes). Finally, although the effect in the control group is evident throughout both hemispheres, there were specific foci where those effects were most prominent. Specifically, the highest modulated interactions (SNIs) were between the left and right medial prefrontal cortices (mPFC), meaning that the interactions between these homologous areas were most frequently decorrelated with increasing PTG score. In addition, parieto-occipital decorrelations with increasing PTG score were more evident in the left than the right hemisphere.

Discussion

We sought to examine the impact of PTG on correlated brain network activity assessed by MEG SNI in a resting state. We had four major findings. First, while the control and PTSD groups reported equivalent PTG scores, the amount of PTG per trauma was much higher in the control than the PTSD group; second, there was a substantial modulation of SNI as a function of PTG in the control but not the PTSD group; third, the vast majority of these SNI vs. PTG relations in the control group were negative, indicating a decrease in SNI strength (i.e., decorrelation) with increasing PTG score; and fourth, those effects were most prominent in the left hemisphere and between left–right mPFC.

Growth from trauma: control versus PTSD

There were no significant group differences in PTG scores between the PTSD and control groups; however,

the amount of PTG per trauma was significantly higher in the control group compared to the PTSD group, highlighting the complex relationship between PTSD and PTG. That is, the controls experienced more positive benefit from trauma than the PTSD group, likely through the process of neural network decorrelation (see below). PTG theorists argue that the cognitive processing and restructuring that occur after trauma lead to alterations in an individual's belief structure of the world, allowing for the integration of the traumatic event (Tedeschi and Calhoun 2004). It may be that the decorrelation in the control group "frees up" neural space which allows PTG to occur.

Neural modulation from PTG: control versus PTSD

Systematic modulations were observed in the control but not the PTSD group. Specifically, (a) GSNI significantly decreased (i.e., decorrelated) with increasing PTG score, and (b) this effect was present only in the controls. These findings are very similar to the findings by James et al. (2013) where a decrease in GSNI with respect to trauma was found in controls only. Thus, the fundamental neural operation in both trauma adaptation and PTG seems to involve decorrelation within brain networks. While the decorrelated trauma and PTG networks are partially overlapping, they are also quite distinct. Specifically, both of them involve the left and right hemispheres, but the effect is more prominent in the left hemisphere for PTG and in the right hemisphere for trauma. For example, the dramatic focus on the right superior temporal gyrus for trauma James et al. (2013) is absent in the PTG case (Fig. 4). In contrast, the dense decorrelation between left and right mPFC observed here is absent in the trauma study. In fact, this is an important qualitative difference between trauma adaptation and PTG—the latter involves interhemispheric decorrelation, whereas decorrelation associated with trauma adaptation is generally confined to short distance, within hemisphere networks.

Neural networks of posttraumatic growth

The strongest PTG-related modulation was observed in the mPFC in the controls, suggesting that this particular region is integral in the processes described above. Research has demonstrated relationships between the mPFC and decision making, executive control, reward-guided learning, and decision making about risk, reward and memory (Euston et al. 2012). In addition, a robust literature supports the role of the mPFC in the expression, encoding and inhibition of fear behavior (Etkin et al. 2011; Courtin et al. 2013), and lesions in the dorsal mPFC or inactivation reduced fear expression (Courtin et al. 2013). Thus, decorrelation in the mPFC may facilitate PTG through decreased threat appraisal, expression of fear and/or need to actively work to inhibit conditioned feared responses.

It is noteworthy that there was essentially no modulation in the mPFC in the PTSD group, in stark contrast to the control group. This lack of relationship suggests that the neural networks of those in the PTSD group may be getting "stuck" with fear encoding, expression, and failure to inhibit fear behavior and thus are unable to integrate and process the traumatic event sufficiently to allow for PTG.

Neural network decorrelation may be a process through which the network is released from the processing of a trauma and is, therefore, available to process other information as has been convincingly argued by our colleagues (James et al. 2013). This explanation corresponds with some of the original explanations of how PTG may occur and many theories regarding the instantiation of PTSD (Tedeschi and Calhoun 2004; Foa and Kozak 1986; Brewin et al. 1996). Those with decorrelated networks may have completed, or nearly completed, the processing and integration theorized to be necessary for PTG to occur, thus leaving their networks free to integrate other information.

Limitations

We sought to increase understanding of PTG-related neural activity. Given that this is only the second study in this area,

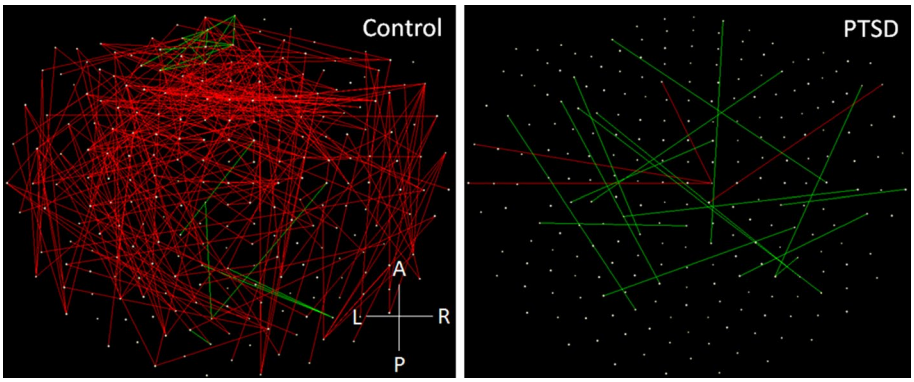

Fig. 4 Two-dimensional (2D) sensor–space plots depicting the negative and positive modulation of LSNI in the control (*left panel*) and PTSD (*right panel*) groups. The *color* intensity is proportional to the maximum PTG vs. $|z'_{ij}|$ slope for a specific sensor (of 247 possible). For both panels: A indicates anterior; *P* posterior, *L* left, and *R* right (color figure online)

we chose to compare two diagnostically distinct groups: those with and without PTSD. Therefore, these results are unlikely to generalize to other more common groups such as those with comorbid mental health problems. However, this work does provide evidence for a non-self-report marker of PTG.

Implications

This is the first MEG study examining how PTG modulates neural activity among healthy veterans and those with PTSD. Overall, the results suggest that PTG is neurally mediated and that neural modulation to PTG is most prominent in healthy veterans and centers around the mPFC. The present study complements previous research highlighting the role of neural adaptation in healthy functioning. Future research aimed at clarifying the neural underpinnings of PTG will be beneficial in terms of refining the construct, potentially serving as a marker of PTG over time, and addressing some of the conceptual issues raised by previous researchers.

Acknowledgments This work was presented at the 29th annual ISTSS conference, Philadelphia, PA, November 7–9, 2013. This work was partially supported by a service-directed grant from the United States Department of Veterans Affairs. We thank Nancy Tabaka, Kari Johnson, Emily Van Kampen, Ann Marie Winskowski, Ryan Miller, Alina Shub, and Alex Alcorn for their help in participant recruitment, consent, and data entry.

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