

Aberrant EEG functional connectivity and EEG power spectra in resting state post-traumatic stress disorder: A sLORETA study



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ABSTRACT

The aim of the present study was to explore the modifications of EEG power spectra and EEG connectivity of resting state (RS) condition in patients with post-traumatic stress disorder (PTSD). Seventeen patients and seventeen healthy subjects matched for age and gender were enrolled. EEG was recorded during 5 min of RS. EEG analysis was conducted by means of the standardized Low Resolution Electric Tomography software (sLORETA). In power spectra analysis PTSD patients showed a widespread increase of theta activity (4.5–7.5 Hz) in parietal lobes (Brodmann Area, BA 7, 4, 5, 40) and in frontal lobes (BA 6). In the connectivity analysis PTSD patients also showed increase of alpha connectivity (8–12.5 Hz) between the cortical areas explored by Pz-P4 electrode. Our results could reflect the alteration of memory systems and emotional processing consistently altered in PTSD patients.

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1. Introduction

Post-traumatic stress disorder (PTSD) is a severe and disabling psychiatric condition characterized by three cluster symptoms which may develop after the exposure to one or more traumatic events: persistent trauma re-living, cognitive and behavioural avoidance and hyperarousal symptoms (APA, 2000; Klimesch, Sauseng, & Hanslmayr, 2007).

The neurobiology of PTSD is characterized by alterations concerning different brain areas (e.g. limbic system, prefrontal cortex) and different neurotransmitter systems (e.g. catecholamine) (for a review see Pitman et al., 2012). Furthermore, recent studies (Bluhm et al., 2009; Cook, Ciorciari, Varker, & Devilly, 2009; Lanius et al., 2010; Lee, Yoon, Kim, Jin, & Chung, 2014; Sripada, King, Garfinkel, et al., 2012; Sripada, King, Welsh, et al., 2012; Yin et al., 2011) documented in PTSD patients and in subjects with a history of trauma alterations in the functional integration between brain areas, a neurophysiological index called “functional connectivity”. This measure refers to the temporal synchrony or association between

signals of two or more spatially separated regions (Fingelkurts & Kahkonen, 2005; Schoffelen & Gross, 2009).

Using fMRI, Sripada and coworkers reported an increase of functional connectivity in the amygdala (Sripada, King, Garfinkel, et al., 2012a) and the hippocampus (Sripada, King, Welsh, et al., 2012b) of PTSD patients; this could reflect the dominance of threat-sensitive circuitry in PTSD, even in resting-state conditions (Sripada, King, Welsh, et al., 2012b). Abnormal functional connectivity in hippocampal network was also observed by Chen and Etkin (2013) who detected this alteration in PTSD patients when compared to Generalized Anxiety Disorder individuals.

Although fMRI is widely used to investigate brain functional connectivity, EEG is suitable to assess instantaneous and lagged synchronization across a wider frequency range, because EEG time-series data directly relate to dynamic postsynaptic activity in the cerebral cortex with a higher temporal resolution (Canuet et al., 2011). Conversely, MR-based methods cannot assess fast-frequency synchronized neuronal activity (Razavi et al., 2013). Finally, the EEG offers a potentially valuable “source of information for researchers and clinicians, since it assesses real-time electrical activity in the brain and is overall a less costly, time-consuming, and complex procedure” (Todder et al., 2012, p. 49). On the other hand, EEG suffers the problem of volume conduction or common sources, which gives rise to spurious correlations between time series recorded from nearby

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electrode (Stam, Nolte, & Daffertshofer, 2007). This problem can be solved by using specific algorithms for the identification of signal sources and for the evaluation of functional connectivity (Pascual-Marqui, Michel, & Lehmann, 1994).

Up to now, only few studies have investigated functional connectivity in PTSD using EEG. Cook et al. (2009) reported that, when compared to control subjects, adults with childhood trauma had significantly higher EEG coherence in the alpha and beta frequency bands over the left temporo-parietal areas and in the right central and temporal areas, respectively. Sponheim et al. (2011), in a sample of soldiers with mild traumatic brain injury, detected that PTSD patients had higher frontal functional connectivity, especially in low frequency bands (delta, theta, alpha, beta 1), than those without PTSD.

Moreover, in a recent network analysis study, Lee et al. (2014) observed that, compared to control subjects, PTSD patients had lower strength and efficacy connections between frontal and central areas, particularly in beta and gamma frequency bands.

As concerns EEG power spectra in PTSD few studies are available. Begic, Hotujac, and Jokic-Begic (2001) reported that un-medicated PTSD, compared to healthy subjects, showed an increase of theta power over the central brain regions, and an increase of beta activity over the frontal, central, and occipital brain regions. The increase of beta power was also replicated by the same authors (Jokic-Begic & Begic, 2003) comparing veterans with PTSD and veterans without PTSD. Todder et al. (2012) also observed that compared to control subjects, PTSD individuals have lower activity on the “low” theta band (4–5 Hz), mainly over the right temporal lobe and on the “high” theta band (6–7 Hz), over both the right and left frontal lobes.

The aim of the present study was to explore the modifications of EEG functional connectivity and scalp EEG power spectra in PTSD patients during resting state (RS) condition. The integration of these parameters contributes to the understanding of EEG correlates of mental disorders (Ford, Goethe, & Dekker, 1986). In order to detect modifications of EEG frequencies, especially their topographic distribution, we used the standardized Low Resolution brain Electric Tomography software (sLORETA), a validated method for localizing the electric activity in the brain based on multichannel surface EEG recordings (Pascual-Marqui et al., 1994). sLORETA has the benefit of superior time resolution of EEG measurements of milliseconds, which is 3-fold better than that of fMRI, with spatial resolution of approximately 7 mm, which is similar to that of fMRI (Grech et al., 2008; Stern et al., 2009). Furthermore, sLORETA benefits from an excellent localization agreement with different multimodal imaging technique (Dumpele, Ball, & Schulze-Bonhage, 2012; Pizzagalli et al., 2004; Vitacco, Brandeis, Pascual-Marqui, & Martin, 2002), also when standard 19-electrodes EEG montage was used (Cannon, Kerson, & Hampshire, 2011; Ridder, Vanneste, Kovacs, Sunaert, & Dom, 2011; Horacek et al., 2007; Muller et al., 2005). Finally, comparing with similar technique (i.e. LORETA), sLORETA gives the best performance both in terms of localization error and ghost sources (Grech, et al., 2008).

2. Materials and methods

2.1. Participants

Seventeen un-medicated patients with PTSD (seven men and ten women, aged 22–57 years mean age: 38.12 ± 10.42) who referred to a specialized trauma centre for treatment of trauma-related psychological disorders were enrolled. All patients received a complete psychiatric interview performed by a trained psychiatrist (BF), and were diagnosed according to the DSM-IV TR criteria (APA, 2000). No psychiatric comorbidities with Axis I and II DSM-IV disorders were observed. In order to exclude any neurological complications all participants also received a complete neurological examination performed by a trained neurologist (GDM). When head injury was suspected a neuroradiological evaluation (brain CT or MRI) was performed.

Table 1
Demographic data and the traumatic events of PTSD patients.

	Age	Sex	Traumatic event
1	55	M	Gun aggression
2	22	M	Family murdered
3	24	F	Rape
4	36	M	Car accident
5	42	F	Car accident
6	28	F	Rape
7	29	M	Fire victim
8	48	F	Physical aggression
9	33	F	Rape
10	42	F	Car accident
11	47	F	Gun aggression
12	25	M	Fire victim
13	43	M	Gun aggression
14	38	F	Rape
15	42	F	Physical aggression
16	37	M	Car accident
16	57	F	Physical aggression

Note: PTSD, post-traumatic stress disorder.

The demographic data and the traumatic events of PTSD patients enrolled in the study are listed in Table 1. A control group of healthy subjects (with no Axis I and II DSM-IV diagnosis) matched for age and gender was also included (seven men and ten women, aged 20–58 years mean age: 37.94 ± 10.32). Exclusion criteria were: left handedness; history of medical, neurologic diseases; psychiatric comorbidity; head trauma; assumption of Central Nervous System active drugs in the 3 weeks before the study; presence of EEG abnormalities at the baseline recording.

The research was approved by a human experimentation ethics committee. After receiving information about the aims of the study, all participants gave their written consent.

2.2. EEG recordings

RS recordings were performed in an EEG Lab, with each subject sitting in a comfortable armchair, with his/her eyes closed, in a quiet, semi-darkened silent room for 5 min.

EEG was recorded by means of a Micromed System Plus digital EEGraph (Micromed® S.p.A., Mogliano Veneto, TV, Italy). EEG montage included 19 standard scalp leads positioned according to the 10–20 system (recording sites: Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, O2), EOG and EKG. The reference electrodes were placed on the linked mastoids. Impedances were kept below $5\text{ k}\Omega$ before starting the recording and checked again at the end of the experimental recording. In particular, impedances of the mastoids reference electrodes were checked to be identical. Sampling frequency was 256 Hz; A/D conversion was made at 16 bit; pre-amplifiers amplitude range was $\pm 3200\ \mu\text{V}$ and low-frequency pre-filters were set at 0.15 Hz.

Artefact rejection (eye movements, blinks, muscular activations, or movement artefacts) was performed on the raw EEG trace, by positioning a marker at the onset of the artefact signal and a further marker at the end of the artefact. Successively, the artefact segment (that is, the EEG signal interval included between the two markers) was deleted. In this way, all the EEG intervals characterized by the presence of artefacts were excluded from the analysis.

After artefact rejection, the remaining EEG intervals were exported into American Standard Code for Information Interchange (ASCII) files, and imported into the sLORETA software. At least 120 s of EEG artefact-free recording (not necessarily consecutive) were analyzed for each subject. The average time analyzed was $193 \pm 21\text{ s}$ and $198 \pm 18\text{ s}$ respectively for PTSD and control group. All EEG analysis was performed by means of the sLORETA software (Pascual-Marqui et al., 1994).

2.3. Frequency analysis

EEG frequency analysis was performed by means of a Fast Fourier Transform algorithm, with a 2 s interval on the EEG signal, in all scalp locations. The following frequency bands were considered: delta (0.5–4 Hz); theta (4.5–7.5 Hz); alpha (8–12.5 Hz); beta (13–30 Hz); gamma (30.5–60 Hz). For frequency analysis, monopolar EEG traces (each electrode referred to joint mastoids) were used. Topographic sources of EEG activities were determined using the sLORETA software. The sLORETA software computes the current distribution throughout the brain volume. In order to find a solution for the three-dimensional distribution of the EEG signal, the sLORETA method assumes that neighbouring neurons are simultaneously and synchronously activated. This assumption rests on evidence from single cell recordings in the brain that shows strong synchronization of adjacent neurons (Kreiter & Singer, 1992; Murphy, Blatter, Wier, & Baraban, 1992). The computational task is to select the smoothest three-dimensional current distribution, a common procedure in signal processing (Grave de Peralta-Menendez and Gonzalez-Andino, 1998; Grave de Peralta Menendez, Gonzalez Andino, Morand, Michel, & Landis, 2000). The

Table 2
Cortical 19 regions of interest (ROIs).

Scalp electrodes	ROI centroid MNI coordinates			Brodmann areas
	x	y	z	
Fp1	-25	65	-5	10, 11
Fp2	25	65	-5	10, 11
F7	-50	40	-10	45, 47, 38
F3	-45	40	30	9, 46
Fz	5	45	50	8, 9
F4	45	40	30	9, 46
F8	50	40	-10	45, 47, 38
T3	-65	-15	-15	21, 22, 42
C3	-50	-20	60	1, 2, 3, 4, 6
Cz	5	-10	70	4, 6
C4	55	-20	55	1, 2, 3, 4, 6
T4	70	-20	-10	21, 22, 42
T5	60	-65	-5	19, 22, 37
P3	40	-70	45	7, 39, 40
Pz	-5	-65	65	7
P4	45	-70	45	7, 39, 40
T6	55	-70	0	19, 22, 37
O1	-20	-100	10	17, 18, 19
O2	20	-100	5	17, 18, 19

Adapted from Canuet et al. (2012).

Note. ROI, region of interest.

result is a true three-dimensional tomography, in which the localization of brain signals is preserved with a low amount of dispersion (Pascual-Marqui et al., 1994).

2.4. Connectivity analysis

The connectivity analysis was performed by the computation of lagged phase synchronization. This connectivity measure is a much more appropriate measure of electrophysiological connectivity, because it decomposes connectivity into instantaneous and lagged components (Canuet et al., 2011; Pascual-Marqui et al., 2011). Furthermore, the lagged phase synchronization index is resistant to non-physiological artefacts, in particular volume conduction and low spatial resolution that usually affect other connectivity indices (Stam et al., 2007). Moreover, the lagged component is purely physiological, and affected minimally by low spatial resolution, which affects the instantaneous component (Pascual-Marqui et al., 2011). For this reason this approach allows to evaluate 'true' connectivity and it has been widely used in clinical physiology (Canuet et al., 2011, 2012; Pagani et al., 2012).

Lagged phase synchronization index measures the similarity (a corrected phase synchrony value) between signals in the frequency domain based on normalized (unit module) Fourier transforms; thus it is related to nonlinear functional connectivity (Canuet et al., 2011). This lagged connectivity measure is thought to be accurately corrected as it represents the connectivity between two signals after the instantaneous zero-lag contribution (artefactual component) has been excluded. Such a correction is necessary when using scalp EEG signals or estimated intracranial signals (EEG tomography) because zero-lag connectivity in a given frequency band is often due to non-physiological effects or intrinsic physics artefacts, in particular volume conduction and low spatial resolution that usually affect other connectivity indices (Stam et al., 2007).

The sLORETA software computes lagged phase synchronization $\rho_{x,y}(\omega)$, by the formula (Pasdaran-Marqui, 2007):

$$\varphi_{x,y}^2(\omega) = \frac{\{\text{Im}[f_{x,y}(\omega)]\}^2}{1 - \{\text{Re}[f_{x,y}(\omega)]\}^2}$$

In this formula " ω " is the discrete frequency considered, " x " and " y " are the EEG sources, "Re" and "Im" indicates the real and the imaginary parts of a complex element; $x(\omega)$ and $y(\omega)$ denote the discrete Fourier transforms of the two signals of interest x and y at frequency " ω ". The general lagged phase synchronization is defined as the partial coherence between the normalized complex-valued stochastic variables (x_ω, y_ω) with the zero-lag effect removed (Pasdaran-Marqui, 2007). Details on sLORETA connectivity algorithm can be found in Pascual-Marqui's studies (2007; 2011).

In order to evaluate the connectivity, 19 Regions of Interest (ROIs) were defined corresponding to the site of the electrode (one for each scalp electrode). We chose the 'single nearest voxel' option: in this way, each ROI consisted of a single voxel, the closest to each seed. The 19 cortical ROIs determined by sLORETA are listed in Table 2.

The sLORETA computed the lagged phase synchronization values between all these ROIs (total $19 \times 19 = 361$ connections). The sLORETA also computed the source reconstruction algorithm previously described (Pascual-Marqui & Biscay-Lirio, 1993; Pascual-Marqui et al., 1994; Pascual-Marqui, Michel, & Lehmann, 1995).

The EEG connectivity analysis was performed on the same blocks of EEG tracings used for power spectra analysis.

2.5. Statistical analysis

Power spectra analysis and EEG connectivity were compared between PTSD patients and control groups for each frequency band. Comparisons were performed using the statistical non-parametric mapping (SnPM) methodology supplied by the sLORETA software (Nichols & Holmes, 2002). This methodology is based on the Fisher's permutation test: a subset of non-parametric statistics. In particular, this is a type of statistical significance test in which the distribution of the test statistic under the null hypothesis is obtained by calculating all possible values of the test statistic under rearrangements of the labels on the observed data points. Correction of significance for multiple testing was computed for the two comparisons between patients and control group for each frequency band: for the correction, we applied the non-parametric randomization procedure available in the sLORETA program package (Nichols & Holmes, 2002).

T-Level thresholds were computed by the statistical software implemented in the sLORETA, which correspond to statistically significant thresholds ($p < 0.05$ and $p < 0.01$) (Friston, Frith, Liddle, & Frackowiak, 1991).

3. Results

EEG recordings suitable for the analysis were obtained for all participants. Visual evaluation of the EEG recordings showed no relevant modifications of the background rhythm frequency, focal abnormalities or epileptic discharges. No subject showed evidence of drowsiness or sleep during the recordings.

3.1. Power spectra analysis

In this analysis the thresholds for significance were $T = 3.801$ corresponding to $p < 0.05$, and $T = 4.324$, corresponding to $p < 0.01$. Significant modifications were observed in the theta band: PTSD patients showed a widespread increase of theta activity in the parietal lobes and a more limited increase in the frontal lobes (Fig. 1). sLORETA software localized these modifications in bilateral precuneus (Brodmann Area, BA 7; $T = 4.61$, $p < 0.01$), in bilateral postcentral gyrus (BA 4,5; $T = 4.33$, $p < 0.01$), in bilateral inferior parietal lobe (BA 40; $T = 4.01$, $p < 0.05$) and in bilateral middle frontal gyrus (BA 6; $T = 4.03$, $p < 0.05$). No significant differences were observed in the other frequency bands.

3.2. Connectivity analysis

In this analysis the thresholds for significance were $T = 4.085$ corresponding to $p < 0.05$, and $T = 4.781$, corresponding to $p < 0.01$.

In PTSD patients a significant modification was observed in the alpha band (Fig. 2). This modification consisted in an increase of lagged phase synchronization between the cortical areas explored by Pz and P4 electrode ($T = 4.27$, $p < 0.05$). No significant differences were observed in the other frequency bands for other electrode pairs.

4. Discussion

The principal aim of the present study was to explore the modifications of EEG functional connectivity and scalp EEG power spectra in PTSD patients during resting state condition. Compared to healthy subjects, individuals with PTSD showed an increase of EEG connectivity between precuneus and right inferior parietal lobe (ROIs explored by Pz and P4 electrodes), in the alpha frequency band. Moreover, we detected a widespread increase of EEG theta power in parietal lobes (BA 7, 4, 5, 40) and, to a lesser extent, in the frontal lobes (BA 6).

Our results are partially consistent with previous EEG and functional imaging studies confirming the important role of parietal lobes (especially precuneus) in PTSD. The increase of theta power in parietal and frontal areas was also previously observed by Begic

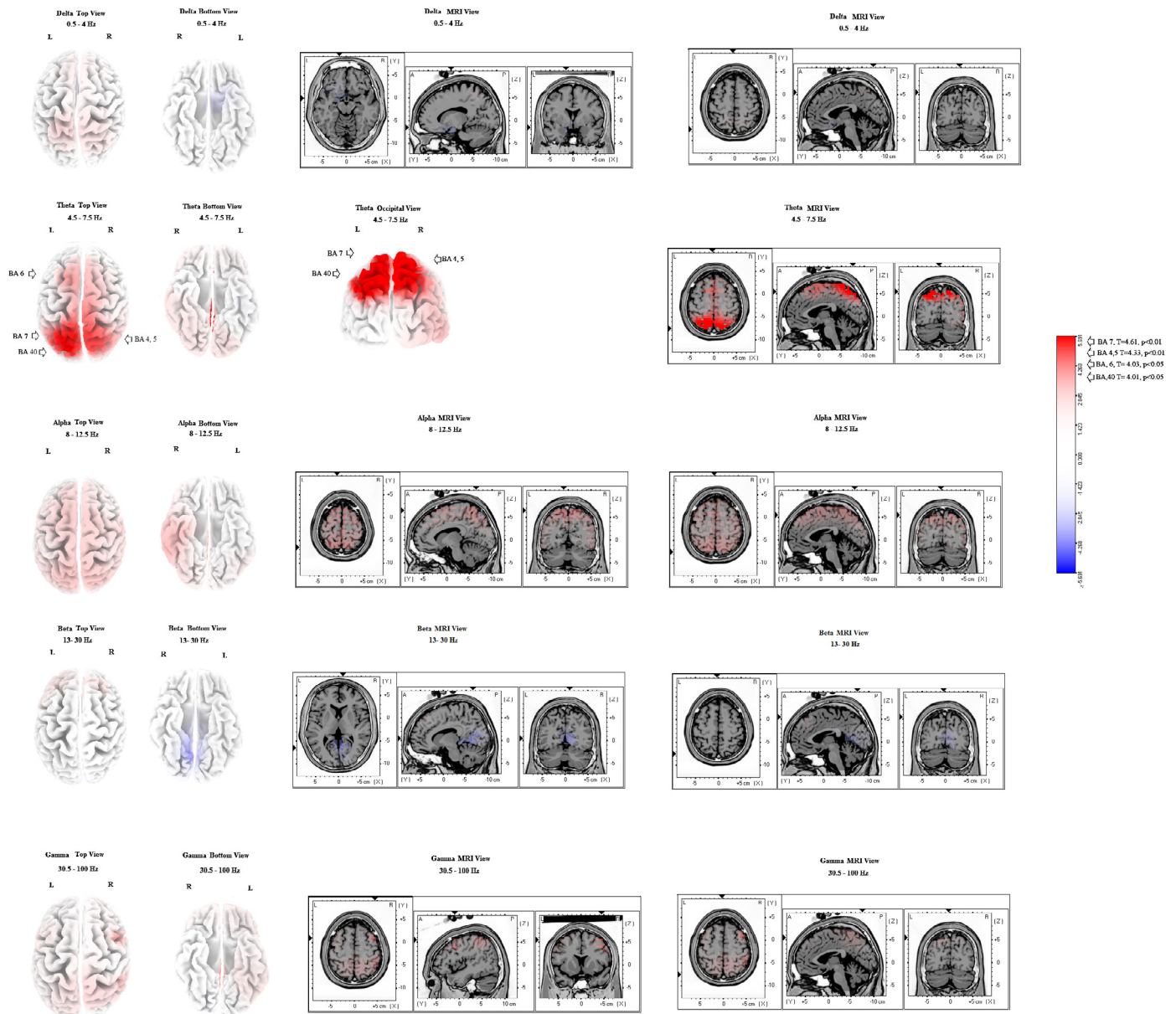


Fig. 1. Results of the sLORETA comparison of EEG power spectra in each frequency band. Coloured spots indicate areas where statistically significant increase of delta EEG spectral power were detected. Threshold values (T) for statistical significance (corresponding to $p < 0.05$ and $p < 0.01$) are reported in the right side of the figure. Red colour indicates significant increase of power spectra. Blue colour indicates reduction of power spectra. PTSD patients showed a widespread increase of theta activity in parietal lobes and lower in frontal lobes. BA = Brodmann areas; L = left; R = right. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article.)

et al. (2001) but not by Todder et al. (2012) who detected a reduction of theta power in PTSD patients. As already suggested (Todder et al., 2012) this difference could reflect the action of psychotropic drug treatment. Indeed, several studies consistently reported that both serotonin reuptake inhibitors and benzodiazepines could affect theta rhythm (Bares et al., 2008, 2010; Kopp, Rudolph, Low, & Tobler, 2004; Yoshimoto et al., 1999). In contrast with Todder et al. (2012) and consistently with Begic et al. (2001), our patients were not receiving any drug treatment at the moment of EEG recording.

In the same way the increase of alpha connectivity was also observed in previous studies (Cook et al., 2009; Sponheim et al., 2011). Cook et al. (2009) reported that, when compared to control subjects, adults with childhood trauma had significantly higher EEG coherence of the alpha in temporo-parietal areas. Similarly, Sponheim et al. (2011) observed in soldiers with PTSD higher

frontal functional connectivity than in those without PTSD in low frequency bands. Furthermore, Metzger et al. (2004) reported abnormal alpha activity in right-sided parietal areas which was also associated with PTSD symptoms.

Abnormal theta power and alpha connectivity in parietal areas observed in our study could reflect the alterations of emotional processing and memory systems, core features of PTSD patients (Brown & Morey, 2012; Hayes, Vanelzakker, & Shin, 2012). Several studies reported that PTSD patients remember emotional stimuli better than neutral stimuli in comparison to healthy controls (Chemtob et al., 1999; Paunovi, Lundh, & Ost, 2002) suggesting a difficulty in disengaging from threat-related information (Chemtob et al., 1999). Furthermore, it is also observed that greater difficulty in emotions regulation is associated with greater PTSD symptom severity (Bonn-Miller, Vujanovic, Boden, & Gross, 2011; Tull, Barrett, McMillan, & Roemer, 2007). Regarding alteration in

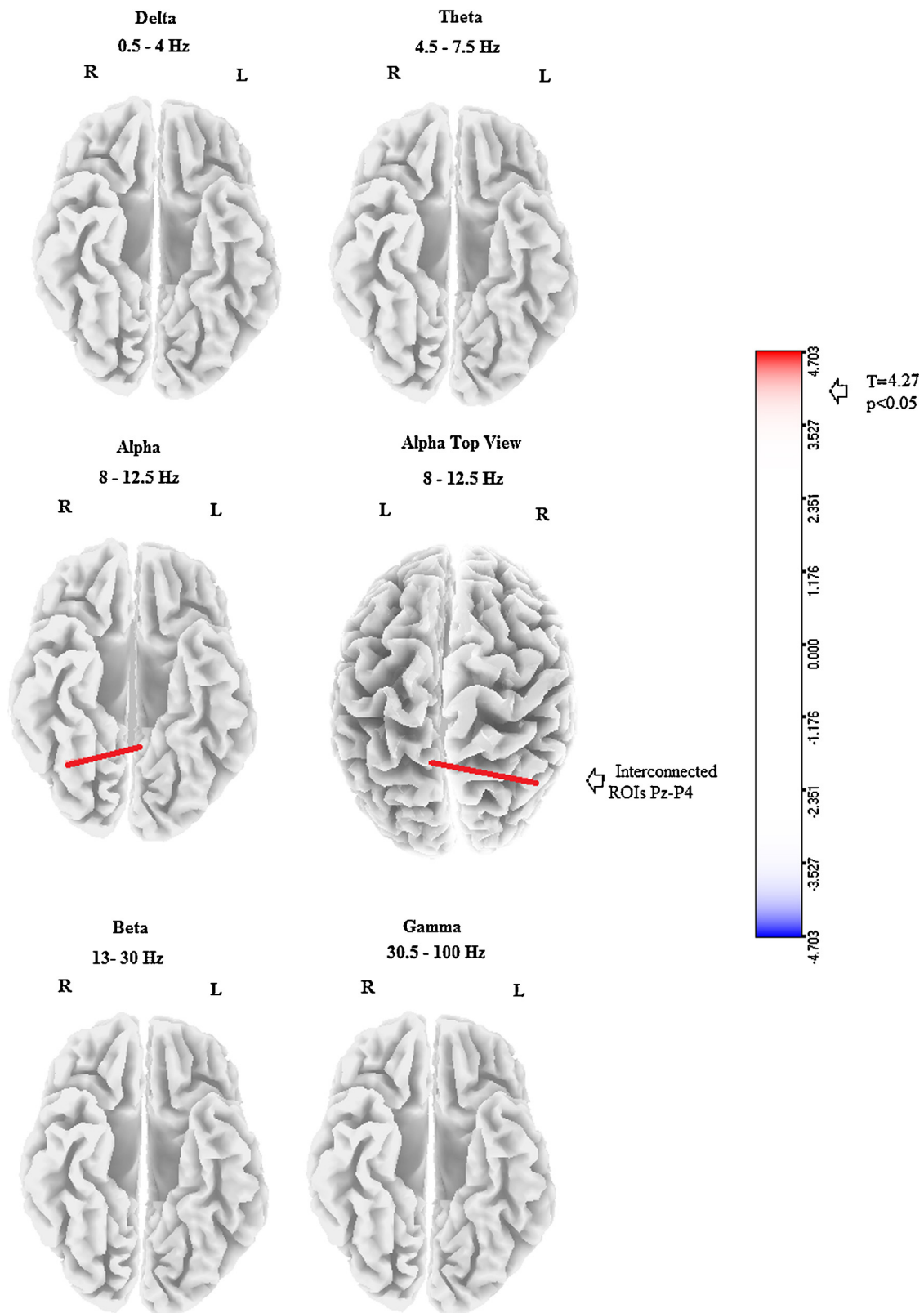


Fig. 2. Results of the sLORETA comparison of EEG lagged phase synchronization in each frequency bands. Threshold values (T) for statistical significance (corresponding to $p < 0.05$) are reported in the right side of the figure. Red lines indicate connections which presented increase of coherence. Blue lines (not present) would indicate reduction of coherence. In the connectivity analysis PTSD patients showed increase of alpha connectivity between the cortical areas explored by Pz and P4 electrode. ROIs = Region of Interests; R = right; L = left. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article.)

memory systems, it has been documented that interference stimuli, such as intrusive thoughts (Schweizer & Dalgleish, 2011) could lead to working memory deficits in patients with PTSD.

It is also known that theta and alpha oscillations play an important role in different memory processes (Jensen, Gelfand, Kounios, & Lisman, 2002; Jensen & Tesche, 2002; Khader, Jost, Ranganath, & Rosler, 2010; Schack & Klimesch, 2002) and in emotion regulation (Aftanas, Pavlov, Reva, & Varlamov, 2003; Knyazev, 2007). Aftanas et al. (2003) reported that, compared to low trait-anxious subjects, high trait subjects exhibited an increase of event-related synchronization in theta power while viewing visual threatening stimuli. This pattern reflects the general bias towards threatening information in anxious patients. Other studies observed an increase in alpha and theta power associated with working memory load (Jensen et al., 2002; Jensen & Tesche, 2002; Schack & Klimesch, 2002) and with long-term-memory encoding (Khader et al., 2010).

Therefore, it is possible to speculate that the abnormal neurophysiological pattern observed in our study reflects several aspects of PTSD psychopathology, such as the high persistent intrusive symptoms (Brewin, Gregory, Lipton, & Burgess, 2010), the difficulty in emotions regulation (Bonn-Miller et al., 2011; Tull et al., 2007), the fragmentation of traumatic memories (Bergmann, 2008; Brewin, 2001), working memory deficits (Schweizer & Dalgleish, 2011) and the generally reduced specificity of autobiographical memories recall (Moradi, Abdi, Fathi-Ashtiani, Dalgleish, & Jobson, 2012; Moradi et al., 2008).

Coherently with our results, some fMRI studies (Bluhm et al., 2009; Lanius et al., 2010; Yin et al., 2011) demonstrated abnormal increase of precuneus functional connectivity, that is also positively correlated with PTSD symptomatology (Bluhm et al., 2009; Lanius et al., 2010). During trauma-related stimuli an increased neuronal activity was reported in parietal cortices (Lanius et al., 2002; Pagani et al., 2012; Piefke et al., 2007) of patients with PTSD. The parietal cortex has been suggested to play an important role in different memory systems such as episodic memory (Cabeza, Ciaramelli, Olson, & Moscovitch, 2008; Wagner, Shannon, Kahn, & Buckner, 2005), and visuospatial working memory retrieval (Berryhill & Olson, 2008). Moreover, precuneus seems to be a crucial structure involved in episodic memory retrieval (Cavanna & Trimble, 2006) and in visual imagery during autobiographical memory recall (Addis, Moscovitch, Crawley, & McAndrews, 2004; Gardini, Cornoldi, De Beni, & Venneri, 2006).

Moreover, it is also interesting to underline that we observed an increase of alpha connectivity in PTSD patients during RS. Recently it was proposed that alpha frequency neural activity reflects an inhibitory mechanism in order to facilitate information processing during cognitive tasks (for a review see Klimesch et al., 2007). For example Klimesch et al. (2007) suggested that the increase of alpha power and alpha coherence associated with memory load during cognitive tasks reflects the attempt “to inhibit interfering memories of the preceding trial” (p. 66). Therefore, it is possible to hypothesize that the aberrant increase of alpha connectivity in PTSD patients during RS could be the outcome of a defensive attempt to inhibit the intrusive traumatic memories. Indeed, it seems that PTSD patients attempt to inhibit interfering information such as traumatic memories and other intrusive trauma related stimuli (Clohessy & Ehlers, 1999; Steil & Ehlers, 2000), also in task free conditions.

However, it must be underlined that this interpretation remains largely speculative due to the absence in our study of memory tasks and psychometric symptomatology assessment. Even though to the best of our knowledge this is the first study which simultaneously investigated EEG functional connectivity and EEG power spectra using an accurate and validated tool to localize electric activity in the brain such as sLORETA.

4.1. Study limitations

The present study has several limitations. One limitation is the small sample size that must be considered when drawing a conclusion. Furthermore, we used scalp EEG recordings, which have an intrinsic limit in space resolution, particularly in the identification of deep subcortical sources. Another limitation is the absence of assessment of depressive and anxiety symptoms severity. Finally, it is important to note that PTSD is a heterogeneous disorder, and the nature of the traumatic event may be affecting symptoms severity (Lancaster, Melka, & Rodriguez, 2009) as well as neurophysiological results (Todder et al., 2012).

Conflict of interest

The authors have no conflicts of interest

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