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RESEARCH ARTICLE

Long range temporal correlations in EEG oscillations of subclinically depressed individuals: their association with brooding and suppression

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Abstract Long-range temporal correlations (LRTC) in brain oscillations have been found to be associated with depression severity in clinically depressed patients. Less is known, however, about the relationships between LRTC and proneness to engage in depression-related cognitive emotion regulation (ER) strategies which characterize both clinically and subclinically depressed (SBD) people. In this study we applied detrended fluctuation analysis to the amplitude envelope of broad band, theta band, and alpha band spontaneous EEG oscillations of a group of SBD individuals and a group of non-depressed individuals (both groups from a sample of healthy adults, N = 120), to whom brooding and thought suppression questionnaires were administered. Between-groups differences were not found for any band scaling exponents at any brain location, but linear correlations pointed out several associations between exponents at frontal, central, parietal, temporal, and occipital sites and maladaptive ER strategies. These results suggest that alterations in brain dynamics are related with the proneness that depressive individuals show to engage in brooding and thought suppression in order to cognitively regulate their emotions.

Keywords Long-range temporal correlations (LRTC) · EEG · Brooding · Thought suppression · Emotion regulation

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Introduction

The emergence of long-range temporal correlations (LRTC) and power-law scaling in complex systems like the brain can be explained by the theory of self-organized criticality (Bak et al. 1987) and suggests that the brain operates near a critical state (Chialvo 2010; Werner 2010; Palva et al. 2013; Pittman-Polletta et al. 2013). Quick reorganization of neural networks during processing demands would be possible by the critical state dynamics of spontaneous oscillations (Linkenkaer-Hansen et al. 2001), thus scaling phenomena would be an essential characteristic of complex, adaptive, healthy systems. In fact, scaling in resting-state EEG has been suggested as a biomarker of pathophysiology in neurodevelopmental disorders (Smit et al. 2011).

Long-range temporal correlations in electroencephalographic oscillations have received increasing attention during the last decade. After their demonstration by Linkenkaer-Hansen et al. (2001), and in parallel with studies regarding their reliability (Nikulin and Brismar 2004), distribution over the scalp (Nikulin and Brismar 2005), genetic contributions to LRTC (Linkenkaer-Hansen et al. 2007), and their existence across a wide age range (Berthouze et al. 2010), several studies focused on the associations between LRTC and depression (Lee et al. 2007; Leistedt et al. 2007; Linkenkaer-Hansen et al. 2005). The results of these studies confirmed the existence of such associations, although the different methods and procedures used to calculate the scaling exponents make comparisons difficult. Lee et al. (2007) used EEG recordings taken during 5 min resting periods with eyes closed that were then segmented into 30-s epochs. They calculated the exponents of the broad band (.6–46 Hz) amplitude oscillations in each epoch on time scales ranging from 25

samples to 145 samples (.1–.6 s). The scaling exponents of depressed patients had relatively higher values in whole brain regions compared to healthy controls, and a significant positive linear correlation was observed between the severity of depression and the scaling exponent over most of the channels. Linkenkaer-Hansen et al. (2005) used EEG recordings taken during 16 min resting periods with eyes closed. Scaling exponents of the theta (3–7 Hz) band amplitude envelope, computed from the modulus of the Hilbert transform on time scales ranging from 5 to 100 s., were larger in control participants than in depressed patients. A significant negative linear correlation was observed between the severity of depression and the scaling exponent of theta oscillations detected over the left temporocentral region. The different sign of the linear correlations (positive versus negative) reported in these studies could be due to the wide range of windows of time during which the scaling exponents were calculated. Linkenkaer-Hansen et al. (2005) reported also a small, albeit significant positive correlation between the scaling exponents of alpha (8–13 Hz) occipitoparietal oscillations and the depression scores.

Patients participating in these studies were classified as depressed based on both depression questionnaires scores (Beck Depression Inventory or Hamilton Depression Rating Scale) and clinical interview, such that all of them were diagnosed as suffering from major depressive disorder. Severely depressed and subclinically depressed (SBD) people share several cognitive impairments and neural dysfunctions as well (Brzezicka 2013), though the latter perform quite normally on some cognitive tasks, therefore differences between more or less depressed individuals do exist. One question that remains open is whether changes in the EEG LRTC are associated with the cognitive emotion regulation (ER) strategies commonly used by people with a depressive ER style even if they cannot be classified as clinically depressed. Addressing this question would be important from a preventive point of view because rumination—especially its brooding facet—has been prospectively associated with the onset of depressive episodes (Nolen-Hoeksema et al. 1993), the maintenance, severity and duration of depressive episodes (Nolen-Hoeksema 2000), heightened psychological distress when facing everyday stressors (Moberly and Watkins 2008), and the mediation of the relationship between high negative affect and depressive symptoms (Verstraeten et al. 2009). On the other hand, thought suppression, and more generally the use of experiential avoidance, has also been associated with depressive symptoms and emotional disorders (Barlow et al. 2004; Hayes et al. 1996; Shahar and Herr 2011). Furthermore, subclinical depression often precedes clinical depression (Brzezicka 2013).

To date the only study addressing that still-open question was carried out by Bornas et al. (2013). In a sample of undergraduate students (N = 56) two correlational analyses were performed, one focusing on the theta (3–7 Hz) band oscillations and the other one taking the broad (1–40 Hz) band oscillations. Significant correlations were found between brooding and parietal and occipital theta band scaling exponents, and between suppression and central, parietal, and occipital theta band scaling exponents. Suppression scores significantly correlated with broad band scaling exponents in central regions as well. All correlations were positive, i.e. people more prone to engage in brooding and suppression had higher scaling exponents (more slowly decaying LRTC with time) thus being consistent with the results reported by Lee et al. (2007) who calculated the scaling exponents in time windows of a similar length (seconds). Linkenkaer-Hansen et al. (2005), however, found negative linear correlations between depression scores and theta band scaling exponents. Bornas et al. (2013) argue that temporal correlations over short windows might correlate with ER strategies (cognitive processes) while LRTC over minutes might reflect depressive states, hence the opposite sign of the reported correlations would not be so surprising. This study had, nevertheless, a number of limitations: scaling exponents were calculated on a limited number of EEG channels, and the spectrum of ER strategies evaluated was very wide (including both positive and negative strategies). Most importantly, participants were undergraduate students and no screening measures were used to identify individuals that might be showing mild depressive symptoms.

The present study aimed to further investigate the associations between resting EEG LRTC and brooding and suppression ER strategies in SBD and non-depressed (ND) individuals. In addition we studied the associations between both broad band and narrow band (theta and alpha) LRTC and depression severity scores in the SBD group. The first hypothesis was that SBD individuals are more prone to engage in brooding and suppression strategies than ND participants. The second hypothesis, based on previous results from studies using windows of similar length (Lee et al. 2007; Bornas et al. 2013), was that positive linear correlations should be found between broad band scaling exponents and depressive ER strategies as well as between theta band exponents and these strategies, and these correlations should be stronger for SBD individuals than for ND participants. Our third hypothesis was twofold: (a) broad band LRTC should be positively correlated with depression severity (based on results reported by Lee et el. 2007) whilst (b) according to the findings reported in Linkenkaer-Hansen et al. (2005) theta band LRTC should be negatively correlated with depression severity, and alpha band LRTC should be positively

correlated with depression severity scores in the SBD group. Partial correlations controlling for depression severity scores were calculated to find out whether LRTC were more associated with ER strategies or depressive states. The last hypothesis was based on the finding that depressed patients had significantly higher scaling exponents than ND healthy controls (Lee et al. 2007; although Hosseinifard et al. (2013) did not find such differences) and on the linear correlation between depression severity scores and scaling exponents: the more depressed the individual, the higher the exponents (using short time windows and broad frequency band, see Lee et al. 2007; or using long time windows and alpha frequency band; see Linkenkaer-Hansen et al. 2005) or the lower the exponents (if using long time windows and theta frequency band; see Linkenkaer-Hansen et al. 2005). In any case the correlations are *linear*, so these changes (increases or decreases) in the exponents are associated with proportional changes in depression severity scores. As our sample was subclinical and hence the severity of depression was lower than in a clinical sample (and higher than in a ND sample) we hypothesized that the exponents would be lower than those reported in a study with a clinical sample that used time windows of similar length (i.e. Lee et al. 2007), and therefore these exponents would be closer to the expected exponents for a ND sample. Differences between exponents from SBD and ND individuals were thus expected to be very small (either statistically significant or not).

Methods

Participants and screening procedure

One hundred and thirty-six healthy university students and staff members signed an informed consent form before enrolling in the study. All procedures were approved by the Bioethics Committee of the University. Participants were recruited via electronic or posted advertisements. All of them were first screened by means of the three-item version of the Patient Health Questionnaire-9 (see description below). Those scoring 4 or more on it were administered the Beck Depression Inventory II (BDI-II) and individually interviewed using the ADIS-IV (Brown et al. 1994). Participants receiving a current diagnostic of anxiety and/or depression were excluded. Additionally, all participants were asked about psychological/psychiatric problems in the past (*Have you ever asked for psychological/psychiatric treatment in the past, and when? Did you receive any formal diagnosis/treatment? If yes, which kind of diagnosis/treatment?*). The answers to these questions were not used to determine the inclusion in one of the two groups in the current study, but only to exclude participants if clinically depressive symptoms or other psychopathological conditions had been present during the last 12 months. Seven participants were excluded due to the presence of an anxiety or depressive disorder; eight left-handers were not analyzed in order to maintain consistency across the data and another one was excluded because of missing data in self-reported measures. The final sample, made up of 120 right-handed adults, was divided into two different groups to be analyzed separately. The SBD group consisted of 28 participants (22 women, Mage = 26.39 years; SD = 8.09; range 18–49; one with past history of major depressive disorder and one with past history of anorexia nervosa) with scores higher than 3 in the short form of the Patient Health Questionnaire-9, whereas 92 participants (67 women, Mage = 29.15 years; SD = 9.17; range 18–49; one with past history of panic disorder without agoraphobia, one with past history of generalized anxiety disorder, and four with a past history of major depressive disorder) with scores lower than 4 in this questionnaire made up the ND group.

Screening and self-reported measures

Patient Health Questionnaire (PHQ)

The three-item version (Craske et al. 2009) of the Patient Health Questionnaire-9 (Kroenke et al. 2001) was used to assign participants to the SBD group. Respondents were asked to rate each item (two depression and one fatigue items) on a 4-point Likert-type scale (1 = not at all; 4 = nearly every day). Those scoring 4 or more were administered the BDI-II and individually interviewed using the ADIS-IV (Brown et al. 1994). Participants receiving anxiety and/or depression diagnoses were excluded. In our sample, internal consistency was alpha = .76.

Beck Depression Inventory II (BDI-II)

This questionnaire (Beck et al. 1996) was used to assess the severity of depression in participants who had a score of 4 or more in the Patient Health Questionnaire-9. The questionnaire contains 21 items to be answered on a 4-point scale. Guidelines to interpret the BDI II scores are: minimal range = 0–13, mild depression = 14–19, moderate depression = 20–28, and severe depression = 29–63. In our sample, internal consistency was alpha = .87.

Ruminative Response Scale (RRS)

To assess rumination we used the shortened RRS (Treynor et al. 2003), consisting of 10 statements rated using a 4-point scale (1 = almost never; 4 = almost always) according to the frequency with which ruminative responses are

expressed when experiencing a dysphoric mood. The instrument contains two subscales: reflection and brooding. In our sample internal consistency was a = .82 for the whole scale and alpha = .79 and alpha = .77 for the brooding and reflection scales, respectively.

White Bear Suppression Inventory (WBSI)

Thought suppression was assessed by means of this inventory (Wegner and Zanakos 1994), a 15-item questionnaire, wherein respondents are asked to rate, using a 5-point scale (1 = strongly disagree; 5 = strongly agree), to which extent each of the statements fits their typical behaviors. In our sample internal consistency was alpha = .92.

EEG recordings

After a 3-min adaptation period, EEG activity was recorded during an 8-min resting baseline following the pattern of 2 min with eyes open (EO), 2 min with eyes closed (EC), 2 min with EO, and 2 min with EC. Therefore each EC condition was preceded by an EO condition. EEG measures were acquired using a 64-channel Brain Vision amplifier (Brain Products, Munich, Germany). Resting EEG activity was recorded using a Lycra stretch cap (Easycap) for F3, F4, C3, C4, P3, P4, O1, O2, T7, and T8 positions placed in accordance with the International 10/20 System and using mastoid electrodes as reference channels. Signals were filtered online with a bandpass filter (.05–40 Hz). A 50 Hz notch filter was also applied. Sampling rate was set at 500 Hz and electrode impedances were kept below 10 kΩ. An electrooculogram (EOG) channel was recorded using two electrodes placed 2 cm above and below the right eye, and ocular correction (Gratton et al. 1983) was applied online using a blink detection algorithm taking the EOG channel as reference. Segments containing residual muscle movements or other forms of artifacts greater than 100 μV were rejected automatically prior to further analysis. From each of the two 120 s (2 min) eyes-closed periods recorded we took the EEG signal from 30 to 90 s, that is, a signal of 60 s (30,000 data points) long. This central part should be the least contaminated by muscle or cognitive features from the preceding EO conditions. Recordings from the eyes-open conditions were not analyzed since none of the previous studies in the field found associations between the scaling exponents from resting with EO and depression scores or ER strategies.

Data analysis and statistics

The long-range correlations in the dynamics of the amplitude of broad band (1–40 Hz) and theta band (3–7 Hz) and alpha band (8–13 Hz) oscillations were evaluated using detrended fluctuation analysis (DFA, Peng et al. 1995). Firstly, a cumulative sum $y(k)$ of the detrended amplitude envelope is calculated for each eyes-closed segment. Secondly, the integrated signal is divided into non-overlapping windows with different lengths (see below) equidistantly distributed on a logarithmic scale. Thirdly, for each window size n, the least-squares fitted line is computed and the variance $F^2(n)$ is calculated in the detrended integrated signal by

$$F(n) = \sqrt{\frac{1}{N}\sum_{k=1}^{N}[y(k) - y_n(k)]^2}$$

where N is the total length and $y_n(k)$ is the y coordinate of the straight line segment in each window

The slope of the line relating log $F(n)$ and log n is the scaling exponent α. When the EEG is completely uncorrelated, the calculation of the scaling exponent yields .5. When applied to the EEG with LRTC, a scaling exponent $.5 < \alpha < 1$ indicates the data are correlated, such that large fluctuations are likely to be followed by large fluctuations and small fluctuations are likely to be followed by small fluctuations. The case of $\alpha = 1$ corresponds to *1/f* noise. As the scaling exponent increases from .5 toward 1, the LRTC in the EEG are more persistent (decaying more slowly with time).

As previous studies applied DFA over quite different time ranges and frequency bands, we analyzed LRTC using two separate methods. Following Lee et al. (2007) we calculated the DFA scaling exponents of the amplitude envelope of broad band (1–40 Hz) oscillations using 50 time scales that ranged from .1 to .6 s. With narrow bandpass filtered oscillations (theta, 3–7 Hz, and alpha, 8–13 Hz), however, we used a method similar to the one developed by Linkenkaer-Hansen et al. (2005) and applied DFA to their amplitude envelope using 50 time scales that ranged from 1 to 6 s. We could not use longer time windows because of the limited length of each epoch (60 s). As pointed out by Nikulin and Brismar (2005), a minimum of ten boxes is required to perform DFA. Scaling exponents were calculated for each EEG channel. Exponents of both eyes-closed conditions were then averaged.

Comparisons between groups, bivariate linear correlations, and partial correlations between self-reported measures and the scaling exponents for each EEG channel were performed using IBM SPSS Statistics 20 (IBM Corp., Germany, 2011). As the sizes of the SBD and ND groups were different the Levene test was used to confirm that the variances of both groups were equivalent. Further, analyses were performed after randomly selecting a number of individuals from the ND group to obtain the same size as the SBD group. Results did not change, and therefore we report only the results of the analyses performed on the original groups.

Table 1 Differences in depressive symptoms (assessed by PHQ), brooding and suppression between the subclinically depressed and the non-depressed groups

	SBD group		ND group		Levene's test F	t	p	95 % CI
	M	SD	M	SD				
Depressive symptoms	4.821	.945	1.913	1.055	1.211 (*ns*)	13.071	<.001	[2.467, 3.349]
Brooding	12.643	3.368	10.261	3.422	.023 (*ns*)	3.236	.002	[.924, 3.839]
Suppression	45.357	11.106	36.402	13.625	1.217 (*ns*)	3.169	.002	[3.359, 14.551]

SBD subclinically depressed, *ND* non-depressed

Results

Differences in depressive symptoms, brooding and suppression scores were highly significant (see Table 1) showing that SBD participants are more prone than ND individuals to engage in these depression-related ER strategies.

Correlational analysis revealed the significant associations depicted in Table 2. As to the broad band, scaling exponents from central areas (C3C4) were associated with suppression and severity of depression, but not significantly with brooding. Scaling exponents from parietal areas (P3P4) were significantly correlated with both brooding and suppression ER strategies and severity of depression (BDI-II scores). The results of the analysis performed on the theta band revealed that parietal (P3P4) scaling exponents were associated with both brooding and severity of depressive symptoms, although no significant associations were found for central (C3C4) areas. On the other hand, brooding and suppression were also negatively associated with scaling exponents from the temporal (T7T8) regions. As to the alpha oscillations, all correlations were negative. Brooding was associated with scaling exponents from frontal and central areas, suppression correlated with occipital exponents, and depression severity scores were associated with scaling exponents from frontal, central, temporal, and occipital regions. The strength and direction of the correlations for left and right sites were almost identical, i.e. asymmetric patterns could not be found in the correlational analysis for any band (for instance, $r_{LRTCalpha\_F3,BDI\text{-}II} = -.539$, and $r_{LRTCalpha\_F4,BDI\text{-}II\cdot} = -.556$, $p < .01$). Partial correlational analysis controlling for depression severity scores revealed only one significant association between suppression and theta band scaling exponents from T7T8 ($r = -.39$, $p = .02$).

No significant correlations between ER strategies and scaling exponents were found in the ND group. However, when the analysis was performed on the whole sample (i.e. including SBD and ND participants), correlations between suppression and brooding scores and temporal sites (T7T8) theta band scaling exponents remained slightly significant.

Further, we performed the Fisher r-to-z transformation for correlation comparison between SBD and ND groups, with a view to formally testing the differences between groups. On the broad band we found a significant difference at central sites regarding to suppression strategy, whereas on the alpha band another significant difference was found at frontal sites regarding to brooding.

The average scaling exponents from both eyes-closed conditions for each channel are presented in Table 3. All the scaling exponents are in the range from .5 to 1, thus revealing the presence of persistent LRTC in both the amplitude envelope of broad band and theta and alpha band oscillations from all brain sites. No significant differences were found between the scaling exponents from SBD and ND groups at any location, even in the few cases where equality of variances could not be assumed. Furthermore, we performed Cohen's d in order to obtain the effect size between SBD and ND groups. All values range from .03 to .41, showing mostly small effect sizes between groups, but also values close to a medium effect.

Discussion

The brain has been suggested to operate at a near critical state in order to maintain a healthy flexible repertoire of responses to environmental demands (Chialvo 2010; Werner 2010; Palva et al. 2013). Long range temporal correlations in EEG oscillations are taken as a signature of these critical brain dynamics, and therefore changes in LRTC are thought to be associated with disease. Smit et al. (2011) suggested that scaling in resting-state EEG might be a biomarker of pathophysiology in neurodevelopmental disorders.

Though several studies in the last decade reported associations between depression severity and LRTC in the EEG from clinically depressed patients (Lee et al. 2007; Leistedt et al. 2007; Linkenkaer-Hansen et al. 2005), to the best of our knowledge this is the first study examining the associations between LRTC in the EEG from subclinically depressed individuals and the depression-related ER strategies of brooding and suppression. These strategies are

Table 2 Bivariate correlations [and 95 % Confidence Intervals] between depressive symptoms (assessed by BDI-II), brooding and suppression and scaling exponents of EEG signals in the subclinically depressed and the non-depressed groups

	Broad band scaling exponents		Theta band scaling exponents		Alpha band scaling exponents			
	C3C4	P3P4	P3P4	T7T8	C3C4	F3F4	O1O2	T7T8
Brooding								
SBD	.195	.366[a,]*	.329*	−.349[a,]*	−.339*	−.495**	−.295	−.105
	[−.192, .530]	[−.008, .650]	[−.050, .625]	[−.639, .028]	[−.632, .039]	[−.733, −.150]	[−.602, .088]	[−.460, .279]
ND	−.055	−.031[a]	.012	−.102[a]	.096	.046	.105	.166
	[−.257, .152]	[−.234, .175]	[−.193, .216]	[−.301,.105]	[−.111, .295]	[−.160, .248]	[−.102, .303]	[−.040, .359]
Z score	1.104	1.754	1.400	−1.091	−1.922	−2.390*	−1.767	−1.197
Suppression								
SBD	.388*	.382[a,]*	.090	−.330*	−.041	−.270	−.325*	−.254
	[.017, .665]	[.010, .661]	[−.293, .448]	[−.626, .049]	[−.408, .337]	[−.584, .115]	[−.623, .055]	[−.573, .132]
ND	−.069	−.046[1]	−.023	−.142	.010	−.012	.070	.062
	[−.270, .138]	[−.248, .160]	[−.227, .183]	[−.337, .065]	[−.195, .214]	[−.216, .193]	[−.137, .271]	[−.145, .263]
Z score	2.019*	1.891	.499	−.831	−.225	−1.140	−1.745	−1.396
Depressive symptoms	.444**	.490**	.429*	.091	−.526**	−.566**	−.443**	−.324*
	[.085, .701]	[.143, .730]	[.067, .691]	[−.292, .449]	[−.752, −.190]	[−.775, −.245]	[−.700, −.084]	[−.622, .056]

Scaling exponents measured by DFA at broad band (1–40 Hz), theta (3–7 Hz), and alpha (8–13 Hz) and at central (average C3C4), parietal (average P3P4), occipital (average O1O2) and temporal (average T7T8) cortex. Fisher *r*-to-*z* transformation for correlation comparison between SBD and ND groups

SBD subclinically depressed, *ND* non-depressed

* $p < .05$; ** $p < .01$

[a] Only in one eyes-closed condition

Table 3 Mean scaling exponents from each EEG channel for the subclinically depressed and the non-depressed groups

	SBD group			ND group			Cohen's *d*
	M	SD	95 % CI	*M*	SD	95 % CI	
Broad band							
C3	.812	.056	[.790, .834]	.833	.101	[.812, .855]	.257
C4	.813	.060	[.790, .837]	.835	.103	[.814, .858]	.261
F3	.786	.068	[.759, .812]	.815	.096	[.794, .836]	.349
F4	.795	.070	[.768, .822]	.820	.098	[.799, .841]	.294
O1	.805	.073	[.776, .834]	.826	.089	[.807, .846]	.258
O2	.810	.081	[.779, .842]	.825	.087	[.807, .844]	.178
P3	.739	.090	[.704, .774]	.782	.116	[.758, .807]	.414
P4	.770	.070	[.744, .798]	.795	.105	[.773, .818]	.280
T7	.827	.095	[.790, .864]	.842	.094	[.822, .862]	.159
T8	.824	.096	[.787, .862]	.838	.102	[.817, .860]	.141
Theta band							
C3	.718	.059	[.695, .741]	.741	.063	[.728, .755]	.377
C4	.723	.073	[.695, .752]	.741	.070	[.726, .756]	.252
F3	.712	.064	[.687, .737]	.730	.067	[.726, .745]	.275
F4	.717	.059	[.694, .740]	.734	.073	[.719, .750]	.256
O1	.721	.060	[.697, .744]	.730	.070	[.716, .746]	.138
O2	.726	.052	[.706, .747]	.729	.071	[.714, .745]	.048
P3	.720	.056	[.699, .742]	.734	.071	[.719, .749]	.219
P4	.712	.051	[.693, .732]	.729	.086	[.711, .748]	.240
T7	.714	.049	[.694, .733]	.725	.069	[.710, .739]	.184
T8	.716	.047	[.698, .735]	.727	.068	[.713, .742]	.188
Alpha band							
C3	.747	.072	[.719, .775]	.766	.087	[.747, .784]	.238
C4	.749	.071	[.721, .777]	.769	.091	[.749, .788]	.245
F3	.755	.076	[.726, .785]	.777	.089	[.757, .796]	.266
F4	.762	.081	[.731, .793]	.779	.093	[.759, .799]	.195
O1	.813	.108	[.771, .855]	.807	.090	[.787, .826]	.060
O2	.821	.105	[.780, .861]	.813	.091	[.793, .832]	.081
P3	.723	.064	[.698, 748]	.751	.086	[.733, .770]	.369
P4	.744	.071	[.717, .772]	.747	.097	[.727, .768]	.035
T7	.840	.122	[.793, .888]	.834	.113	[.810, .858]	.051
T8	.847	.109	[.804, .889]	.836	.106	[.814, .859]	.102

None of the comparisons between the scaling exponents of the SBD and the ND groups was statistically significant

SBD subclinically depressed, *ND* non-depressed

among the characteristic strategies that clinically depressed patients use to cognitively regulate their emotions, and the first hypothesis was that SBD individuals would be more prone to engage in brooding and suppression ER than non-depressed individuals. The differences found in this study were highly significant, thus confirming this hypothesis.

The second hypothesis was that positive linear correlations should be found between broad band scaling exponents and depressive ER strategies as well as between theta band exponents and these strategies (Bornas et al. 2013), and that these correlations should be stronger for SBD individuals than for ND participants. Although no associations were found between alpha band LRTC and ER strategies in the study by Bornas et al. (2013), we included the alpha band in the present correlational analysis because of the presence of the SBD group in the sample. Further, correlations were also calculated for the whole sample, including SBD and ND individuals, to check the hypothesis that results from Bornas et al. (2013) could be due, at least in part, to the fact that some individuals with mild symptoms of depression could have been included in their sample.

Brooding was positively correlated with both broad band and theta band LRTC from the parietal region (P3P4) and negatively correlated with theta band LRTC from temporal regions (T7T8) and alpha band LRTC from frontal (F3F4) and central (C3C4) areas in the SBD group, but not in the ND group. Suppression was positively correlated with broad band LRTC from central regions (C3C4) and parietal areas (P3P4) and negatively correlated with theta band LRTC from temporal regions (T7T8) and alpha band LRTC from occipital (O1O2) sites in the SBD group, but not in the ND group. These results are consistent with previous findings reporting similar associations between EEG dynamics in central and parietal areas and depression-related ER strategies (Bornas et al. 2013). Although the correlation coefficients of both groups were highly different, when we formally tested for these differences we only found two significant results; on broad band at central sites in suppression and on alpha band at frontal sites in brooding. In addition we found negative correlations between these strategies and theta band LRTC from temporal sites and alpha band LRTC from central, frontal, and occipital areas. These findings have not been reported previously. However, a negative correlation between left temporocentral theta band LRTC and depression severity was reported by Linkenkaer-Hansen et al. (2005), and we will discuss this issue shortly. When the same analysis was performed on the whole sample (i.e. including SBD and ND individuals), some of the strongest correlations found in the SBD group remained significant though r values were lower. Therefore, we can speculate that the results reported in Bornas et al. (2013) might have been due to the unnoticed inclusion of mildly depressed individuals in their sample.

As to the third hypothesis, Lee et al. (2007) reported positive correlations between broad band scaling exponents from almost all sites (F3, F4, C3, C4, T3, T4 and O1) and depression scores. When we used the same window range and a very close band amplitude, we obtained similar results for central areas and parietal areas as well. We did not find, however, associations between frontal, occipital, and temporal LRTC and depression scores. It seems reasonable to think that while severely depressed patients show a slower decay of LRTC widely distributed over the scalp, these slower decays are more specifically located in parietal and central regions in individuals classified as subclinically depressed. When larger time windows were used for DFA of theta and alpha band oscillations (thus approaching the procedure described in Linkenkaer-Hansen et al. 2005), only theta LRTC from parietal sites (P3P4) were positively associated with depression severity while alpha LRTC from frontal, central, occipital and temporal areas were negatively correlated with depression severity scores. Based on the associations found by Linkenkaer-Hansen et al. (2005) we predicted just the opposite results (i.e. negative correlations for the theta band and smaller positive correlations for the alpha band). It should be noticed, however, that the longest window in our study was 6 s while they were able to use windows up to 100 s. Further, we used ten EEG channels while they used three MEG areas. These methodological differences could explain the different results of both studies.

The strong negative correlations we found between the alpha LRTC and the depression scores are perhaps not surprising if we consider that the relationships between frontal alpha power and depression have been known since the early eighties (Shaffer et al. 1983; Henriques and Davidson 1990; Heller et al. 1998; Davidson 2004) though there is still debate as to the precise meaning of those relationships (see Miller et al. 2013, for a recent review of the two more accepted theoretical models—the valence/mood model versus the approach and withdrawal model—of frontal lateralization). Nikulin and Brismar (2005) reported significant positive linear correlations between alpha scaling exponents and alpha power, and therefore the bilateral breakdown of LRTC in frontal and central alpha oscillations would correspond to a bilateral decrease in alpha power in SBD individuals who, in addition, were prone to engage in brooding to regulate their emotions. The proneness to use depression-related ER strategies was associated, as we mentioned above, with a breakdown of LRTC in temporal theta oscillations also.

The abnormal dynamics in the theta frequency band, although likely to be of neocortical origin, is related to the changes in limbic-cortical pathways that have been identified in patients with major depressive disorder; further, Gray's motivational theory states that theta oscillations are connected to memory, anxiety and punishment learning (Gray 1982). Linkenkaer-Hansen et al. (2005) proposed that the observed breakdown of temporal correlations in theta oscillations may reflect impaired mnemonic functions operating when stimulus-independent thoughts take place during eyes-closed wakeful rest. These impaired mnemonic functions have also been associated with EEG complexity in other studies (e.g. Talebi et al. 2012). Participants in the SBD group were more prone to engage in negative ER strategies than participants in the ND group. In a similar way, the breakdown of temporal correlations in theta oscillations in this study may reflect impaired mnemonic functions operating when SBD participants engaged in maladaptive ER strategies during the wakeful rest period. The altered dynamics at temporal sites may also be consistent with the results reported by Atagün et al. (2013). In a study that used an auditory oddball paradigm, these authors found differences between euthymic bipolar patients and healthy controls in the T7 and T8 slow theta (4–6 Hz) response to simple as well as non-target stimuli.

These differences seem to be related to short-term memory functions (for a recent review of the functions of theta rhythms, see Colgin 2013).

Perhaps the breakdown of LRTC in the alpha band would reflect this engagement also, as the alpha oscillations in wakeful resting conditions have been considered "a prototype of a dynamic process which governs a large ensemble of integrative brain functions" (Schürmann and Başar 2001). In line with this view, Knyazev (2007) underlined the inhibitory function of alpha oscillations that should inhibit "unnecessary or conflicting processes to the task in hand" and thus alpha would play a key role in many cognitive processes that require such inhibitory action. We can speculate, therefore, that the breakdown of alpha LRTC leads to a lack of inhibitory function that allows brooding and ruminative processes to emerge. Indeed, in EC conditions the alpha LRTC are stronger than in EO conditions due to the lack of visual sensory input (Linkenkaer-Hansen et al. 2004), and Nikulin and Brismar (2005) suggested that, similarly, disruption of the alpha LRTC may be caused by increased sensory input by a high level of alertness that in our opinion, conceivably, would be associated with engaging in cognitive brooding ER strategies. Unfortunately, however, we did not ask participants to report what they were actually thinking during these periods.

The last hypothesis concerned the differences between the scaling exponents of the SBD and ND groups. Based on previous studies with severely depressed patients that either found significant differences between patients and healthy subjects (Lee et al. 2007) or not (Hosseinifard et al. 2013), we predicted smaller differences and actually did not find significant differences at any brain site. Therefore, having a scaling exponent of some magnitude cannot be used as a marker or a diagnosis tool for SBD individuals despite it seeming useful for more severe depressive disorders. This result makes the findings of the associations between LRTC in the brain and the depression-related ER strategies in the SBD group (and in the ND group as well) discussed above more relevant. However, as the clinical group was subthreshold, Cohen's d was also calculated because even a small effect could be of interest for future research in more severely ill populations. All results were comprised between .03 and .41, showing mostly small effect sizes but also a few values close to a medium effect.

Further research is needed to understand the precise meaning of the correlations (both positive and negative correlations) found in this study, but the big picture shows that changes in frontal, central, parietal, occipital and temporal LRTC are associated with cognitive depression-related ER strategies. Scaling and LRTC in brain dynamics reflect multistability, i.e. the coexistence of multiple operating regimes in biological systems (Pittman-Polletta et al. 2013), which allows the system to shift effortlessly between different cognitive tasks. Therefore, when LRTC are somehow altered it seems plausible to think of a decrease in multistability that would in turn lead to less flexible dynamics. Brooding, rumination, suppression, perseverative thinking or worry, are rigid cognitive strategies that may be the result of a decrease in the multistability properties of the brain. This loss of flexibility characterizes subclinically or mildly depressed people. As Brzezicka (2013) points out, people who suffer mild forms of depression may show serious impairments in tasks requiring flexible thinking.

To sum up, in addition to the already known associations between depression severity scores and EEG LRTC, this study shows that depression-related ER strategies (specifically brooding and thought suppression) are also associated with LRTC. If we assume that the use of these strategies, jointly with the current or past presence of depressive mood, appear to render individuals more likely to develop depressive episodes, then these results may add information about potential risk markers of the onset or recurrence of depressive disorders. Nevertheless, the inclusion of eight individuals with a history of depression (and other related disorders) in our samples makes it possible (though not likely) that the EEG abnormal dynamics reflects also a scar effect of past depressive symptoms.

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