Annals of Depression and Anxiety

Open Access

Austin Publishing Group

Review Article

Long Range Temporal Correlations in EEG and Depression

Fiol-Veny A, Balle M and Bornas X*
Department of Psychology, University of the Balearic Islands, Spain

***Corresponding author:** Bornas X, Department of Psychology, University of the Balearic Islands, Carretera de Valldemossa km. 7.5, 07122 Palma, Mallorca, Spain, Tel: 34971172580; Fax: 34971173190; Email: xavier.bornas@gmail.com

Received: November 24, 2014; **Accepted:** March 18, 2015; **Published:** March 31, 2015

Abstract

The relationship between Long Range Temporal Correlations (LRTC) from EEG oscillations and depression or depression-related emotion regulation strategies has been a focus of interest in the last few years and its demonstration through Detrended Fluctuation Analysis (DFA) has been determined by several authors. Despite this, a wide range of methods and procedures have been used to obtain these measures, leading to a large amount of hardly comparable results. In this review we summarize the main outcomes and find that there are consistencies between these studies, but also many inconsistencies that make obvious the need of a unified line of investigation. We also propose some suggestions for the future in order to improve our knowledge about LRTC and depression.

Keywords: Long range temporal correlations; EEG; Depression; Emotion regulation; Detrended fluctuation analysis

Abbreviations

LRTC: Long Range Temporal Correlations; EEG: Electroencephalogram; DFA: Detrended Fluctuation Analysis; ER: Emotion Regulation; BDI: Beck Depression Inventory; HDRS: Hamilton Depression Ranting Scale; SCID: Structured Clinical Interview for DSM-IV; REM: Rapid Eye Movement; CERQ: Cognitive Emotion Regulation Questionnaire; WBSI: White Bear Supression Inventory; RRS: Ruminative Response Scale

Introduction

Depression is one of the most common and disabling disorders and the fact that EEG may be a useful tool for investigating brain regional mechanisms underlying depressive disorders, has already been noted by some authors [1-3]. For instance, Richard J. Davidson developed a model [4] that considers the role of the anterior brain asymmetry and suggests that differences in prefrontal asymmetry activation are a diathesis that biases the person's affective style and modulates its vulnerability to develop depression. In addition to this model-based research (and other research studies based also on a linear perspective on EEG activity) a non-linear, complexity-oriented research field has been growing during the last decades with the general purpose of obtaining a better comprehension of the brain dynamics. It is within this theoretical framework that several studies addressed the relationship between EEG Long Range Temporal Correlations (LRTC) and depression [5-10] and between LRTC and cognitive Emotion Regulation (ER) strategies commonly used by people with a depressive ER style [11,12]. This style is observed in individuals who engage in maladaptive cognitive ER strategies such as ineffective attempts to avoid or to suppress expressions of emotion and unwanted thoughts (e.g. brooding, rumination, suppression, etc).

Neural oscillations are known to show great variability and apparently random changes over time, even in resting state [13]. In recent years, the dynamical structure of EEG ongoing oscillations has been broadly studied [13-15]. LRTC were first demonstrated in the amplitude of 10 and 20 Hz spontaneous neuronal oscillations by Linkenkaer-Hansen et al. [14]. This temporal structure indicates the presence of auto correlations that decay slowly and remain significant at time scales from seconds to minutes (i.e. a relatively *long range* of time). A feature of these correlations is their power-law scaling behaviour which indicates that the underlying processes are not governed by a unique characteristic scale, thus allowing us to deduce that the process is self-similar. According to the theory of self-organized criticality [16], the fact that complex systems follow a power-law behaviour lead us to think in a common mechanism that brings the system into a critical state where it is self-organized during processing demands [14]. At present, there are several neuroscientists who consider the brain as a system which tends to self-organized criticality [17-20].

The Detrended Fluctuation Analysis (DFA) [21] is a non-linear analysis technique that permits the detection of long range correlations in seemingly non-stationary time series, through the value of an exponent obtained from it, named scaling exponent α. This is a quantitative parameter that represents the autocorrelation properties of a time series. Since the studies reviewed in this paper use DFA to investigate LRTC in brain activity it is worth describing briefly how it works. First of all, the EEG signal is integrated, y (k), by a cumulative sum of the amplitude envelope. The envelope of an oscillating signal is a smooth line which outlines its extremes. Then, the integrated time series is divided into segments of equal length. The trend of each segment is obtained by a least-squares line and subsequently, the series is detrended by subtracting in each segment its local trend. The next step consists of dividing the detrended integrated signal into non-overlapping windows with different lengths equidistantly distributed on a logarithmic scale. For each window size n, the variance F^2 (n) is calculated in the detrended signal. Finally, the slope of the line relating log F (n) and log n is the scaling exponent α. The presence of LRTC is proved by a scaling exponent $0.5 < \alpha < 1$, which indicates the data are correlated, such that

Citation: Fiol-Veny A, Balle M and Bornas X. Long Range Temporal Correlations in EEG and Depression. Ann Depress Anxiety. 2015;2(1): 1041.

Table 1: Summary of the reviewed studies (sorted by publication year).

Study	Condition	Groups	EEG Frequency Band	Windows Size	Main Findings
Linkenkaer-Hansen et al. [5]	Resting	D ND	Theta (3 – 7 Hz) Alpha (8 – 13 Hz)	5 – 100s	α D < α ND Negative correlation α – DS in theta Small positive correlation α – DS in alpha
Lee et al. [6]	Resting	D ND	Broad (0.6 – 46 Hz)	0.1 - .06s	α D > α ND Positive correlation α – DS in broad band
Leistedt et al. [7]	Sleep	D men ND men	Broad (0.5 – 25 Hz)	0.16 – 2s	SS2 & SWS: α D < α ND Negative correlation α – DS in SS2 & SWS
Leidstedt et al. [8]	Sleep	DR men ND men	Broad (0.5 – 25 Hz)	0.16 – 2s	No differences α D – α ND
Bornas et al. [11]	Resting	ND	Theta (3 – 7 Hz) Alpha (8 – 13 Hz) Broad (1 – 40 Hz)	1 – 6 s 0.1 – 0.6s	Positive correlation α ND – ERS(-)
Hosseinifard et al. [9]	Resting	D ND	Alpha (8 – 13 Hz) Beta (13 – 30 Hz) Delta (0.5 – 4 Hz) Theta (4 – 8 Hz)	Not available	No differences α D – α ND
Bachmann et al. [10]	Resting	D women ND women	Broad (0.3 – 70 Hz)	0.1 – 1.1s	α D < α ND
Bornas et al. [11]	Resting	ND SBD	Theta (3 – 7 Hz) Alpha (8 – 13 Hz) Broad (1 – 40 Hz)	1 – 6 s 0.1 – 0.6s	No differences α D – α SBD Positive correlation α SBD – ERS(-)/DS in broad band Negative correlation α SBD – ERS(-)/DS in alpha Positive & negative correlation α SBD – ERS(-)/DS in theta

Note. D: Depressed; ND: Non-Depressed; α: Scaling Exponent; DR: Depression in Remission; SBD: Sub Clinically Depressed; DS: Depression Scores; SS2: Sleep Stage 2; SWS: Slow Wave Sleep; ERS (-): Negative Emotion Regulation Strategies.

large fluctuations are likely to be followed by large fluctuations and small fluctuations are likely to be followed by small fluctuations. The presence of LRTC in EEG signals has been repeatedly reported by using DFA [5-7,11,12,14,15,22-24].

This paper is an attempt to review and unify the existent evidence that connects LRTC in EEG and depression. We used the following databases: Scopus, Web of Science, Google Scholar and Pub med. The terms considered in the searching process were:['long range temporal correlations' OR' long range correlations' OR 'scaling' OR 'scaling exponents' OR 'detrended fluctuation analysis']AND['depression' OR 'depressed' OR 'emotion regulation' OR 'emotion regulation strategies']AND['EEG']. Eight research studies satisfied these search criteria and were subsequently reviewed (Table 1).

Current evidence

Most of these studies have focused on brain signals from depressed patients and healthy controls in resting or sleep conditions with eyes closed. Some of the procedure details (as the frequency band or the windows size selected) are only reported in the Table 1, in order to simplify and make the information more clear and schematic. Before we go any further, we should have in mind that the LRTC are more persistent, so decaying more slowly with time, as the scaling exponent increases from 0.5 toward 1.

Lee et al. [6] used EEG recordings taken during 5 min resting periods. To classify the subjects as depressed, the Beck Depression Inventory (BDI) [25] and the 17-item Hamilton Depression Ranting Scale (HDRS) [26] were used, as well as a DSM-IV interview. The scaling exponents of depressed patients had relatively higher values in whole brain regions in comparison to healthy controls, and a significant positive linear correlation was observed between the severity of depression and the exponents over most of the channels.

On the other hand, Linkenkaer-Hansen et al. [5] used MEG recordings taken during 16 min resting periods. Participants were classified as depressed based on their scores on the 17-item HDRS and the Structured Clinical Interview for DSM-IV (SCID). Scaling exponents were larger in control participants than in depressed patients, and a significant negative linear correlation was observed between the severity of depression and the exponents of theta oscillations over the left temporocentral region. They also found a marginal, albeit significant, positive correlation between the exponents of alpha occipitoparietal oscillations and the depression scores.

Hosseinifard et al. [9] studied the utility of nonlinear analysis of resting EEG signals taken during 5 min, to discriminate between depressed and control subjects. For assessing the depression severity they used a DSM-IV interview and the BDI questionnaire. Unfortunately, the windows size selected is not available in this article. Though they studied several frequency bands, they found no significant differences in scaling exponents between both groups.

Recently, Bachmann et al. [10] used 5 min of resting EEG recordings from a sample of depressed and healthy women. The scaling exponents were calculated over the fitting range 0.1-1.1s. The 17-item HRDS and the ICD-10 criteria were used to classify the individuals as depressed. The exponents from the non-depressed were significantly higher than those from the depressed participants.

Leistedt et al. [7] focused on the sleep EEG from a sample of men with major depressive disorder and healthy controls. The patients were diagnosed according to DSM-IV-TR criteria and depressive symptoms were rated by means of the 24-item HRDS [27]. The exponents were lower during sleep stage 2 and slow wave sleep in the depressed group, and a negative linear correlation was observed between exponents and depression severity during slow wave sleep. They concluded that these results could be an explanation of the typical sleep fragmentation observed in major depressive episodes.

In another study, Leistedt et al. [8] used also a sample of untreated depressed and healthy men, although the patients were in full to partial remission, and followed the same method described above to calculate DFA. Both remission statuses were assessed with the 24-item HRDS. They found no significant differences in scaling exponents between

groups during the three sleep stages (2, slow wave sleep and REM). They argued this is a sign of similar underlying neuronal dynamics in both groups and also an argument in favor to the non-permanency of the LRTC modifications observed in depressed patients.

One open-question is whether changes in the EEG LRTC are linked with cognitive ER strategies commonly used by people with a depressive ER style even if they cannot be classified as clinically depressed. In an attempt to answer that question and to conciliate the two procedures used by Lee et al. [6] and Linkenkaer-Hansen et al. [5], Bornas et al. [11] carried out a study with a sample of non-depressed individuals. The associations between LRTC of spontaneous oscillations and negative ER strategies related to depression were explored using two different correlational analyses. Following Linkenkaer-Hansen et al. [5] the DFA was performed on the theta band oscillations while resembling Lee et al. [6] the exponents of the broad band oscillations were calculated. The questionnaires were the Cognitive Emotion Regulation Questionnaire (CERQ) [28], the White Bear Suppression Inventory (WBSI) [29], and the Ruminative Response Scale (RRS) [30] to assess ER strategies, and the BDI-II [31] to assess depressive symptomatology. All correlations were positive, that means people more prone to engage in these strategies had higher scaling exponents.

Bornas et al. [12] tried to shed some light on the issue by exploring the associations between EEG LRTC and depression-related ER strategies (brooding and supression) in a sample of sub clinically depressed and non-depressed individuals. They also focused on the relation between LRTC and depression severity on the sub clinically depressed participants. According to several authors [32,33], severely depressed and sub clinically depressed people share cognitive impairments and, probably, neural dysfunctions, though the latter perform quite normally on some cognitive tasks. DFA's analyses were performed to the amplitude envelope of the broad, theta and alpha band spontaneous EEG oscillations and the procedure followed was equal from the one used by Bornas et al. [11]. The participants were asked to respond the WBSI, RRS and BDI-II questionnaires. Between-groups differences were not found but there were several linear correlations between LRTC and maladaptive ER strategies and severity of depression in the subclinically depressed group, in almost the whole brain. Thus, scaling exponents from broad band correlated positively with both strategies and depression, while scaling exponents from alpha band did it in a negative way. Theta band scaling exponents showed positive associations with brooding and depression at parietal sites, whereas correlated negatively with brooding and suppression at temporal regions. These findings suggest that alterations in brain dynamics are related with the proneness that depressive individuals show to engage in brooding and thought suppression.

Discussion and Conclusion

The importance of the relationship between EEG LRTC and depression has been highlighted during the last decade. Although the evidence in the field is growing and the DFA is the mostly used method to detect LRTC, the specific details like the range of window sizes or the frequency bands chosen in each studyare quite different, making results hardly comparable and reporting a large amount of inconsistent findings.

As we have seen, Lee et al. [6] found that scaling exponents from depressed patients were significantly higher than those from healthy controls, the opposite of the findings reported by Linkenkaer-Hansen et al. [5], Leistedt et al. [7] and Bachmann et al. [10]. Furthermore, Hosseinifard et al. [9] did not even find those differences; neither did Leidstedt et al. [8] with their sample of patients with depression in remission, nor Bornas et al. [12] with their sample of subclinically depressed individuals. There is also a wider variety of results regarding the association between the scaling exponents and the severity of depression, as well as the depression-related ER strategies, resulting in both positive and negative correlations. In this respect, one study reports positive linear correlations between exponents and depression severity [6], two studies show mostly negative linear correlations [5,7], and another one finds both positive and negative correlations between these variables [12]. Regarding the relationship between the exponents and the ER strategies, one of the studies finds positive linear correlation [11] whereas the other one reports both positive and negative linear correlations [12].

As we previously remarked, we need to be careful with this summary, taking into account the variety of procedures used in these studies. To start with, the wide range of windows of time used to calculate the scaling exponents is probably affecting the outcomes, as several authors have already pointed out [10-12]. Moreover, the studies mentioned have focused in the amplitude envelope of different EEG rhythms (broad, alpha, beta, delta, theta), thus we mostly have isolated results. To date, the only research that try to unify some of the procedures used to calculate DFA are the ones carried out by Bornas et al. [11,12], obtaining results that can help to clarify the issue. Additionally, the exploration of neuronal dynamics in subclinically depressed individuals and the depression-related ER strategies add some relevant information to the big picture since subclinical depression often precedes clinical depression [32,33] and the strategies above-mentioned have certainly an important role in depression [34-40]. In Bornas et al. [11] all correlations were positive for all frequency bands, being consistent with the results reported by Lee et al. [6] who calculated the scaling exponents in time windows of a similar length (seconds) and opposite to Linkenkaer-Hansen et al. [5], who calculated the exponents over minutes. They argued that the 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 (in theta band) would not be so surprising.Bornas et al. [12] found positive correlations between broad band exponents and both strategies and depression (similar to Lee et al. [6] and Bornas et al. [11]) and positive correlations between theta scaling exponents and depression, according to Bornas et al. [11]. However, they also found some results not reported previously: negative correlations between alpha scaling exponents and both depression and strategies, and between theta scaling exponents and ER strategies. They conclude that the different results observed in both studies could be due to the probable unnoticed inclusion of individuals with mild depression symptoms in the sample from Bornas et al. [11], since no screening measures were used to identify these individuals.Another point to take in consideration regarding the variety of results is the condition in which the EEG signals were recorded. All studies were done in resting conditions, excepting the ones from Leidstedt et al. [7,8] which were

performed during sleep. Finally, the instruments used to classify the subjects into groups and rate the depressive symptomatology severity can also be a point of differentiation, given that there are several studies that criticize the use of both instruments due to the modest association reported between their total scores [41,42].

Future research

Taking in consideration the broad range of outcomes derived from these studies, we propose a few future lines of research that may help to improve our current knowledge. First of all, it might be worthy to investigate which is the most relevant frequency bands implied in EEG dynamics related to depression and which are the most appropriate time windows to detect them. Having this information, research outcomes would probably be more comparable and thus more consistent findings should be expected. As previously stated, depression assessment has mainly been based on questionnaire measurements, as BDI or HRDS, and clinical interviews. The previous studies have only considered the depressive symptomatology level from people who suffer from major depressive disorder or subclinical depression, or healthy participants. In our opinion, with this procedure we can be missing important information derived from type of depression (unique episode or recidivate, dysthymia, bipolar depression) and hence losing nuances that could give us a key to understand the issue at hand. We should also consider the difference between depressive states and depressive processes, as for example the ER strategies above mentioned or attention biases. Bornas et al. [12] already went deeper into that when they suggested that the LRTC over short windows might be reflecting depressive processes (ER strategies) whereas LRTC over longer windows might be related with depressive state. Studying the mechanisms of these two concepts separately (states and processes) would be an essential target for the future since previous research seems to point to the fact that these mechanisms are different. Related to that, assessing the individuals not only in resting conditions but also while they are performing some cognitive task which implies worry or ruminative thoughts could deeply enrich the information about the depressive processes and its relationship with the EEG dynamics. To sum up, evidence presented here highlights the relation between EEG LRTC and depression and suggests that, in the future, LRTC could be an important feature to take into account in the assessment of depression.

References

1. Henriques JB, Davidson RJ. Left frontal hypoactivation in depression. J Abnorm Psychol. 1991; 100: 535-545.
2. Knott V, Mahoney C, Kennedy S, Evans K. EEG power, frequency, asymmetry and coherence in male depression. Psychiatry Res. 2001; 106: 123-140.
3. Omel'chenko VP, Zaika VG. [Changes in the EEG rhythms in endogenous depressive disorders and the effect of pharmacotherapy]. Fiziol Cheloveka. 2002; 28: 30-36.
4. Davidson RJ. Anterior electrophysiological asymmetries, emotion, and depression: conceptual and methodological conundrums. Psychophysiology. 1998; 35: 607-614.
5. Linkenkaer-Hansen K, Monto S, Rytsala H, Suominen K, Isometsa E, Kahkonen S. Breakdown of long-range temporal correlations in theta oscillations in patients with major depressive disorder. J Neurosci. 2005; 25: 10131-10137.
6. Lee JS, Yang BH, Lee JH, Choi JH, Choi IG, Kim SB. Detrended fluctuation analysis of resting EEG in depressed outpatients and healthy controls. Clin Neurophysiol. 2007; 118: 2489-2496.
7. Leistedt S, Dumont M, Lanquart JP, Jurysta F, Linkowski P. Characterization of the sleep EEG in acutely depressed men using detrended fluctuation analysis. Clin Neurophysiol. 2007; 118: 940-950.
8. Leistedt S, Dumont M, Coumans N, Lanquart JP, Jurysta F, Linkowski P. The modifications of the long-range temporal correlations of the sleep EEG due to major depressive episode disappear with the status of remission. Neuroscience. 2007; 148: 782-793.
9. Hosseinifard B, Moradi MH, Rostami R. Classifying depression patients and normal subjects using machine learning techniques and nonlinear features from EEG signal. Comput Methods Programs Biomed. 2013; 109: 339-345.
10. Bachmann M, Suhhova A, Lass J, Aadamsoo K, Vohma U, Hinrikus H. Detrended fluctuation analysis of EEG in depression. Proceedings of XIII Mediterranean Conference on Medical and Biological Engineering and Computing; 2013. Sevilla, Spain. Springer. 2014.
11. Bornas X, Noguera M, Balle M, Morillas-Romero A, Aguayo-Siquier B, Tortella-Feliu M, et al. Long-Range Temporal Correlations in Resting EEG. J Psychophysiol. 2013; 27: 60-66.
12. Bornas X, Fiol-Veny A, Balle M, Morillas-Romero A, Tortella-Feliu M. Long range temporal correlations in EEG oscillations of subclinically depressed individuals: their association with brooding and suppression. Cogn Neurodyn. 2015; 9: 53-62.
13. Berthouze L, James LM, Farmer SF. Human EEG shows long-range temporal correlations of oscillation amplitude in Theta, Alpha and Beta bands across a wide age range. Clin Neurophysiol. 2010; 121: 1187-1197.
14. Linkenkaer-Hansen K, Nikouline VV, Palva JM, Ilmoniemi RJ. Long-range temporal correlations and scaling behavior in human brain oscillations. J Neurosci. 2001; 21: 1370-1377.
15. Nikulin VV, Brismar T. Long-range temporal correlations in electroencephalographic oscillations: Relation to topography, frequency band, age and gender. Neuroscience. 2005; 130: 549-558.
16. Bak P, Tang C, Wiesenfeld K. Self-organized criticality: An explanation of the 1/f noise. Phys Rev Lett. 1987; 59: 381-384.
17. Chialvo DR. Emergent complex neural dynamics. Nat Phys. 2010; 6: 744-750.
18. Werner G. Fractals in the nervous system: conceptual implications for theoretical neuroscience. Front Physiol. 2010; 1: 15.
19. Palva JM, Zhigalov A, Hirvonen J, Korhonen O, Linkenkaer-Hansen K, Palva S. Neuronal long-range temporal correlations and avalanche dynamics are correlated with behavioral scaling laws. Proc Natl Acad Sci USA. 2013; 110: 3585-3590.
20. Pittman-Polletta BR, Scheer FA, Butler MP, Shea SA, Hu K. The role of the circadian system in fractal neurophysiological control. Biol Rev Camb Philos Soc. 2013; 88: 873-894.
21. Peng CK, Havlin S, Stanley HE, Goldberger AL. Quantification of scaling exponents and crossover phenomena in nonstationary heartbeat time series. Chaos. 1995; 5: 82-87.
22. Linkenkaer-Hansen K, Smit DJ, Barkil A, van Beijsterveldt TE, Brussaard AB, Boomsma DI, et al. Genetic contributions to long-range temporal correlations in ongoing oscillations. J Neurosci. 2007; 27: 13882-13889.
23. Nikulin VV, Brismar T. Long-range temporal correlations in alpha and beta oscillations: effect of arousal level and test-retest reliability. Clin Neurophysiol. 2004; 115: 1896-1908.
24. Smit DJ, de Geus EJ, van de Nieuwenhuijzen ME, van Beijsterveldt CE, van Baal GC, Mansvelder HD, et al. Scale-free modulation of resting-state neuronal oscillations reflects prolonged brain maturation in humans. J Neurosci. 2011; 31: 13128-13136.
25. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiatry. 1961; 4: 561-571.
26. Hamilton M. A rating scale for depression. J Neurol Neurosurg Psychiatry. 1960; 23: 56-62.

27. Hamilton M. Development of a rating scale for primary depressive illness. Br J Soc Clin Psychol. 1967; 6: 278-296.

28. Garnefski N, Kraaij V, Spinhoven P. Negative life events, cognitive emotion regulation and emotional problems. Pers Individ Dif. 2001; 30: 1311–1327.

29. Wegner DM, Zanakos S. Chronic thought suppression. J Pers. 1994; 62: 616-640.

30. Treynor W, Gonzalez R, Nolen-Hoeksema S. Rumination reconsidered: A psychometric analysis. Cognitive Ther Res. 2003; 27: 247-259.

31. Beck AT, Brown GK, Steer RA. Beck Depression Inventory II (BDI-II) Manual. San Antonio: The Psychological Coorporation. 1996.

32. Cuijpers P, de Graaf R, van Dorsselaer S. Minor depression: risk profiles, functional disability, health care use and risk of developing major depression. J Affect Disord. 2004; 79: 71-79.

33. de Graaf LE, Huibers MJ, Cuijpers P, Arntz A. Minor and major depression in the general population: does dysfunctional thinking play a role? Compr Psychiatry. 2010; 51: 266-274.

34. Nolen-Hoeksema S, Morrow J, Fredrickson BL. Response styles and the duration of episodes of depressed mood. J Abnorm Psychol. 1993; 102: 20-28.

35. Nolen-Hoeksema S. The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. J Abnorm Psychol. 2000; 109: 504-511.

36. Moberly NJ, Watkins ER. Ruminative self-focus, negative life events, and negative affect. Behav Res Ther. 2008; 46: 1034-1039.

37. Verstraeten K, Vasey MW, Raes F, Bijttebier P. Temperament and risk for depressive symptoms in adolescence: mediation by rumination and moderation by effortful control. J Abnorm Child Psychol. 2009; 37: 349-361.

38. Barlow DH, Allen LB, Choate ML. Toward a unified treatment for emotional disorders. Behav Ther. 2004; 35: 205-230.

39. Hayes SC, Wilson KG, Gifford EV, Follette VM, Strosahl K. Experimental avoidance and behavioral disorders: a functional dimensional approach to diagnosis and treatment. J Consult Clin Psychol. 1996; 64: 1152-1168.

40. Shahar B, Herr NR. Depressive symptoms predict inflexibly high levels of experiential avoidance in response to daily negative affect: a daily diary study. Behav Res Ther. 2011; 49: 676-681.

41. Davies B, Burrows G, Poynton C. A comparative study of four depression rating scales. Aust N Z J Psychiatry. 1975; 9: 21-24.

42. Richter P, Werner J, Heerlein A, Kraus A, Sauer H. On the validity of the Beck Depression Inventory. A review. Psychopathology. 1998; 31: 160-168.

Ann Depress Anxiety - Volume 2 Issue 1 - 2015
ISSN : 2381-8883 | www.austinpublishinggroup.com

Citation: Fiol-Veny A, Balle M and Bornas X. Long Range Temporal Correlations in EEG and Depression. Ann Depress Anxiety. 2015;2(1): 1041.