Research Article

The evaluation of structural differences between the sleep EEGs of depressive and normal subjects by using itakura distance measure: A preliminary study

Levent ÖZBEK[1], **Levent SÜTÇIGIL[2]**, **Hamdullah AYDIN[3]**, **Kazım Cihan CAN[4]**,
Sinan YETKIN[5], **Asuhan PAR[5]**, **Fuat ÖZGEN[6]**

[1]*Department of Statistics, Faculty of Science, Ankara University, 06100, Türkiye*
[2]*Department of Psychology, Faculty of Economics and Administrative Sciences, Hasan Kalyoncu University, Gaziantep, 27 27410, Türkiye*
[3]*Department of Psychology, Faculty of Economics and Administrative Sciences, Altınbaş University, 34218, Türkiye*
[4]*Department of Psychiatry, Ankara City Hospital, 06800, Türkiye*
[5]*Department of Psychiatry, Sağlık Bilimleri University, Gülhane Medical Application and Research Center, 06010, Türkiye*
[6]*Clinic of Psychiatry, Bayındır Hospital, Ankara, 06530, Türkiye*

ABSTRACT

Electroencephalogram (EEG): It is used to diagnose, monitor, and manage neurophysiological disorders related to epilepsy and sleep disorders. The definition of sleep and wakefulness in polysomnography is also made with the EEG technique. The relationship between depression and sleep disturbances has been examined in many epidemiological and clinical studies. Clinical observations and studies suggest that the changes in sleep structure in depression are sensitive, even specific. This study aims to research the structural differences in sleep EEGs of healthy subjects and subjects with depressive disorder between their non-rapid eye movement (NREM), non-rapid eye movement (N2), and rapid eye movement (REM) stages by using the Itakura Distance Measure. In comparison between the N2 and REM epochs of the healthy subjects, the distance is short. In the comparison between N2 and REM epochs of depressed subjects with each other and healthy subjects, the distance has been found to be large. The study indicates that the sleep EEG of the patients differs in the N2 stage as much as it does in REM.

Cite this article as: Özbek L, Sütçigil L, Aydın H, Can KC, Yetkin S, Par A, Özgen F. The evaluation of structural differences between the sleep EEGs of depressive and normal subjects by using itakura distance measure: A preliminary study. Sigma J Eng Nat Sci 2024;42(3):795–804.

*Corresponding author.
*E-mail address: ozbek@science.ankara.edu.tr

INTRODUCTION

Sleep is a physiologic, periodic resting state of the body in which the eyes are closed and sensational functions are diminished. Rather than an inactive phase of the body, sleep is a dynamic process and has many functions that help to sustain homeostasis and reconstruct the body and the central nervous system. Sleep is the primary function of the brain and plays an essential role in an individual's performance, learning ability, and physical movement.

Humans spend around one-third of their lives sleeping, and conditions such as insomnia and obstructive sleep apnea are frequent and can severely affect physical health. 50–70 million people suffer from sleep disorders in the United States. In addition, more than 90% of patients with depressive disorders suffer from sleep disorders. Sleep apnea is estimated to be common in 2–4% of adults and 1–3% of children. Approximately 33% of the world's population suffers from insomnia symptoms [1].

Changes in EEG may carry important clinical information. Accurate detection and characterization of such EEGs can be valuable for the clinical assessment of the neurological condition. The scoring of sleep is an important step in the investigation of sleep structure, the classification of diseases, and the selection of appropriate treatment applications.

There are four sleep stages: one for rapid eye movement (REM) sleep and three for non-REM (NREM) sleep. These stages are determined based on an analysis of brain activity during sleep, which shows distinct patterns that characterize each stage. The classification of sleep stages was updated in 2007 by the American Academy of Sleep Medicine (AASM). Before that, most experts referred to five sleep stages, but today, the AASM›s definitions of the four stages represent the consensus understanding of the sleep cycle [1].

The human body goes through two stages of sleep: (1) rapid eye movement (REM) and (2) non-rapid eye movement (NREM) sleep; This sleep is further divided into three stages, N1-N3. Sleep quality and the time spent in each sleep stage may vary due to depression, aging, traumatic brain injuries, medications.

N1 (Stage 1), light sleep (5%): This is the lightest stage of sleep and begins with more than 50% of alpha waves being replaced by low-amplitude mixed waves.

N2 (Stage 2), Deeper Sleep (45%): This stage is characterized by the presence of sleep spindles, K-complexes, or both. Stage 2 sleep lasts approximately 25 minutes in the first cycle and becomes longer with each successive cycle, eventually accounting for approximately 45% of total sleep.

N3 (Stage 3), Deepest Non-REM Sleep (25%): This is considered the deepest stage of sleep and is characterized by signals with much lower frequencies and higher amplitudes, known as delta waves. This stage is the most difficult to wake up from.

REM (25%): REM is associated with dreaming and is not considered a restful sleep phase. Although the EEG resembles that of an awake person, the skeletal muscles are atonic and inactive, except for the respiratory muscles of the eyes and diaphragm, which remain active. This phase usually begins 90 minutes after falling asleep, and each REM cycle extends throughout the night. The first period usually lasts 10 minutes, and the last period lasts about an hour. REM is when dreaming occurs [2].

Depression is a common mood disorder that might cause a persistent feeling of sadness, a loss of interest, and impairments of memory and concentration. Depressed patients normally experience cognitive impairment and suffer long and severe emotional depression. In severe cases, some patients will experience paranoia and illusions. As a result, diagnosing depression in its early curable stages is critical and may save a patient's life [3].

The symptoms of depression appear mostly as behavioral ones. Normally, the help of psychiatrists or counselors is sought for diagnosis and treatment. Identification of depression in its early stages is crucial to preventing it from reaching a severe and irreversible state. The electroencephalogram (EEG) may be used as a tool for making an objective diagnosis of depression [4].

EEG is widely used in brain function studies. Recently, many studies [5, 7, 8] have demonstrated the relationship between depression and EEG. Many studies [7, 9, 10, 11, 12, 14] showed asymmetries in the EEG of depressed patients over the frontal cortex. Moreover, the EEG showed significant differences between healthy subjects and depressed patients in many research studies [6, 14, 15, 16].

Research on the human brain is being carried out intensively to understand the mechanisms underlying depression. The most commonly used diagnosis of depression is a scale-based interview with a psychologist or psychiatrist. Current depression detection methods are labor intensive and results depend on the doctor's experience. As a result, many patients with depression are not accurately diagnosed and do not receive optimal treatment. Therefore, finding appropriate and effective methods for detecting depression is an important research topic. With the latest advances in technology, the use of physiological data for the diagnosis of mental disorders opens a new avenue for an objective and accurate tool for detecting depression. Among all kinds of physiological data, electroencephalogram (EEG) reflects emotional human brain activity in real time [3].

In Article 4, recent studies on computer-aided diagnosis (CAD) of depression using EEG signals are presented. Chaos theory and nonlinear dynamical methods are used to extract the bispectrum, power spectrum, phase entropies, wavelet energy and entropy, correlation dimension, fractal dimension, largest Lyapunov entropy, and approximate entropy [4].

Ahmadlou et al. [17] investigated EEGs obtained from patients with major depressive disorder using wavelet-chaos methodology.

Puthankattil et al. [18] extracted relative wavelet energy parameters from the discrete wavelet transform (DWT) coefficients and used ANN to classify the EEG signal into normal and depressed classes. Ahmadlou et al. [19] presented a novel nonlinear method for the analysis of brain dynamics.

Faust et al. [20] present a depression diagnosis support system using entropies extracted from the wavelet packet decomposition coefficients of the EEG signal.

Bachmann et al. [21] compared the linear and nonlinear methods for depression detection based on EEG signals. Even though a number of papers have been published using nonlinear methods, there are other nonlinear methods [21–46] that are worth exploring for the EEG-based diagnosis of depression.

Sleep disturbances are common in depression. It has been estimated that more than 90% of patients with major depressive disorder have concurrent sleep problems [47]–[49]. Sleep problems may manifest as insomnia, hypersomnia, early morning awakenings, frightening dreams, or excessive sleepiness in the daytime in depressive patients [50]. Soehner et al. have found that the presence of insomnia is associated with more severe depression [51]. It has been observed that chronic sleep disturbances are associated with the recurrence of depressive episodes and suicide [52–53]. In polysomnographic examinations, it is observed that more time is needed to fall asleep, there is a shortened REM latency and a longer duration of the first REM period, there is increased REM intensity during sleep, there is less slow-wave sleep, and there is increased wakefulness during sleep in depressive patients [54]–[55]. Armitage et al. [56] have reported that there was a difference in terms of wave amounts of fast and slow frequencies between the sleep EEGs of depressed and healthy subjects [54]. Röschke reported that there were differences in theta and beta activity during an NREM sleep period [57].

Figure 1 shows sample normal (a) and depression (b) EEG data.

A number of studies have used autoregressive (AR) models to detect changes in the EEG signal [58–60]. Based on the AR model parameters, Itakura distance has been used for automatic sleep stage classification effectively [58]. The Itakura distance measure used for such quantification requires the same order for the AR models in all EEG segments. In their pilot study, Estrada et al. investigated EEG and EOG signals acquired from 10 sleep apnea patients undergoing overnight polysomnography and used the Itakura distance to classify sleep stages [58].

To our knowledge, no previous study has investigated the structural differences of sleep EEGs with the Itakura Distance between depressive patients and healthy subjects. In this preliminary study, we aimed to clarify more specific points in EEG signals by comparing signals between depressive patients and healthy subjects. To do this, EEG signals from every different sleep stage are isolated and compared with each other. This study aims to research the structural differences between healthy subjects and subjects with depressive disorder by using the Itakura Distance Measure.

MATERIALS AND METHODS

A- Subjects: The study group consists of two male subjects (H1 and H2) (ages 23–24) with major depressive disorder and two male subjects (D1 and D2) (ages 23) without any psychiatric complaints or findings who were under polysomnographic examination in the Sleep Research Center of Gülhane Training and Research Hospital Department of Psychiatry. The subjects were not on any medication. In sleep records, a polysomnographer named Somnostar Alpha is used. The polysomnographer uses a 0.5–35 Hz filter and takes samples at 200 Hz (low-Hz
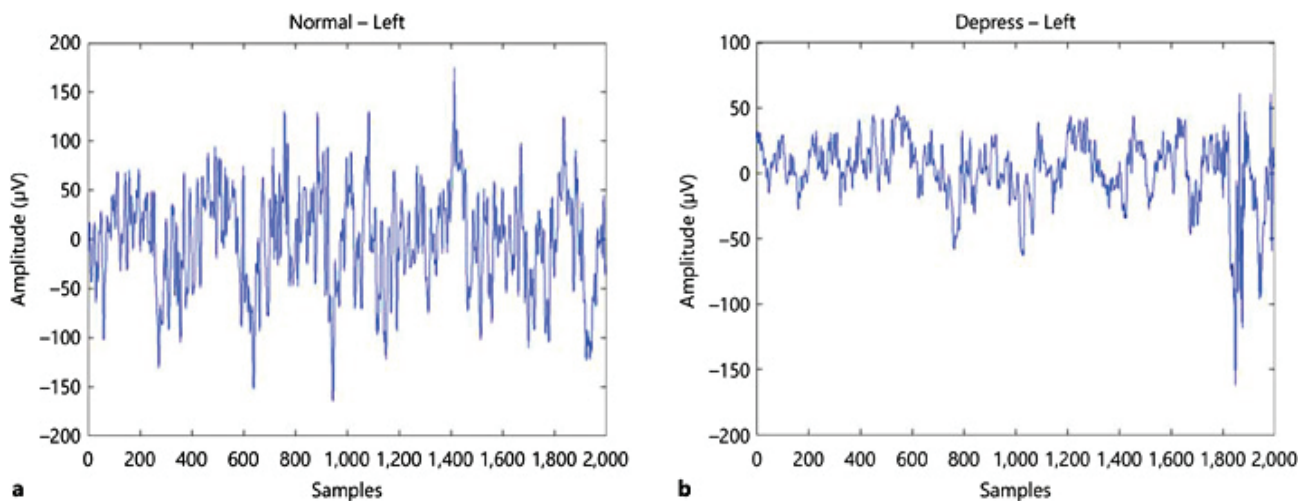

Figure 1. Sample normal and depression EEG data.

filter [Hz]: 0.5; high-Hz filter [Hz]: 35; sampling rate: 200 Hz). The EEG record obtained from the polysomnographer is evaluated using Matlab software (The Mathworks, Massachusetts, USA).

B- Polysomnographic examination: In polysomnographic records, two EEGs (C3 and A2, C4 and A1), two EOGs (left and right), two EMGs (mental or submental and tibial muscular), and an ECG are used. EEG records were evaluated and scored by a psychiatrist. The first night is regarded as adaptation night. Each polysomnographic examination is scored in epochs lasting 30 seconds, which consist of 6000 spots. [1] The Itakura distance was calculated by using the epoch corresponding to the first N1 of H1 and the epoch corresponding to the first N1 of H2. Later, the calculation of the Itakura distance was done by comparing the second corresponding N1 epoch of H1 with the second N1 epoch of H2. The calculated Itakura distances were graphed and interpreted visually. This idea was used separately for all other phases, and the Itakura distances were calculated for each phase and presented in a graphic form. Epochs with artifacts were not evaluated. The C3-A2 record taken on the second night is used for sleep EEG data.

C- Statistical method: In parametric modeling, a mathematical model is fitted to a sampled signal in order to study a time series. The AR modeling technique can be formulated either in the frequency domain as a spectral matching problem or in the time domain as a linear prediction problem. The code written in MATLAB was developed to calculate the Itakura distance by the researchers in the study.

Statistical Analysis

There are two methods to quantify the differences between the two waveforms. The first of these is a spectral distance measure, which measures the difference between the power spectra of two waveforms. The second method is called the Itakura distance measure, which measures the distance between the AR parameter vectors of the two waveforms. This method has been successfully applied in speech processing applications to measure the distance between two speech utterances modeled as AR processes [61]. A more extensive account of these and other distance measures can be found in [62].

Itakura distance measure

Let the AR model of a segment of baseline EEG be given by:

$$x(t) = a_{1t}x(t-1) + a_{2t}x(t-2) + ... + a_{pt}x(t-p) + e(t) \quad (1)$$

and that of a segment of EEG during the experiment be given by:

$$y(t) = b_{1t}y(t-1) + b_{2t}y(t-2) + ... + b_{pt}y(t-p) + v(t) \quad (2)$$

Here, we assume that the optimal order for both segments of EEG is p. Let the optimal AR parameter vector in

each case be given by, $\alpha = (1, a_1, ..., a_p)$ and $\beta = (1, b_1, ..., b_p)$, respectively. The Itakura distance measure to test how far β is from optimality if it is used to model $x(t)$ is defined as,

$$d_I(\alpha, \beta) = \log \frac{\beta^T R_x \beta}{\alpha^T R_x \alpha} \quad (3)$$

[61] where R_x is the autocorrelation matrix of $x(t)$ at given by:

$$R_x = \begin{bmatrix} r_x(0) & r_x(1) & ... & r_x(p) \\ r_x(1) & r_x(0) & ... & r_x(p-1) \\ \vdots & \vdots & \vdots & \vdots \\ r_x(p) & r_x(p-1) & ... & r_x(0) \end{bmatrix} \quad (4)$$

The individual elements of the above matrix denote the autocorrelation of $x(t)$ at different lags, and they can be estimated as follows:

$$r_x(k) = \frac{1}{N} \sum_{n=1}^{N-k} x(n)x(n+k), \quad k = 0, 1, 2, ..., p \quad (5)$$

Similarly, the AR parameter vector α can be tested to see how well models work $y(t)$ by defining another Itakura distance measure given by

$$d_I(\beta, \alpha) = \log \frac{\alpha^T R_y \alpha}{\beta^T R_y \beta} \quad (6)$$

A symmetric distance measure can then be defined by combining the above two distance measures as follows:

$$d_I^{'}(\alpha, \beta) = \frac{1}{2}\left[d_I(\alpha, \beta) + d_I(\beta, \alpha)\right] \quad (7)$$

[61-63].

In this study, each sleep EEG epoch was modeled with the AR(4) model, and the parameters were estimated based on that (optimal AR model order determination using the Akaike Information Criterion (AIC)).

The parameters in the AR model were considered to be the parameters that changed over time and were estimated by the Kalman filter. Using a time-varying autoregressive model, we take advantage of the fact that the EEG signals are not always the same. The time-varying coefficient of the AR model is estimated using the Kalman filter (see Appendix).

Two healthy subjects and two subjects with depression have been studied. Each epoch is compared with the same-numbered epoch of the other patient. Using the Itakura Distance Measure, it is determined whether there is a structural difference between epochs or not. Itakura

Distance Measure uses AR (4), which is one of the time-series models, and the autocorrelations in each epoch.

According to this distance measure, the comparison of REM epochs for H1 and H2 is shown in Figure 2.

The comparison of the N2 epochs of H1 and H2 is shown in Figure 3.

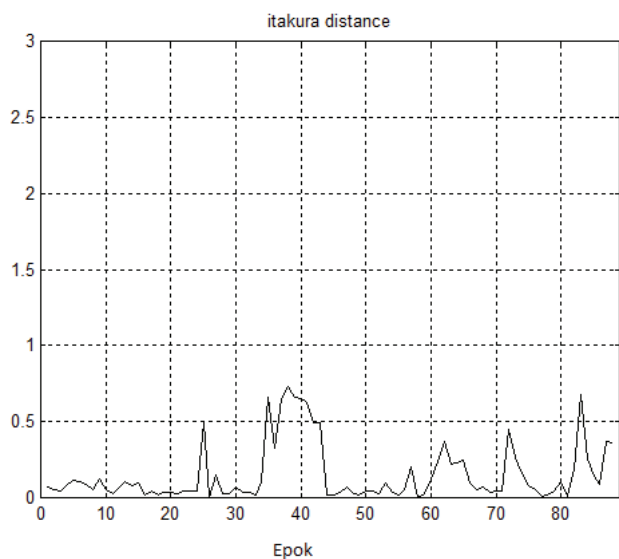The comparison of REM epochs for D1 and H1 is shown in Figure 4.

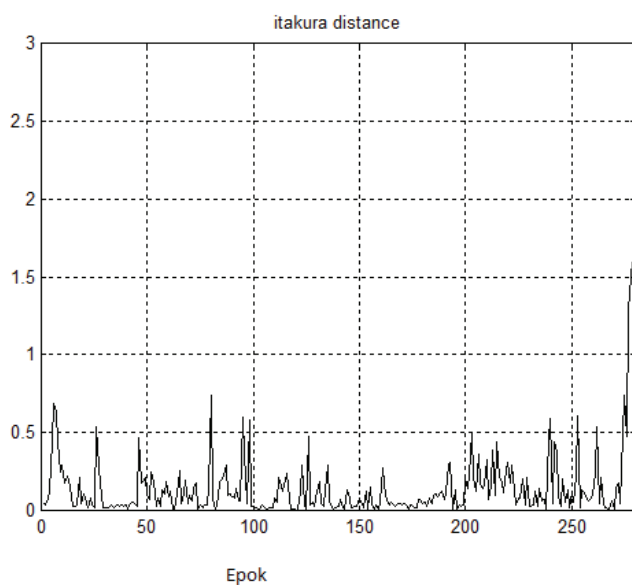**Figure 2.** The comparison between the REM epochs of subjects H1 and H2.

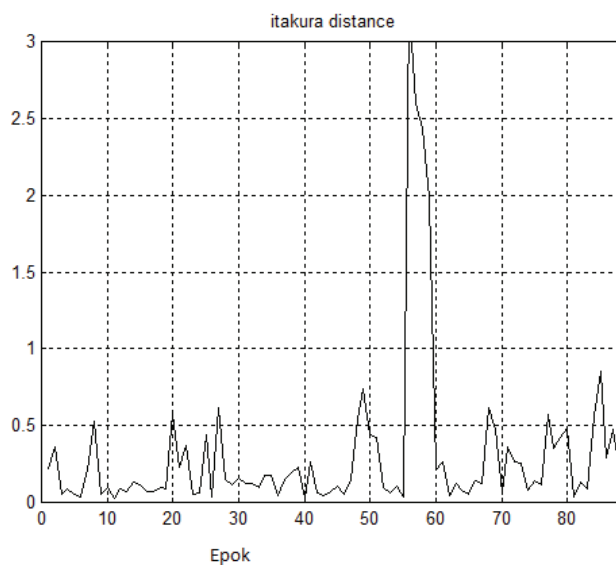**Figure 3.** The comparison between period 2 epochs of the healthy subjects H1 and H2.

Figure 4. The comparison between the REM epochs of subject number one with depressive disorder and the healthy subject number one.
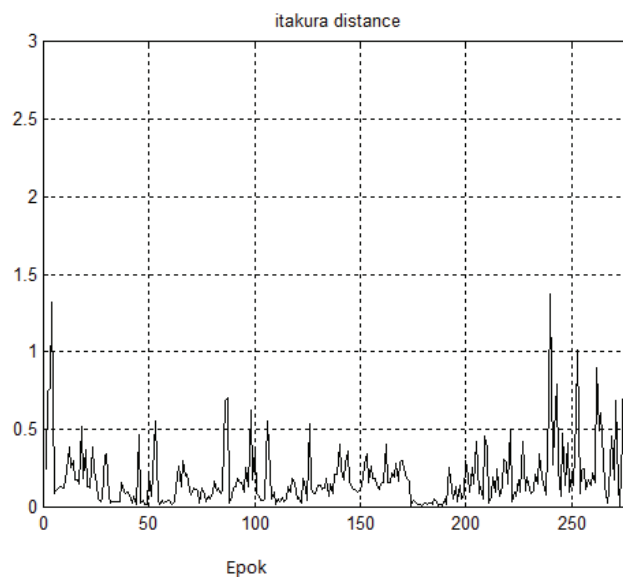

Figure 5. The comparison between period 2 epochs of subject number one with depressive disorder and the healthy subject number one.

The comparison of the N2 epochs of D1 and H1 is shown in Figure 5.

The comparison of the N2 epochs of D1 and H2 is shown in Figure 6.

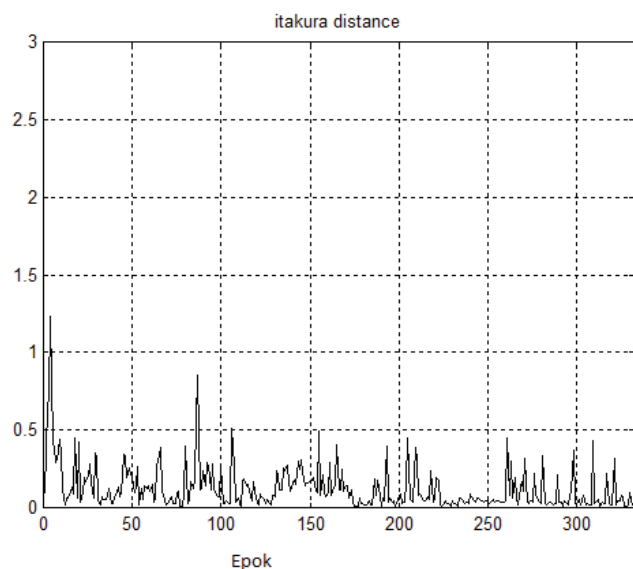The comparison of N2 epochs in D1 and D2 is shown in Figure 7.

Figure 6. The comparison between period 2 epochs of subject number one with depressive disorder and the healthy subject number two.
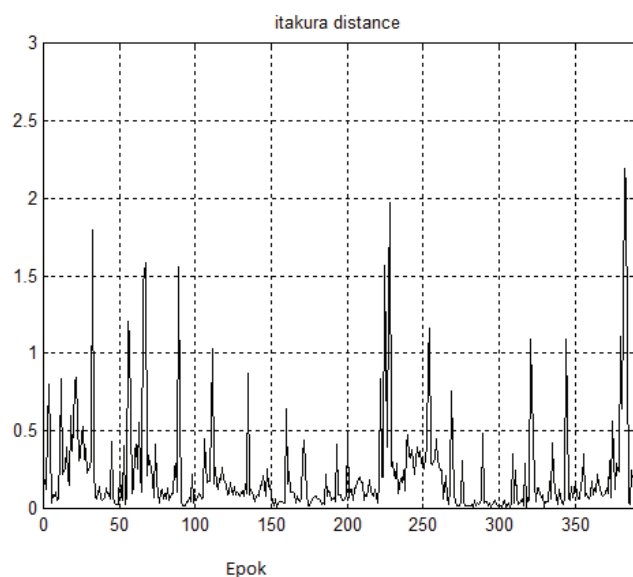

Figure 7. The comparison between period 2 epochs of subjects one and two with depressive disorder.

RESULTS AND DISCUSSION

The current study found that the distance was short when the N2 and REM epochs of the healthy subjects were compared, respectively (Figure 2 and Figure 3). This result shows that these two wave models are structurally similar to each other. The distance has been found to be large when the N2 and REM epochs of depressive subjects are compared with those of healthy subjects (Figures 4 and Figures 5). This result shows that the EEG signals of the subjects in the two groups are structurally different from each other. It is known that sleep disturbances are common in depression patients. It is accepted that approximately 5% of the total sleep time is N1, 50% is N2, and 20% is N3 and N4 sleep. The remaining 25% is REM sleep. Since N2 and REM stages comprise 65–80% of total sleep, they provide important clues in understanding the quality of sleep.

The findings obtained in our study are compatible with the literature. Polysomnographic studies in sleep studies have shown that depressed patients require more time to fall asleep, have a shorter REM latency and a longer first REM period, increased REM intensity in sleep, decreased slow-wave sleep, and increased wakefulness during sleep [54][55]. Armitage et al. [56] found a difference in the amount of waves of fast and slow frequencies between the sleep EEGs of depressed and healthy subjects [54]. Röschke reported differences in theta and beta activity during a NREM sleep period [57].

Patients with depression show characteristic sleep-EEG changes [72], including:

(i) Impaired sleep continuity

(ii) Disinhibition of REM sleep: shortened REM latency or sleep-onset REM periods

(iii) Changes in non-REM sleep (decreased stage N2)

CONCLUSION

The study indicates that the sleep EEGs of the depressed patients differ in N2 and REM stages from those of healthy subjects with the use of the Itakura Distance Measure. This method may be used as an indicator to assist psychiatrists in sleep research and clinical diagnosis. By combining this information with clinical findings, it may be easier for the psychiatrist to make a diagnosis based on objective numerical data.

The major limitation of this study is its small sample size. Further studies with larger sample sizes may reveal structural differences more accurately. This can be an important resource for researchers and psychiatrists evaluating patients in the clinical setting in terms of making objective decisions based on quantitative data.

AUTHORSHIP CONTRIBUTIONS

Authors equally contributed to this work.

DATA AVAILABILITY STATEMENT

The authors confirm that the data that supports the findings of this study are available within the article. Raw data that support the finding of this study are available from the corresponding author, upon reasonable request.

CONFLICT OF INTEREST

The author declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

ETHICS

There are no ethical issues with the publication of this manuscript.

REFERENCE

[1] Aboalayon KAI, Faezipour M, Almuhammadi WS, Moslehpour S. Sleep stage classification using EEG signal analysis: A comprehensive survey and new investigation. Entropy 2016;18:272. [CrossRef]

[2] Patel AK, Reddy V, Shumway KR, Araujo JF. Physiology, sleep stages. Available at: https://www.ncbi.nlm.nih.gov/books/NBK526132/. Accessed on May 13, 2024.

[3] Cai H, Han J, Chen Y, Sha X, Wang Z, Hu B, et al. A pervasive approach to EEG-based depression detection. Complexity 2018;2018:5238028. [CrossRef]

[4] Yasin S, Othmani A, Raza I, Hussain SA. Machine learning based approaches for clinical and non-clinical depression recognition and depression relapse prediction using audiovisual and EEG modalities: A comprehensive review. Comput Biol Med 2023;159:106741. [CrossRef]

[5] Acharya UR, Sudarshan VK, Adeli H, Santhosh J, Koh JE, Adeli A. Computer-aided diagnosis of depression using EEG signals. Eur Neurol 2015;73:329−336. [CrossRef]

[6] Bachmann M, Lass J, Hinrikus H. Single channel EEG analysis for detection of depression. Biomed Signal Process Control 2017;31:391−397. [CrossRef]

[7] Deslandes AC, de Moraes H, Pompeu FA, Ribeiro P, Cagy M, Capitão C, et al. Electroencephalographic frontal asymmetry and depressive symptoms in the elderly. Biol Psychol 2008;79:317−322. [CrossRef]

[8] Hinrikus H, Suhhova A, Bachmann M, Aadamsoo K, Võhma U, Pehlak H, et al. Spectral features of EEG in depression. Biomed Tech (Berl) 2010;55:155−161. [CrossRef]

[9] Debener S, Beauducel A, Nessler D, Brocke B, Heilemann H, Kayser J. Is resting anterior EEG alpha asymmetry a trait marker for depression? Findings for healthy adults and clinically depressed patients. Neuropsychobiology 2000;41:31−37. [CrossRef]

[10] Koles ZJ, Lind JC, Flor-Henry P. A source-imaging (low-resolution electromagnetic tomography) study of the EEGs from unmedicated men with schizophrenia. Psychiatry Res 2004;130:171−190. [CrossRef]

[11] Nissen C, Feige B, Nofzinger EA, Voderholzer U, Berger M, Riemann D. EEG slow wave activity regulation in major depression. Somnologie 2006;10:36−42. [CrossRef]

[12] Hu B, Majoe D, Ratcliffe M, Qi Y, Zhao Q, Peng H, et al. EEG-based cognitive interfaces for ubiquitous applications: Developments and challenges. IEEE Intell Syst 2011;26:46−53. [CrossRef]

[13] Coan JA, Allen JJ. Frontal EEG asymmetry as a moderator and mediator of emotion. Biol Psychol 2004;67:7−49. [CrossRef]

[14] Grin-Yatsenko VA, Baas I, Ponomarev VA, Kropotov JD. Independent component approach to the analysis of EEG recordings at early stages of depressive disorders. Clin Neurophysiol 2010;121:281−289. [CrossRef]

[15] Kwan Y, Baek C, Chung S, Kim TH, Choi S. Resting-state quantitative EEG characteristics of insomniac patients with depression. Int J Psychophysiol 2018;124:26−32. [CrossRef]

[16] Shen J, Zhang X, Wang G, Ding Z, Hu B. An improved empirical mode decomposition of electroencephalogram signals for depression detection. IEEE Trans Affect Comput 2022;13:262−271. [CrossRef]f

[17] Ahmadlou M, Adeli H, Adeli A. Fractality analysis of frontal brain in major depressive disorder. Int J Psychophysiol 2012;85:206−211. [CrossRef]

[18] Puthankattil SD, Joseph PK. Classification of EEG signals in normal and depression conditions by ANN using RWE and signal entropy. J Mech Med Biol 2012;12:1240019. [CrossRef]

[19] Ahmadlou M, Adeli H, Adeli A. Spatiotemporal analysis of relative convergence of EEGs reveals differences between brain dynamics of depressive women and men. Clin EEG Neurosci 2013;44:175−181. [CrossRef]

[20] Faust O, Ang PAC, Puthankattil SD, Joseph PK. Depression diagnosis support system based on EEG signal entropies. J Mech Med Biol 2014;14:1450035. [CrossRef]

[21] Bachmann M, Lass J, Suhhova A, Hinrikus H. Spectral asymmetry and Higuchi›s fractal dimension measures of depression electroencephalogram. Comput Math Methods Med 2013;2013:251638. [CrossRef]

[22] Mandelbrot BB. The Fractal Geometry of Bature. 2nd ed. New York: Times Books; 1982.

[23] Higuchi T. Approach to an irregular time series on the basis of the fractal theory. Phys D: Nonlinear Phenom 1988;31:277−283. [CrossRef]

[24] Marwan N, Thiel M, Nowaczyk NR. Cross recurrence plot based synchronization of time series. Nonlinear Process Geophys 2002;9:325−331. [CrossRef]

[25] Marwan N, Romano MC, Thiel M, Kurths J. Recurrence plots for the analysis of complex systems. Phys Rep 2007;438:237−329. [CrossRef]

[26] Chua KC, Chandran V, Acharya UR, Lim CM. Computer-based analysis of cardiac state using entropies, recurrence plots and Poincare geometry. J Med Eng Technol 2008;32:263−272. [CrossRef]

[27] Chua KC, Chandran V, Acharya UR, Lim CM. Cardiac state diagnosis using higher order spectra of heart rate variability. J Med Eng Technol 2008;32:145−155. [CrossRef]

[28] Chua KC, Chandran V, Acharya UR, Lim CM. Application of higher order spectra to identify epileptic EEG. J Med Syst 2011;35:1563–1571. [CrossRef]

[29] Richman JS, Moorman JR. Physiological time-series analysis using approximate entropy and sample entropy. Am J Physiol Heart Circ Physiol 2000;278:H2039–H2049. [CrossRef]

[30] Zhang C, Wang H, Wang H, Wu MH. EEG-based expert system using complexity measures and probability density function control in alpha sub-band. Integr Comput Aid Eng 2013;20:391–405. [CrossRef]

[31] Pincus SM, Keefe DL. Quantification of hormone pulsatility via an approximate entropy algorithm. Am J Physiol 1992;262:E741–E754. [CrossRef]

[32] Wolf A, Swift JB, Swinney HL, Vastano JA. Determining Lyapunov exponents from a time series. Phys D: Nonlinear Pheno 1985;16:285–317. [CrossRef]

[33] Dangel S, Meier PF, Moser HR, Plibersek S, Shen Y. Time series analysis of sleep EEG. Comput Assist Phys 1999;14:93–95.

[34] Lee JM, Kim DJ, Kim IY, Park KS, Kim SI. Detrended fluctuation analysis of EEG in sleep apnea using MIT/BIH polysomnography data. Comput Biol Med 2002;32:37–47. [CrossRef]

[35] Yuan Q, Zhou W, Yuan S, Li X, Wang J, Jia G. Epileptic EEG classification based on kernel sparse representation. Int J Neural Syst 2014;24:1450015. [CrossRef]

[36] Osorio I. Automated seizure detection using EKG. Int J Neural Syst 2014;24:1450001. [CrossRef]

[37] Acharya UR, SV, Bhat S, Adeli H, Adeli A. Computer-aided diagnosis of alcoholism-related EEG signals. Epilepsy Behav 2014;41:257–263. [CrossRef]

[38] Hu M, Liang H. Perceptual suppression revealed by adaptive multi-scale entropy analysis of local field potential in monkey visual cortex. Int J Neural Syst 2013;23:1350005. [CrossRef]

[39] Acharya UR, Yanti R, Zheng JW, Krishnan MM, Tan JH, Martis RJ, et al. Automated diagnosis of epilepsy using CWT, HOS and texture parameters. Int J Neural Syst 2013;23:1350009. [CrossRef]

[40] Martis RJ, Acharya UR, Lim CM, Mandana KM, Ray AK, Chakraborty C. Application of higher order cumulant features for cardiac health diagnosis using ECG signals. Int J Neural Syst 2013;23:1350014. [CrossRef]

[41] Rodríguez-Bermúdez G, García-Laencina PJ, Roca-Dorda J. Efficient automatic selection and combination of EEG features in least squares classifiers for motor imagery brain-computer interfaces. Int J Neural Syst 2013;23:1350015. [CrossRef]

[42] Lin LC, Ouyang CS, Chiang CT, Yang RC, Wu RC, Wu HC. Early prediction of medication refractoriness in children with idiopathic epilepsy based on scalp EEG analysis. Int J Neural Syst 2014;24:1450023. [CrossRef]

[43] Koppert M, Kalitzin S, Velis D, Lopes da Silva F, Viergever MA. Dynamics of collective multi-stability in models of multi-unit neuronal systems. Int J Neural Syst 2014;24:1430004. [CrossRef]

[44] Cong F, Phan AH, Astikainen P, Zhao Q, Wu Q, Hietanen JK, et al. Multi-domain feature extraction for small event-related potentials through nonnegative multi-way array decomposition from low dense array EEG. Int J Neural Syst 2013;23:1350006. [CrossRef]

[45] Fu R, Wang H. Detection of driving fatigue by using noncontact EMG and ECG signals measurement system. Int J Neural Syst 2014;24:1450006. [CrossRef]

[46] Bauer PR, Kalitzin S, Zijlmans M, Sander JW, Visser GH. Cortical excitability as a potential clinical marker of epilepsy: A review of the clinical application of transcranial magnetic stimulation. Int J Neural Syst 2014;24:1430001. [CrossRef]

[47] Aydin H, Ozgen F. Effect of imipramine on REM: Paradoxical or parallel? Eur Neuropsychopharmocol 1992;2:389–391. [CrossRef]

[48] Ford DE, Cooper-Patrick L. Sleep disturbances and mood disorders: an epidemiologic perspective. Depress Anxiety 2001;14:3–6. [CrossRef]

[49] Spiegelhalder K, Regen W, Nanovska S, Baglioni C, Riemann D. Comorbid sleep disorders in neuropsychiatric disorders across the life cycle. Curr Psychiatry Rep 2013;15:364. [CrossRef]

[50] Luca A, Luca M, Calandra C. Sleep disorders and depression: Brief review of the literature, case report, and nonpharmacologic interventions for depression. Clin Interv Aging 2013;8:1033–1039. [CrossRef]

[51] Soehner AM, Kaplan KA, Harvey AG. Prevalence and clinical correlates of co-occurring insomnia and hypersomnia symptoms in depression. J Affect Disord 2014;167:93–97. [CrossRef]

[52] Fawcett J, Scheftner WA, Fogg L, Clark DC, Young MA, Hedeker D, et al. Time-related predictors of suicide in major affective disorder. Am J Psychiatry 1990;147:1189-1194. [CrossRef]

[53] Malik S, Kanwar A, Sim LA, Prokop LJ, Wang Z, Benkhadra K, et al. The association between sleep disturbances and suicidal behaviors in patients with psychiatric diagnoses: A systematic review and meta-analysis. Syst Rev 2014;3:18. [CrossRef]

[54] Armitage R. Microarchitectural findings in sleep EEG in depression: Diagnostic implications. Biol Psychiatry 1995;37:72–84. [CrossRef]

[55] Ozgen F, Aydin H, Gulcat Z. Sleep pattern in major depressives. J Sleep Res 2000;9:145.

[56] Armitage R, Roffwarg HP, Rush AJ, Calhoun JS, Purdy DG, Giles DE. Digital period analysis of sleep EEG in depression. Biol Psychiatry 1992;31:52–68. [CrossRef]

[57] Röschke J, Mann K. The sleep EEG's microstructure in depression: Alterations of the phase relations between EEG rhythms during REM and NREM sleep. Sleep Med 2002;3:501–505. [CrossRef]

[58] Estrada E, Nava P, Nazeran H, Behbehani K, Burk J, Lucas E. Itakura distance: A useful similarity measure between EEG and EOG signals in computer-aided classification of sleep stages. Conf Proc IEEE Eng Med Biol Soc 2005;2005:1189−1192. [CrossRef]

[59] Eva OD, Lazar AM, Fira M. Normalized Itakura distance based discrimination used in a motor imagery brain computer interface paradigm. Available at: http://www.bulipi-eee.tuiasi.ro/archive/2015/fasc.4/p8_f4.pdf. Accessed on May 13, 2024.

[60] Gharbali AA, Najdi S, Fonseca JM. Investigating the contribution of distance-based features to automatic sleep stage classification. Comput Biol Med 2018;96:8−23. [CrossRef]

[61] Itakura F. Minimum prediction residual principle applied to speech recognition. IEEE Trans Acoust 1975;23:67−72. [CrossRef]

[62] Basseville M. Distance measures for signal processing and pattern recognition. Signal Process 1989;18:349−369. [CrossRef]

[63] Muthuswamy J, Thakor NV. Spectral analysis methods for neurological signals. J Neurosci Methods 1998;83:1−14. [CrossRef]

[64] Oikonomou VP, Tzallas AT, Fotiadis DI. A Kalman filter based methodology for EEG spike enhancement. Comput Methods Programs Biomed 2007;85:101−108. [CrossRef]

[65] Pardey J, Roberts S, Tarassenko L. A review of parametric modelling techniques for EEG analysis. Med Eng Phys 1996;18:2−11. [CrossRef]

[66] Penny WD, Roberts SJ. Dynamic models for nonstationary signal segmentation. Comput Biomed Res 1999;32:483−502. [CrossRef]

[67] Olbrich E, Achermann P, Meier PF. Dynamics of human sleep EEG. Neurocomput 2003;52:857−862. [CrossRef]

[68] Ozbek L, Sutcigil L, Aydın H, Yetkin S, Ozgen F. A statistical overview on sleep scoring. Ankara Univ Fac Sci Commun 2020;62;115−122. [CrossRef]

[69] Ozbek L. Kalman Filtresi. 1st ed. Ankara: Akademisyen Kitabevi; 2017. [CrossRef]

[70] Aliev FA, Ozbek L. Evaluation of convergence rate in the central limit theorem for the Kalman filter. IEEE Trans Autom Control 1990;44:1905−1909. [CrossRef]

[71] Ozbek L, Efe M. An adaptive extended Kalman filter with application to compartment models. Commun Stat Simul Comput 2007;33:145−158. [CrossRef]

[72] Steiger A, Pawlowski M. Depression and sleep. Int J Mol Sci 2019;20:607. [CrossRef]

Appendix

Let y be the one-channel EEG signal. We suppose that an AR can describe the EEG. The AR model has found many applications in EEG analysis, although EEG is a non-stationary signal [64]–[68]. The AR model is given as:

$$y(t) = b_1 y(t-1) + b_2 y(t-2) + ... + b_p y(t-p) + v(t)$$

where p is the order of the model. Since the EEG is a non-stationary signal, we let the AR parameter vary with time.

$$y(t) = b_{1t} y(t-1) + b_{2t} y(t-2) + ... + b_{pt} y(t-p) + v(t)$$

or in vector notation

$$y(t) = \varphi(t)^T \theta(t) + v(t)$$

where $\phi(t) = [y(t-1), y(t-2),..., y(t-p)]^T$ is a px1 vector and $\theta(t) = [b_{1t}, b_{2t},..., b_{pt}]^T$. The vector $\theta(t)$ contains the AR parameters and varies in time:

$$\theta(t) = \theta(t-1) + w(t)$$

where $w(t)$ is Gaussian noise with zero mean and covariance $R_1(t)$. This describes an AR model for the EEG signal with a time-varying coefficient in state-space form. To estimate those coefficients, we use the KF. Consider the dynamical linear model given as:

$$\theta(t) = \theta(t-1) + w(t)$$

$$y(t) = \varphi(t)^T \theta(t) + v(t)$$

where $\theta(t)$ is a px1 state vector at time t, $y(t)$ is an observation at time t, and $w(t)$ and $v(t)$ are the states and observation noise which are assumed white and Gaussian, respectively. The KF is given with the following equations:

$$K(t) = [P(t|t-1)\varphi(t)][\varphi(t)^T P(t|t-1)\varphi(t) + R_2(t)] \tag{A3}$$

$$P(t|t-1) = P(t-1|t-1) + R_1(t) \tag{A4}$$

$$e(t) = y(t) - \hat{y}(t) \tag{A5}$$

$$\hat{y}(t) = \varphi(t)^T \hat{\theta}(t|t-1) \tag{A6}$$

$$\hat{\theta}(t) = \hat{\theta}(t|t-1) + K(t)e(t) \tag{A7}$$

$$P(t|t) = P(t|t-1) - K(t)\varphi(t)^T P(t|t-1) \tag{A8}$$

where $K(t)$ Kalman gain [69-71].