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## Study of Electroencephalographic Patterns in Segmental and Non-segmental Vitiligo Patients: An Open Randomized Study

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ABSTRACT

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### Background: The course of vitiligo is unpredictable because it is an acquired condition. Clinically, it is indicated by clearly defined depigmented macules **Article information** that result from malfunction and loss of melanocytes. 09-09-2023 **Received:** Aim of the work: To study electroencephalographic patterns in segmental and non-segmental vitiligo patients to record any changes in the electroencephalographic activity as related to the stress condition of the 08-10-2023 Accepted: brain in vitiligo patients, an open randomised study. Patients and Methods: This was a cross-sectional study that included 30 DOI: vitiligo patients considered the patient group as well as another 30 age-10.21608/IJMA.2023.235277.1806. matched healthy people considered the control group. EEG was applied once to all participants. EEG recordings were used to quantify brain activity. \*Corresponding author The new data point of 1.6 Hz was estimated using a linear interpolation between the two discrete data points [1.5 and 2 Hz] with a window length of Email: 2 s and a 0.1 Hz increment. mohamed.mahmoud15891@gmail.com **Results:** The most common type of vitiligo was the progressive non-segmental Citation: Mohamed MMM, Ezz-Elddin one [93.3%]. The initial lesion appeared more frequently on the face and SM, Elrewiny EM, Hassan MAS. neck [60%], acral [40%], limb [36.7%], and trunk [26.7%]. The frequency Study of Electroencephalographic of EEG waves was significantly higher in the patients' group than in the Patterns in Segmental and Noncontrol group [p<0.001]. Alpha, C3, 8.0-12.0 Hz, alpha, C4, 8.0-12.0 Hz, segmental Vitiligo Patients: An Open beta 2, C3, 20.0-34.0 Hz, and beta 2 waves, C4.20.0-34.0 Hz, were Randomized Study. IJMA 2023 significantly higher in the patient's group [P<0.001]. There were significant September; 5 [9]: 3590-3600. doi: differences observed in the beta 1 waves [C3 and C4] [p = 0.0385 and 10.21608/IJMA.2023.235277.1806. 0.0148, respectively]. Conclusion: The high frequency of alpha and beta waves indicates the presence of stress among vitiligo patients. Stress from metabolic and psychological factors may affect vitiligo patients' susceptibility to the disease's progression. Additional research is needed on lifestyle elements that affect vitiligo, including health behaviors, mental makeup, and the effect of social life on individuals and groups.

Keywords: Electroencephalography; Segmental; Non-segmental; Vitiligo.



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### **INTRODUCTION**

Vitiligo is an acquired disease with a variable course. From a clinical perspective, it is indicated by clearly defined depigmented macules that result from melanocyte malfunction and loss. It is the most prevalent depigmentation condition, influencing 0.5 to 2.0% of people worldwide, and is not gender- or race-specific <sup>[1]</sup>.

Segmental subtypes of vitiligo, which only affect a small proportion of people [5-16%], are distinguished from non-segmental subtypes [NSV]. By subtype, the disease's onset and progression may differ. Additionally, vitiligo patients may face severe psychosocial symptoms such as depression and inadequate self-esteem <sup>[2]</sup>.

Genetic factors, oxidative stress-induced melanocyte destruction, defective intracellular biochemical signal pathways, autoimmune processes, a deficiency in melanocyte adhesion, and nervous system abnormalities are only a few of the pathogenic causes that are likely multifactorial <sup>[3]</sup>.

Certain neuroinflammatory manifestations in peripheral tissues, such as the skin, can be brought on by or made worse by stress. In fact, stress can start or worsen a variety of skin conditions, including atopic dermatitis, psoriasis, urticaria, and partial or total baldness. Just recently, the specific mechanism behind stressrelated or worsened skin conditions has been understood. As a result, current experimental data suggests that the brain and skin share a number of neuroendocrine mechanisms. This alleged "brain-skin connection" is an intriguing new field of study <sup>[4]</sup>.

Due to their same embryonic ancestor, the ectoderm, the skin, and the brain are physically and functionally interconnected in both directions [the skin-brain axis and the brain-skin axis]<sup>[5]</sup>.

The postcentral gyrus receives somatosensory impulses from the skin, and the sensory homunculus there represents the entire surface of the body. These inputs trigger endocrine, immunological, neurovegetative, emotional, and behavioral responses. Cutaneous impulses may possibly have a significant impact on how the brain develops. For instance, while C-tactile afferents exercise neural pathways involved in social-emotional learning, consistent gentle touch throughout childhood might be necessary for the growth of a social brain <sup>[6]</sup>. The hypothalamic-pituitary-adrenal [HPA] axis and the autonomic nervous system are two physiological mechanisms via which the brain affects the skin <sup>[7]</sup>.

In a healthy state, HPA axis hormones help maintain skin homeostasis, a balance between pro- and anti-inflammatory responses, and antibacterial defenses. The HPA axis, nevertheless, could have changed in response to stress, causing pro-inflammatory hormonal effects, tissue receptors that are resistant to glucocorticoids, and mast cell activation in the skin<sup>[8]</sup>.

The neurological theory has grown to be a significant part of vitiligo, and serum IFN $\alpha$  levels were raised in depressed vitiligo patients. In order to stop melanocytosis, central IFN $\alpha$  causes the expulsion of substance P [SP] from the dorsal root ganglion [DRG]. The CNS microenvironment is disturbed by peripheral IFN $\alpha$ . The hypothalamic-pituitary-adreno-cortical [HPA] axis is the key neural system pathway through which IFN $\alpha$  mediates depression <sup>[9]</sup>.

The gold standard for analyzing the electrophysiological mechanisms associated with epilepsy as well as many other central nervous system disorders is electroencephalography, which enables a functional evaluation of electrical brain cortical activity. Although morphological imaging produces supplementary information, it is unable to take the place of the EEG, the fundamental tool for functional analysis. EEG provides the additional great benefits of being non-invasive, simple to use, and enabling control tests when follow-up is required, even at the patient's bedside <sup>[10]</sup>.

It seems that this is the first time to correlate skin disease with brain function and to study the pattern of electroencephalogram in a skin disease to disclose the relation between the brain and skin, especially as the two organs are originating from the same embryological origin.

### **PATIENTS AND METHODS**

### Study design

This was a cross sectional study.

### Sample size

The study included 30 vitiligo patients considered the patient group as well as another

30 age-matched healthy people considered the control group.

### Site of data collection

Vitiligo patients attending the Dermatology Outpatient Clinic at Al-Hussein University Hospital.

### Patients

Thirty patients with vitiligo who were recruited from the dermatology clinic and 30 other controls were recruited from hospital medical records of healthy patients who attended to perform EEG for investigation.

### **Inclusion criteria**

**Patients group:** Patients with segmental and non-segmental vitiligo, patients from both genders [males and females], and age groups of 18–45 years old were included in the study.

**Control group:** The control group consisted of 30 age-matched healthy participants.

### **Exclusion criteria**

Patients having EEG changes due to other causes, pregnant and lactating females, patients with a present or past history of epilepsy, children and elders, and active and passive smokers were excluded from the study. The study was disqualified due to patients with a history of intense activity outside of regular activities, concurrent dermatological illnesses, pregnancy, or chronic renal, liver, or cardiovascular disease history.

### Methods

All patients were subjected to demographic data [gender and age], full history taking [disease duration, clinical type, course participation factor, family history of the disease, and history of recurrence], and a thorough general examination.

Dermatological examination was done by Wood's light for the presence of lesions and their sites [face/neck, acral, limb, or trunk].

Investigation with electroencephalography [EEG] was applied once to all participants.

EEG recordings were used to evaluate brain function by trained neurophysiologists in a quiet

room with closed windows to reduce noise from outside the building. For 8 minutes, it was captured while the individuals were lying down on a comfy chair and closing their eyes.

The neurophysiologist used low auditory input to keep the participants awake while they were about to nod off after being told to stay awake <sup>[11]</sup>.

19 bridge silver and silver chloride electrodes were mounted on a plastic prewired head cap and applied to the patients' scalps using the 10-20 system [namely: Fp1, Fp2, F3, F4, F7, F8, Fz, C3, C4, Cz, P3, P4, Pz, T3, T4, T5, T6, O1, and O2], using an electroconductive material put on the skin between the electrodes to provide a good connection [Micromed System Plus Electroencephalograph, Galileo].

In order to remove frequencies beyond the range of interest, the signals were sampled at 512 Hz and then filtered using an offline first-order zero-phase Butterworth band-pass filter with cutoff frequencies set, respectively, at 1.6 and 70 Hz.

Following manual removal of any parts of the signal impacted by ocular, muscular, or other sorts of artefacts and independent component evaluation, power line noise was eliminated using a 50 Hz notch filter. Following this pre-processing, spectral and connectivity studies were used to obtain the primary EEG rhythms and prominent frequencies.

The following frequency ranges were taken into consideration: gamma 1 [30-50 Hz], gamma 2 [50-70 Hz], theta [4-8 Hz], alpha [8-13 Hz], beta [13-30 Hz], and delta [1.6-4 Hz]. The 1.5 and 2 Hz data points had been recorded since 0.5 Hz increment bins were taken into account for all bands; however, the delta rate band's lower cutoff was 1.6 Hz <sup>[11, 12]</sup>. The new data point of 1.6 Hz was then estimated by performing a linear interpolation between the two discrete data points [1.5 and 2 Hz] with a window length of 2 s and a 0.1 Hz increment.

### **Ethical considerations**

Oral and written informed consent was obtained from every person participating in the study. Ethical approval was obtained from the Ethical Review Committee of the Faculty of Medicine, Al-Azhar University, Cairo, Egypt.

### Statistical analysis

A statistical study was conducted using IBM's SPSS version 26.0. For quantitative elements, descriptive data were provided as means  $\pm$  SD, and for qualitative parameters, as percentages. The Student's t-test and the Mann-Whitney U-test were employed for the evaluations of the quantitative parameters with a distribution and an asymmetric normal distribution, respectively. Based on the applicable statistical principles, the Fisher test, or  $\chi^2$  test, was applied for the qualitative parameters.

### RESULTS

# Demographics, history and clinical characteristics of the studied patients [n=30]

The study included 30 patients suffering from segmental and non-segmental vitiligo and 30 control participants. More than half of patients [56.7%] were females, and 43.3% were males. The mean age of the patient group was 27.9  $\pm$  9.27, ranging from 18 to 45 years, and the mean age of the controls was 27.3  $\pm$  6.55, ranging from 18 to 45 years old [Table 1].

# Vitiligo characteristics and history among the studied patients [n=30]

The mean duration of the disease was  $5.3 \pm 3.6$  years, and most participants [73.3%] suffered from the disease for a duration of more than 2 years. All cases experienced a rapid spread of the disease. The most common type of vitiligo was the progressive non-segmental one [93.3%]. 20% of participants had a positive family history of Vitiligo, and 40% had a history of recurrence of the disease [Table 2].

### **Dermatological examination**

Table [3] indicated the dermatological examination of the studied patients according to the lesion site. The initial lesion appeared more frequently on the face and neck [60%], acral [40%], limb [36.7%], and trunk [26.7%].

### Investigation using Electroencephalography [EEG]

Figures [1] and [2] showed the frequency of alpha and beta waves in patients and control groups.

### **History of Treatment**

Table [4] showed the treatment options for vitiligo patients. The majority of patients [73.3%] were treated by topical combined therapy, followed by narrow-band ultraviolet B NB-UVB [43.3%] and excimer [43.3%]. Vitamin D3 was received by 10% of patients. Systematic steroids were received by 16.7% of patients.

### **EEG findings**

Table [5] showed that the results in the tested groups differed in a statistically significant manner in many dimensions. The frequency of EEG waves was significantly higher in the patients' group than in the control group [p<0.001]. Alpha, C3, 8.0-12.0 Hz, alpha, C4, 8.0-12.0 Hz, beta 2, C3, 20.0-34.0 Hz, and beta 2 waves, C4.20.0-34.0 Hz, were significantly higher in the patient's group [P<0.001]. There were significant differences observed in the beta 1 waves [C3 and C4] [p = 0.0385 and 0.0148, respectively] [Figures 3 and 4].

Variable	Parameter	Patients group [n=30]	Control group	p-value
Demographic characteristics				
Gender, n [%]	Male	13 [43.3%]	12 [40%]	0.611
	Female	17 [56.7%]	18 [60%]	0.011
Age	Mean $\pm$ SD	$27.9 \pm 9.27$	$27.3\pm6.55$	0.707
	Median [IQR]	23.5 [18-45]	23.19 [18-45]	0.707

 Table [1]: Demographics, history and clinical characteristics of the studied patients [n=30]

Vitiligo characteristics	Parameters	N=30	p-value	
Disease duration [years], n [%]	$\leq 2$ years	8 [26.7%]		
	> 2 years	22 [73.3%]	0.03	
	Mean $\pm$ SD	$5.3 \pm 3.6$	0.05	
	Median [IQR]	4 [1-15]		
Onset of the disease, n [%]	Rapid	30 [100%]		
	Slow	0 [0%]	-	
Clinical type, n [%]	Segmental	2 [6.7%]	0.01	
	Non-segmental	28 [93.3%]	0.01	
Course, n [%]	Progressive	28 [93.3%]		
	Stable	2 [6.7%]	0.01	
Stress as a participating factor, n [%]	Yes	30 [100%]		
Family history	Yes	6 [20%]	0.021	
	No	14 [46.7%]	0.021	
History of recurrence	Yes	12 [40%]	0.047	
	No	18 [60%]	0.047	

Table [2]: Vitiligo characteristics and history among the studied patients

 Table [3]: Lesion sites among the studied patients

Variable	Parameter	N [%]
Lesion sites	Face/neck	18 [60%]
	Acral	12 [40%]
	Limb	11 [36.7%]
	Trunk	8 [26.7%]

### Table [4]: Treatment options among the studied patients

Variable		Parameter	N [%]
Treatment option	Topical	Combined	22 [73.3%]
		NUVB	13 [43.3%]
		Excimer	13 [43.3%]
		Vit. D3 alone	3 [10%]
		TCI [topical calcineurin	0 [0%]
		inhibitor] alone	
		Steroid alone	0 [0%]
	Systemic steroid		5 [16.7%]

**Table [5]:** Comparison between EEG wave frequencies and amplitudes in patients and control groups

Variable	Parameter	Patients [n=30]	Control [n=30]	p-value
Frequency of EEG waves	Mean $\pm$ SD	$20.34\pm2.838$	$9.19\pm2.20$	
	Median	20.65	9.33	<0.001*
	Quartiles	14.2 - 24.1	6.31-10.24	
Alpha, C3, 8.0–12.0 Hz	Mean $\pm$ SD	$10.06\pm0.637$	$5.93 \pm 0.22$	
	Median	10.085	5.93	<0.001*
	Quartiles	8.84-11.34	5.43-6.13	
Alpha, C4, 8.0–12.0 Hz	Mean $\pm$ SD	$10.34\pm0.359$	$5.54\pm0.16$	
	Median	10.325	5.51	<0.001*
	Quartiles	9.46-10.91	5.29-5.83	
Beta 1, C3, 15.0–20.0 Hz	Mean $\pm$ SD	$4.626\pm0.182$	$4.61\pm0.11$	
	Median	4.645	4.58	0.0385
	Quartiles	4.31-4.95	4.56-5.01	
Beta 1, C4, 15.0–20.0 Hz	Mean $\pm$ SD	$4.764\pm0.59$	$4.35\pm0.14$	
	Median	4.725	4.38	0.0148
	Quartiles	4.25-4.98	4.01-4.51	
Beta 2, C3, 20.0–34.0 Hz	Mean $\pm$ SD	$10.788 \pm 0.47$	3.98±0.02	
	Median	10.655	3.98	<0.001*
	Quartiles	9.59–11.78	3.96-4.01	
Beta 2, C4, 20.0–34.0 Hz	Mean $\pm$ SD	$11.54 \pm 0.81$	$4.31\pm0.16$	
	Median	11.79	4.35	<0.001*
	Quartiles	9.91-12.65	3.99-4.51	



Figure [1]: EEG recording performed on an 18 years old male vitiligo patient. the High-beta wave's relative wave frequency, which ranges from two to four minutes. The greatest scores were seen in the temporal lobe. There was a distinction between C3 and C4 in the left temporal lobe particularly. Strong responses were seen on each side of the temporal lobe in C4. Therefore, it is assumed that the variations in wave frequency for the temporal lobe's High beta wave are helpful when evaluating stressful circumstances.



**Figure [2]:** A 40 years old female. EEG of normal healthy case with comment waves alpha&beta. The relative Alpha wave's wave frequency was shown to range around 2 and 4 minutes. The parietal lobe had the highest values, although there was a clear distinction between C3 and C4 in the frontal lobe. Prefrontal lobes [Fp1, Fp2] in C3 did not exhibit any discernible differences. Therefore, it is assumed that the variations in the frequency of waves for the frontal lobe's relative Alpha wave are helpful when evaluating a non-stressful situation.



Figure [3]: Frequency of alpha and beta waves among vitiligo patients



Figure [4]: Frequency of alpha and beta waves among normal group

### **DISCUSSION**

Stressful life circumstances cause vitiligo <sup>[13]</sup>. In the present investigation, the frequency of EEG waves in the sick group was substantially higher compared to the control group. The patient's group had considerably greater levels of alpha, C3, C4, alpha, C3, 20.0–34.0 Hz, beta 2, C3, 20.0–34.0 Hz, and beta 2 waves, C4.20.0–34.0 Hz.

Vitiligo participants in this study experience stress at a slightly higher rate than the control group <sup>[14]</sup>. These results support those of **Lai** *et al.* <sup>[15]</sup> who discovered that people with vitiligo are marginally more likely to develop an affective disorder or develop indicators of stress than people without vitiligo. Additionally, Öztekin and Öztekin <sup>[16]</sup> discovered that vitiligo patients had considerably lower sleep quality and an increased prevalence of depression than the control group. Additionally, **Hamidizadeh** *et al.* <sup>[17]</sup> discovered that vitiligo patients had much higher levels of tension and despondency than healthy subjects. These findings concur with those of **Cupertino** *et al.* <sup>[13]</sup> who claimed that stressful life events cause vitiligo.

The current results agreed with another study done by **Henning** *et al.* <sup>[18]</sup> who used a standardized perceived stress scale [PSS] to quantify this variable in vitiligo patients compared to people without vitiligo in their study to determine the association between stress and vitiligo. They also examined a clinical dataset with the use of the knowledgelinking program ROCKET to determine the prevalence of stress-related illnesses in the vitiligo patient group. A total of 100 individuals with vitiligo and 25 control subjects without vitiligo from a pool of individuals in a current database conducted a web-based questionnaire to measure their levels of felt stress. Subjects also detailed the characteristics of their disease state, such as the body areas that were affected and the severity, duration, and activity of their vitiligo. People with vitiligo reported feeling stress substantially more than people without vitiligo. ROCKET analysis indicated that vitiligo progression was preceded by symptoms of metabolic-related disorders [i.e., "stress"]. There was no association between perceived stress and the severity or stage of the condition, indicating that increased stress may not only be a result of pigment depletion. The information lends more credence to the idea that stress might cause vitiligo to develop [18].

In line with the current study, Manolache and Benea <sup>[19]</sup> investigated the role of stress in the progression of vitiligo and alopecia areata. Stressful situations were rated using Holmes and Rahe's social readjustment assessment index. Stressful incidents were reported by more than 65% of patients [combined alopecia areata and vitiligo], as opposed to 22% of controls. Particularly in women, the average incidence of stressful events changed significantly between patients and controls in the vitiligo group [P =0.02]. In both patient groups, prospective stressful situations happened more frequently. Patients with vitiligo reported personal issues in 47% of instances [one-third of which were connected to exams], and 31% of cases were connected to work or financial issues. Once more, when contrasted with controls, this was statistically significant [P = 0.0002].

Similarly, **Jena**<sup>[20]</sup> conducted an investigation to identify the EEG wave variations brought on by stressful events. For the study, sixty-two medical school graduates were chosen. Every subject had an EEG obtained. The investigation came to the conclusion that patients who are unable to tolerate or adjust to their environment experience EEG alterations under stressful conditions. The extent to which stress affects brain function may be measured by EEG. A recent study agreed with the current findings and approved that alpha waves were visible in the EEG of patients under mild to moderate stress since they were mentally at ease and may have handled the event. However, the alpha wave was supplanted by the beta wave when attentiveness and anxiety levels rose <sup>[21]</sup>.

Another study agreed with the current findings and stated that the EEG of individuals who were under a lot of stress revealed beta waves. Beta waves occurred more frequently than baseline waves did <sup>[22, 23]</sup>. The EEG was a beta wave in people who were under a lot of stress. However, as their stress levels increased, their EEG displayed theta waves, maybe as a result of anger or disappointment <sup>[24]</sup>.

This was the first study to highlight the role of EEG as a successful tool in the diagnosis of stress in vitiligo patients. Other studies have emphasized the application of EEG in the diagnosis of various dermatological disorders. In agreement with the current study, a prior investigation by Liu ChKh et al. [25] used EEG methods to examine 51 psoriasis patients. There were 38 cases when the basic rhythms of the brain's bioelectric activity changed. Individuals with disseminated skin processes and extended disease duration [over 5 years] displayed the most obvious abnormalities [types III and V]. There was shown to be a correlation between the clinical course of the procedure and the EEG characteristics.

In line with the current study, Marshall et al. <sup>[26]</sup> examined whether higher levels of experienced stress are associated with worse performance on two working memory tests. Electroencephalographic recordings and behavioral performance were combined to provide insight into the brain mechanisms that cumulative stress affects. Therefore, cortical oscillatory activity in the theta, alpha, and gamma bandwidths was examined while young and old conducted two separate working memory tasks [a Sternberg and N-back paradigm]. According to behavioral data, older participants performed worse on both tasks due to a greater stress score. Elderly subjects in the highest-stress group showed a decrease in alpha and gamma event-related synchronization, suggesting that greater degrees of experienced stress could affect their capacity to actively maintain a stimulus in working memory and inhibit irrelevant information from interfering with successful maintenance. The results show that chronic stress has a negative impact on cognitive ageing.

Ehrhardt et al. [27] agreed with the current study by adding 38 patients to separate the EEG correlates of stress from early identification of stress-related circumstances and tracking of stress reactions during therapy. When doing the Paced Auditory Serial Addition Test [PASAT; cognitive effort; n = 32] in comparison to resting state [n = 33], they discovered a drop in frontal alpha and an increase in beta power, with linear mixed models adjusting for missing values in some situations. When the PASAT was conducted under time pressure [n = 29] or when a social-evaluative threat [video camera; n = 29] was included, no difference in EEG power was discovered. These results indicate that frontal EEG power can distinguish between stress and resting state but not more subtle variations of the stress responses.

In line with the current study, sixty-two medical students were chosen for the investigation by Jena<sup>[20]</sup> to determine the alterations to EEG waves caused by exam stress using the Medical Students Stressor Questionnaire. Mild, moderate, high, and severe stress levels were assigned to them. All subjects had their EEGs taken in two different situations: baseline, which represented daily life, and exam stress. In order to contrast the alterations in the two scenarios, a paired t-test was used. The baseline EEG was an alpha wave in patients under mild to moderate stress, while the exam stress EEG was a beta wave. The baseline EEG and the stress-related assessment EEG were both beta waves in participants under significant stress. When subjects were under examination stress, the EEG was a theta wave instead of a beta wave at baseline. The alteration is statistically significant for every stress level. Baseline EEG was alpha wave because people with mild and moderate stress levels were able to deal with the circumstance. However, as a result of their heightened stress levels and inability to deal during the assessment, the EEG revealed beta waves. Both the baseline EEG [lower frequency wave] and the EEG during test stress [higher frequency waves] were beta waves in patients who were under a lot of stress. Baseline EEG exhibited a beta wave in participants under severe stress, but examination stress EEG displayed a theta wave due to disappointment and frustration. The results of this research show that exam stress can affect brain function to a certain extent, as measured by EEG.

Alshebly *et al.* <sup>[28]</sup> agreed with the current study and conducted yet another investigation to

recognize stress using EEG data. A certain range of frequencies in the spectrum experience changes in their activity due to stress, and these alterations can be observed and studied. The information demonstrates how stress alters the ratio of beta waves to alpha waves in the brain. The ratio's fluctuations will be able to indicate the level of stress that was experienced.

**Conclusion:** To sum up, this was the first study to emphasize the significance of EEG as a technique for accurately diagnosing stress in people with vitiligo. In accordance with other studies, the results of this study show that people with vitiligo exhibit elevated levels of stress. In the current study, participants with vitiligo displayed substantial EEG results other than those of the normal healthy group. For the two electrodes [C3 and C4], the alpha and beta powers rose. Patients with vitiligo have elevated frequencies of alpha and beta waves, which suggests that they are under stress. Stress from metabolic and psychological factors may affect vitiligo patients' susceptibility to the disease's start and progression. Future research is necessary to look at any connections between the onset and development of vitiligo and any other neurological or psychiatric conditions. Our data as a whole implied that stress may have a role in the development and progression of vitiligo.

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### REFERENCES

- 1. Bergqvist C, Ezzedine K. Vitiligo: A Review. Dermatology. 2020;236[6]:571-592. doi: 10.1159/000506103.
- 2. Picardo M, Taïeb A, editors. *Vitiligo*. Springer, Heidelberg & New York: 2019. doi: 10.1007/978-3-319-62960-5.
- 3. Seneschal J, Boniface K, D'Arino A, Picardo M. An update on Vitiligo pathogenesis. Pigment Cell Melanoma Res. 2021 Mar;34[2]:236-243. doi: 10.1111/pcmr.12949.
- Frisoli ML, Essien K, Harris JE. Vitiligo: Mechanisms of Pathogenesis and Treatment. Annu Rev Immunol. 2020 Apr 26;38:621-648. doi: 10.1146/annurev-immunol-100919-023531.
- 5. Rashighi M, Harris JE. Vitiligo Pathogenesis and Emerging Treatments. Dermatol Clin. 2017;35[2]: 257-265. doi: 10.1016/j.det.2016.11.014.
- 6. Boniface K, Seneschal J, Picardo M, Taïeb A. Vitiligo: Focus on Clinical Aspects,

Immunopathogenesis, and Therapy. Clin Rev Allergy Immunol. 2018 Feb;54[1]:52-67. doi: 10.1007/s12016-017-8622-7.

- Abdel-Malek ZA, Jordan C, Ho T, Upadhyay PR, Fleischer A, Hamzavi I. The enigma and challenges of vitiligo pathophysiology and treatment. Pigment Cell Melanoma Res. 2020 Nov;33[6]:778-787. doi: 10.1111/pcmr.12878.
- Dahir AM, Thomsen SF. Comorbidities in vitiligo: comprehensive review. Int J Dermatol. 2018 Oct;57[10]:1157-1164. doi: 10.1111/ijd.14055.
- Bishnoi A, Parsad D. Clinical and Molecular Aspects of Vitiligo Treatments. Int J Mol Sci. 2018 May 18;19[5]:1509. doi: 10.3390/ ijms19051509.
- 10. Passeron T. Medical and Maintenance Treatments for Vitiligo. Dermatol Clin. 2017 Apr;35[2]:163-170. doi: 10.1016/j.det.2016.11.007.
- 11. Jobert M, Wilson FJ, Roth T, Ruigt GS, Anderer P, Drinkenburg WH, *et al.* Guidelines for the recording and evaluation of pharmaco-sleep studies in man: the International Pharmaco-EEG Society [IPEG]. Neuropsychobiology. 2013;67[3]: 127-67. doi: 10.1159/000343449.
- 12. Malver LP, Brokjaer A, Staahl C, Graversen C, Andresen T, Drewes AM. Electroencephalography and analgesics. Br J Clin Pharmacol. 2014 Jan;77 [1]:72-95. doi: 10.1111/bcp.12137.
- Cupertino F, Niemeyer-Corbellini JP, Ramos-E-Silva M. Psychosomatic aspects of vitiligo. Clin Dermatol. 2017 May-Jun;35[3]:292-297. doi: 10.1016/j.clindermatol.2017.01.001.
- 14. Nasser MA, Raggi El Tahlawi SM, Abdelfatah ZA, Soltan MR. Stress, anxiety, and depression in patients with vitiligo. Middle East Current Psychiatry. 2021 Dec;28:1-10. doi: 10.1186/ s43045-021-00120-w.
- 15. Lai YC, Yew YW, Kennedy C, Schwartz RA. Vitiligo and depression: a systematic review and meta-analysis of observational studies. Br J Dermatol. 2017 Sep;177[3]:708-718. doi: 10.1111/ bjd.15199.
- Öztekin A, Öztekin C. Sleep quality and depression in vitiligo patients. Eurasian J Fam Med. 2020;9[1]:35-41. doi: 10.33880/ejfm. 2020090105
- 17. Hamidizadeh N, Ranjbar S, Ghanizadeh A, Parvizi MM, Jafari P, Handjani F. Evaluating prevalence of depression, anxiety and hopelessness in patients with Vitiligo on an Iranian population.

Health Qual Life Outcomes. 2020 Feb 3;18[1]:20. doi: 10.1186/s12955-020-1278-7.

- Henning SW, Jaishankar D, Barse LW, Dellacecca ER, Lancki N, Webb K, *et al.* The relationship between stress and vitiligo: Evaluating perceived stress and electronic medical record data. PLoS One. 2020;15[1]:e0227909. doi: 10. 1371/journal.pone.0227909.
- 19. Manolache L, Benea V. Stress in patients with alopecia areata and vitiligo. J Eur Acad Dermatol Venereol. 2007 Aug;21[7]:921-8. doi: 10.1111/j.1468-3083.2006.02106.x.
- 20. Jena SK. Examination stress and its effect on EEG. Int J Med Sci Pub Health. 2015;11[4]:1493-7. doi: 10.5455/ijmsph.2015.23042015308.
- 21. Niemic C. Studies of emotion: a theoretical and empirical review of psychophysiological studies of emotion. 2004. doi: hdl.handle.net/1802/3032.
- 22. Lindsley DB. Psychological phenomena and the electroencephalogram. Electroencephalogr Clin Neurophysiol. 1952 Nov;4[4]:443-56. doi: 10. 1016/0013-4694[52]90075-8.
- 23. Papanicolaou AC, Loring DW, Deutsch G, Eisenberg HM. Task-related EEG asymmetries: a comparison of alpha blocking and beta enhancement. Int J Neurosci. 1986 Aug;30[1-2]:81-5. doi: 10.3109/00207458608985658.
- 24. Schacter DL. EEG theta waves and psychological phenomena: a review and analysis. Biol Psychol. 1977 Mar;5[1]:47-82. doi: 10.1016/0301-0511 [77]90028-x.
- 25. Liu ChKh, Tsoĭ ChP, Dé BD, Timoshin GG. Elektroéntsefalograficheskie issledovaniia pri psoriaze [Electroencephalographic studies in psoriasis]. Vestn Dermatol Venerol. 1990;[3]:27-8. Russian. PMID: 2368489.
- 26. Marshall AC, Cooper NR, Segrave R, Geeraert N. The effects of long-term stress exposure on aging cognition: a behavioral and EEG investigation. Neurobiol Aging. 2015 Jun;36[6]:2136-44. doi: 10.1016/j.neurobiolaging.2015.02.026.
- 27. Ehrhardt NM, Fietz J, Kopf-Beck J, Kappelmann N, Brem AK. Separating EEG correlates of stress: Cognitive effort, time pressure, and social-evaluative threat. Eur J Neurosci. 2022 May;55[9-10]:2464-2473. doi: 10.1111/ejn.15211.
- Alshebly YSa, Sidek KA, Johar MGM, editors. Stress recognition using Electroencephalogram [EEG] signal. Journal of Physics: Conference Series; 2020: IOP Publishing. doi: 10.1088/1742-6596/1502/1/012052.



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