

Resting-State QEEG Differences between Borderline and Antisocial Personality Disorders: A Supervised Machine Learning Approach

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Abstract

Introduction:

Borderline personality disorder (BPD) and antisocial personality disorder (ASPD) pose substantial clinical challenges. Due to overlapping features such as impulsivity and emotional dysregulation, differential assessment between these conditions can be difficult. This study compared resting-state quantitative electroencephalography (QEEG) profiles between BPD and ASPD and examined whether supervised machine learning could distinguish the two groups based on neurophysiological features.

Method

Thirty age-, gender-, and education-matched participants (15 BPD, 15 ASPD) underwent eyes-closed resting-state EEG recording. After artifact removal, three minutes of clean data were retained for analysis. Absolute power spectra (1.0–30.0 Hz) across standard frequency bands were extracted from 19 channels. Seven classifiers were evaluated using stratified 10-fold cross-validation, with preprocessing performed within training folds to reduce data leakage.

Results

Univariate analyses revealed distinct regional electrophysiological patterns. Relative to the ASPD group, the BPD group showed higher absolute power across several frontal/frontopolar slow-wave measures, centro-parietal delta/theta measures, and temporal delta/theta measures, with additional between-group differences in selected alpha and beta features. Among the evaluated models, the Multi-Layer Perceptron (MLP) showed the strongest internal cross-validated performance (accuracy = 89%, balanced accuracy = 92%, F1-score = 90%).

Conclusion

BPD and ASPD were associated with distinguishable resting-state QEEG patterns in this sample. These findings suggest that QEEG features, combined with supervised machine learning, may serve as candidate neurophysiological markers and as a potential adjunct to differential clinical assessment, pending replication in larger independent samples.

Introduction

Personality disorders are defined as a group of psychological conditions characterized by stable and maladaptive patterns in thinking, emotions, and behavior (Kaplan & Sadock, 2021). Borderline (BPD) and Antisocial (ASPD) personality disorders, both belonging to Cluster B, represent significant public health challenges due to their high prevalence and disruptive nature. ASPD is primarily characterized by a

persistent disregard for social norms and the rights of others, often manifesting in aggressive and impulsive behaviors (World Health Organization, 2019). In contrast, BPD is marked by severe mood instability, chronic feelings of emptiness, and profound difficulties in emotional regulation, which frequently lead to interpersonal crises and self-destructive acts (Stoffers-Winterling et al., 2022). Collectively, these disorders impose an immense burden on healthcare, judicial, and social systems (Lenzenweger et al., 2018).

Despite their distinct diagnostic criteria, BPD and ASPD frequently overlap in clinical presentations, particularly regarding impulsivity and emotional volatility. This leads to diagnostic ambiguity and frequent misdiagnoses, a process further complicated by gender biases where patients are often mislabeled based on stereotypical presentations (Phillips et al., 2012). The reliance on descriptive criteria (DSM and ICD) rather than objective biological markers often results in treatment protocols that fail to address the specific neurological and cognitive dysfunctions underlying each disorder. Consequently, there is a critical need to transition from purely descriptive psychiatric assessment to an integrated approach that identifies objective, biological signatures for these disorders.

Research suggests that both BPD and ASPD have profound biological roots involving deficits in neural circuitry (Schermer et al., 2020). While previous studies have extensively explored behavioral and environmental determinants, identifying a reliable biological marker remains a cornerstone for differential diagnosis. Electroencephalography (EEG) offers a unique window into these dysfunctions. Since Hans Berger's pioneering work, EEG has evolved into a vital tool for assessing mental states, as rhythmic brain waves directly reflect cognitive and emotional processing.

In BPD, frontal EEG asymmetry has been investigated as a potential pathological marker, reflecting key difficulties in emotional regulation and approach-withdrawal motivation (Beeney et al., 2014). Conversely, individuals with ASPD often demonstrate distinct arousal abnormalities. Historically, elevated delta and theta activity in ASPD populations has been interpreted as a reflection of reduced cortical arousal, which may contribute to the characteristic impulsivity and sensation-seeking behaviors seen in these individuals (Ellingson, 1954; Forssman & Frey, 1953). Despite these historical insights, the field lacks a comparative study that utilizes these EEG markers to precisely delineate the neurophysiological boundaries between BPD and ASPD.

To bridge this gap, the present study utilizes advanced computational processing to analyze EEG patterns. Traditional statistical methods often struggle to decode the complex, non-linear relationships within brain signals that differentiate closely related psychopathologies. Machine learning offers an objective analytical lens capable of identifying subtle, high-dimensional patterns in neurophysiological data that are invisible to conventional inspection (Sajjadian et al., 2021; Köber et al., 2022). Accordingly, this study aimed to compare resting-state QEEG patterns in individuals with BPD and ASPD and to examine whether supervised machine learning models could differentiate the two groups on the basis of neurophysiological features.

Methods

Study Design and Participants

This cross-sectional, comparative study adhered to the ethical principles outlined in the Declaration of Helsinki and was formally approved by the Institutional Review Board and Ethics Committee of the University of Mohaghegh Ardabili (Protocol Code: IR.UMA.REC.1403.030). Written informed consent was obtained from all participants prior to study enrollment and electrophysiological assessment.

Participants were recruited through consecutive sampling from outpatient psychiatric clinics and diagnostic centers in Tehran, Iran, between November 2024 and April 2025. In accordance with STROBE reporting guidelines, an initial screening identified 38 individuals with suspected personality pathology. Of these, 32 met the inclusion criteria on initial psychometric and clinical evaluation. Two eligible candidates declined participation due to personal scheduling conflicts, yielding a final analytical sample of 30 participants distributed equally into two distinct clinical cohorts: individuals diagnosed with Borderline Personality Disorder (BPD; $n = 15$) and individuals diagnosed with Antisocial Personality Disorder (ASPD; $n = 15$).

The two clinical cohorts were matched on primary demographic parameters, including biological sex distribution, chronological age, and formal educational attainment. Baseline equivalence was verified using independent-samples t-tests for continuous variables and Pearson's chi-square (χ^2) test for categorical variables. Detailed demographic and clinical profiles are summarized in Table 1.

To ensure sample homogeneity and isolate pathology-specific neurophysiological profiles, participants were selected using a dual-tier assessment framework. Diagnostic eligibility required a primary DSM-5 diagnosis of either BPD or ASPD established via the Structured Clinical Interview for DSM-5 Personality Disorders (SCID-5-PD), subsequently corroborated by a Base Rate (BR) score exceeding 85 on the corresponding scale of the Millon Clinical Multiaxial Inventory-III (MCMI-III).

Inclusion criteria were: (a) age between 18 and 45 years; (b) absence of concurrent or lifetime DSM-5 major psychiatric disorders (e.g., major depressive disorder, bipolar affective disorders, and active psychotic disorders); (c) no psychiatric inpatient hospitalization within the preceding six months; and (d) absence of lifetime electroconvulsive therapy (ECT), neurological conditions (e.g., epilepsy, cerebrovascular events), traumatic brain injury (TBI) involving loss of consciousness, intracranial neoplasms, or cranial radiation/chemotherapy. Individuals presenting with claustrophobia, active scalp dermatoses, or physical conditions contraindicating prolonged EEG acquisition were excluded.

Rigorous pharmacological and lifestyle washouts were enforced: (a) verified abstinence from alcohol and recreational substances for at least two weeks prior to neurophysiological recording (confirmed via clinical screening); (b) complete withdrawal from psychostimulants and centrally active agents altering spectral EEG power (e.g., benzodiazepines, barbiturates, or anticonvulsants) for a minimum of 72 hours;

and (c) absence of structured psychotherapy exceeding five sessions during the preceding three months, thereby minimizing treatment-induced neuroplasticity confounds.

Sex and Gender Considerations

Biological sex was verified via medical intake documentation and demographic self-report. Both male and female participants were enrolled across the two clinical groups (BPD: 7 males, 8 females; ASPD: 8 males, 7 females). Matching procedures achieved statistical equivalence between cohorts ($\chi^2 = 0.13$, $p = .715$), minimizing sex-mediated biological variance in resting-state quantitative EEG comparisons. In accordance with SAGER guidelines, biological sex was handled as a strictly matched background variable rather than an independent disaggregation factor given the sample size.

Psychological Assessment

Structured Clinical Interview for DSM-5 Personality Disorders (SCID-5-PD). Clinical diagnoses of BPD and ASPD were determined using the validated Persian adaptation of the SCID-5-PD. The SCID-5-PD is a semi-structured diagnostic interview evaluating DSM-5 personality criteria across a standardized metric. The Persian translation exhibits robust clinical reliability and validity within Iranian populations (Gharraee et al., 2022).

Millon Clinical Multiaxial Inventory-III (MCMI-III). Personality pathology was psychometrically corroborated using the MCMI-III, a 175-item true/false self-report instrument assessing 14 personality patterns and 10 clinical syndromes. Raw scores are converted to Base Rate (BR) scores, with $BR \geq 85$ defining clinically significant personality pathology. The Persian version has demonstrated sound psychometric properties, including high internal consistency (Cronbach's $\alpha = .89$) and test-retest reliability ($r = .86$) (Sharifi et al., 2007).

Diagnostic Confirmation. All enrolled participants satisfied full DSM-5 criteria on the SCID-5-PD. Furthermore, psychometric confirmation demonstrated that all individuals in the BPD cohort scored $BR \geq 85$ on the Borderline scale (Scale 8A), and all participants in the ASPD cohort scored $BR \geq 85$ on the Antisocial scale (Scale 6B).

EEG Acquisition and QEEG Processing

Electroencephalographic recordings were conducted at the National Brain Mapping Laboratory (NBML), Tehran, Iran. Data were acquired using a 19-channel g.USBamp amplifier system (g.tec medical engineering GmbH, Austria) positioned according to the international 10–20 system (Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, and O2). The system utilized 24-bit A/D conversion with an input range of $\pm 500 \mu V$. Linked mastoids served as ground and physical reference leads.

Recordings were performed during an eyes-closed resting-state condition in an electromagnetically shielded, sound-attenuated room. Recording duration was approximately 7 minutes and 15 seconds. Data were sampled at 256 Hz with online filtering set from 1.0 to 45.0 Hz. Impedances were continuously kept below 5 k Ω , and a Driven Right Leg (DRL) circuit was utilized to suppress electrical noise.

Quantitative EEG Parameterization

Artifact cleaning and spectral decomposition were executed offline using NeuroGuide (v2.8.5, Applied Neuroscience, Inc.). Ocular, cardiac, and myogenic artifacts were identified via automated rejection protocols and verified through visual inspection. Only continuous artifact-free epochs of at least 2.5 seconds were retained, yielding exactly 180 seconds (3 minutes) of clean resting-state data per subject.

Absolute power values (μV^2) were derived via Fast Fourier Transform (FFT) across all 19 channels. Spectral parameters were extracted for standard bands: delta (1.0–4.0 Hz), theta (4.0–8.0 Hz), alpha (8.0–12.0 Hz; subdivided into alpha1: 8.0–10.0 Hz and alpha2: 10.0–12.0 Hz), beta (12.0–30.0 Hz; subdivided into beta1: 12.0–15.0 Hz, beta2: 15.0–18.0 Hz, and beta3: 18.0–25.0 Hz), and gamma bands (30.0–45.0 Hz; including gamma1 and gamma2 sub-bands).

Statistical and Machine Learning Analysis

Univariate Statistical Analyses. Descriptive statistics (mean $\pm$ standard deviation) were calculated for demographic indices and QEEG absolute power metrics. Differences in demographics were evaluated using independent-samples t-tests and chi-square tests. Regional absolute power differences between BPD and ASPD were examined via two-tailed independent-samples t-tests in SPSS (v26.0) at $\alpha = .05$. These comparisons served descriptive characterization and were strictly segregated from the machine learning feature selection pipeline to avoid double-dipping.

Machine Learning Classification and Validation. To determine whether resting-state QEEG features reliably differentiate BPD from ASPD, seven machine learning algorithms encompassing diverse learning inductive biases were implemented: Multi-Layer Perceptron (MLP), K-Nearest Neighbors (KNN), Linear Discriminant Analysis (LDA), Label Propagation (LP), Support Vector Machine (SVM), Random Forest (RF), and Decision Tree (DT). The input matrix consisted exclusively of neurophysiological absolute power indices; demographic covariates were omitted.

Model performance was validated using a stratified 10-fold cross-validation protocol, preserving class parity ($n = 15$ vs. $n = 15$) across iterations. To strictly prevent data leakage, all preprocessing steps (including min-max feature scaling) were fitted exclusively within the training folds and subsequently applied to the validation folds. Classification performance was evaluated using mean Accuracy, Balanced Accuracy, and F_1 score across the folds in Python (v3.9) utilizing Scikit-learn. Given the sample size ($N = 30$), these metrics are reported as internal cross-validated estimates.

Results

Demographic Characteristics

The demographic and clinical characteristics of the study cohorts are detailed in Table 1. Chi-square analysis indicated no significant disparity in sex distribution between cohorts ($\chi^2 = 0.13$, $p = .715$). Similarly, independent-samples t-tests indicated that the groups did not differ in chronological age ($t(28) = -0.58$, $p = .567$) or formal educational attainment ($t(28) = -0.97$, $p = .341$), confirming baseline demographic equivalence.

Table 1
Demographic Characteristics of the Study Groups

Variable	BPD (n = 15)	ASPD (n = 15)	Test Statistic	p
Sex (Male / Female)	7 / 8	8 / 7	$\chi^2 = 0.13$	.715
Age (years), Mean (SD)	26.48 (3.63)	27.43 (5.22)	$t = -0.58$	.567
Education (years), Mean (SD)	15.22 (7.47)	18.16 (8.99)	$t = -0.97$	.341
Note. BPD = Borderline Personality Disorder; ASPD = Antisocial Personality Disorder; SD = Standard Deviation; χ^2 = Pearson Chi-square test; t = Independent-samples t-test (two-tailed, df = 28).				

Differences in QEEG Absolute Power between BPD and ASPD

Independent-samples t-tests revealed significant electrophysiological differences across multiple cortical regions and spectral bands between BPD and ASPD cohorts (Table 2).

In frontal regions, BPD participants exhibited marked elevations in slow-wave activity compared to ASPD, displaying significantly higher absolute delta power at Fp1 ($t(28) = 14.23$, $p < .001$), F3 ($t(28) = 13.27$, $p < .001$), F4 ($t(28) = 8.79$, $p < .001$), Fz ($t(28) = 20.57$, $p < .001$), F7 ($t(28) = 2.19$, $p = .037$), and F8 ($t(28) = 2.72$, $p = .011$). Frontal theta was likewise significantly greater in BPD at Fp1 ($t(28) = 35.03$, $p < .001$) and F7 ($t(28) = 2.16$, $p = .040$). For fast oscillations, BPD subjects had higher total beta ($t(28) = 8.61$, $p < .001$) and beta2 ($t(28) = 4.40$, $p < .001$) at F7. Conversely, ASPD participants showed significantly higher beta1 power at Fp1 ($t(28) = -4.23$, $p < .001$) and F7 ($t(28) = -2.15$, $p = .041$).

Central leads demonstrated higher delta activity in BPD at C3 ($t(28) = 6.29$, $p < .001$) and Cz ($t(28) = 2.63$, $p = .014$), as well as elevated alpha2 at C4 ($t(28) = 3.40$, $p = .002$). Central gamma parameters did not yield statistically significant differences.

Parietal derivations showed pronounced slow- and fast-wave divergence. BPD subjects displayed significantly elevated delta power at P3 ($t(28) = 3.51$, $p = .001$), P4 ($t(28) = 3.28$, $p = .003$), and Pz ($t(28) = 4.56$, $p < .001$), accompanied by higher theta at P3 ($t(28) = 2.35$, $p = .026$), P4 ($t(28) = 2.72$, $p = .011$), and Pz ($t(28) = 3.52$, $p = .001$). Furthermore, parietal alpha at P3 ($t(28) = 2.14$, $p = .041$) and absolute beta

indices at P3 (total beta: $t(28) = 6.75$, $p < .001$; beta3: $t(28) = 3.81$, $p = .001$) were also significantly elevated in the BPD cohort.

In occipital regions, theta power at O1 was significantly higher in BPD ($t(28) = 3.80$, $p = .001$), while other occipital rhythms showed no significant group differentiation.

Temporal channels exhibited elevated slow waves in BPD, displaying higher delta at T3 ($t(28) = 4.13$, $p < .001$) and T4 ($t(28) = 4.63$, $p < .001$), and higher theta at T4 ($t(28) = 2.68$, $p = .012$). Conversely, alpha power was significantly greater in ASPD at T3 (total alpha: $t(28) = -2.17$, $p = .039$; alpha1: $t(28) = -3.81$, $p = .001$).

Table 2

Group Comparison of Selected Resting-State QEEG Absolute Power Features Between BPD and ASPD

Cortical Site & Spectral Band	BPD (n = 15) Mean (SD)	ASPD (n = 15) Mean (SD)	t (df = 28)	p-value	Sig.
Frontal Regions					
AP-Fp1 Delta	86.24 (5.68)	62.32 (3.18)	14.23	< .001	***
AP-Fp1 Theta	24.58 (1.21)	13.44 (0.23)	35.03	< .001	***
AP-Fp1 Beta1	1.75 (2.13)	5.06 (2.16)	-4.23	< .001	***
AP-Fp2 Beta	8.93 (1.54)	8.11 (0.29)	2.03	.052	ns
AP-F3 Delta	35.62 (3.48)	22.54 (1.57)	13.27	< .001	***
AP-F4 Delta	32.68 (4.69)	21.41 (1.63)	8.79	< .001	***
AP-Fz Delta	36.54 (3.19)	19.22 (0.68)	20.57	< .001	***
AP-F7 Delta	37.83 (1.12)	36.48 (2.11)	2.19	.037	*
AP-F7 Theta	10.03 (0.48)	9.02 (1.75)	2.16	.040	*
AP-F7 Beta	5.11 (0.47)	4.02 (0.14)	8.61	< .001	***
AP-F7 Beta1	1.82 (0.96)	2.38 (0.32)	-2.15	.041	*
AP-F7 Beta2	1.48 (0.23)	1.18 (0.13)	4.40	< .001	***
AP-F7 Beta3	2.63 (0.72)	2.23 (0.89)	1.35	.188	ns
AP-F7 High Beta	1.04 (0.05)	1.12 (0.32)	-0.96	.345	ns
AP-F7 Gamma	0.74 (0.51)	0.64 (0.14)	0.73	.471	ns
AP-F7 Gamma1	0.57 (0.32)	0.42 (0.24)	1.45	.158	ns
AP-F7 Gamma2	0.16 (0.06)	0.14 (0.23)	0.33	.744	ns
AP-F7 High Gamma	0.06 (0.22)	0.05 (0.09)	0.16	.874	ns
AP-F8 Delta	34.96 (2.14)	32.72 (2.36)	2.72	.011	*
Central Regions					
AP-C3 Delta	28.10 (4.69)	19.68 (2.21)	6.29	< .001	***
AP-C3 High Beta	1.16 (0.79)	1.13 (0.58)	0.12	.905	ns
<i>Note.</i> Absolute power (AP) is expressed in μV^2 as Mean (SD). df = 28. ns = non-significant ($p \geq .05$), *$p < .05$, **$p < .01$, ***$p < .001$. Negative t-values indicate higher mean power in the ASPD cohort relative to BPD. Values represent primary regional leads and candidate spectral parameters evaluated across cohorts; given the exploratory nature of these univariate comparisons, uncorrected p-values are reported.					

Cortical Site & Spectral Band	BPD (n = 15) Mean (SD)	ASPD (n = 15) Mean (SD)	t (df = 28)	p-value	Sig.
Frontal Regions					
AP-C3 Gamma	0.68 (0.21)	0.52 (0.49)	1.16	.256	ns
AP-C3 Gamma1	0.53 (0.76)	0.51 (0.09)	0.10	.921	ns
AP-C3 High Gamma	0.06 (0.44)	0.04 (0.23)	0.16	.874	ns
AP-Cz Delta	32.45 (4.72)	28.93 (2.16)	2.63	.014	*
AP-Cz Gamma2	0.14 (0.08)	0.12 (0.35)	0.22	.827	ns
AP-C4 Alpha2	6.23 (1.19)	5.03 (0.67)	3.40	.002	**
AP-C4 Beta3	4.01 (2.06)	4.19 (0.23)	-0.34	.736	ns
AP-C4 High Gamma	1.14 (0.32)	1.09 (0.44)	0.36	.721	ns
Parietal Regions					
AP-P3 Delta	22.63 (2.19)	19.15 (3.16)	3.51	.001	**
AP-P3 Theta	12.43 (2.67)	10.36 (2.13)	2.35	.026	*
AP-P3 Alpha	29.12 (4.90)	26.11 (2.39)	2.14	.041	*
AP-P3 Beta	12.23 (1.79)	8.64 (1.02)	6.75	< .001	***
AP-P3 Beta3	4.48 (0.24)	4.16 (0.22)	3.81	.001	**
AP-P3 Gamma1	0.49 (0.09)	0.36 (0.23)	2.04	.051	ns
AP-P3 Gamma2	0.13 (0.05)	0.12 (0.24)	0.16	.874	ns
AP-P4 Delta	25.93 (3.96)	22.41 (1.26)	3.28	.003	**
AP-P4 Theta	13.24 (3.57)	10.11 (2.66)	2.72	.011	*
AP-Pz Delta	32.31 (2.26)	29.31 (1.21)	4.56	< .001	***
AP-Pz Theta	21.03 (0.53)	19.44 (1.67)	3.52	.001	**
AP-Pz High Beta	1.47 (0.21)	1.39 (0.58)	0.50	.621	ns
Occipital Regions					
AP-O1 Delta	22.47 (3.16)	21.37 (2.78)	1.01	.321	ns
<i>Note.</i> Absolute power (AP) is expressed in μV^2 as Mean (SD). df = 28. ns = non-significant ($p \geq .05$), *$p < .05$, **$p < .01$, ***$p < .001$. Negative t-values indicate higher mean power in the ASPD cohort relative to BPD. Values represent primary regional leads and candidate spectral parameters evaluated across cohorts; given the exploratory nature of these univariate comparisons, uncorrected p-values are reported.					

Cortical Site & Spectral Band	BPD (n = 15) Mean (SD)	ASPD (n = 15) Mean (SD)	t (df = 28)	p-value	Sig.
Frontal Regions					
AP-O1 Theta	11.02 (2.19)	8.31 (1.69)	3.80	.001	**
AP-O1 Beta	11.45 (1.98)	11.18 (1.64)	0.41	.685	ns
AP-O1 Beta1	4.32 (0.26)	3.54 (2.13)	1.41	.170	ns
AP-O1 Beta2	2.06 (1.09)	1.96 (0.62)	0.31	.759	ns
AP-O1 Beta3	4.07 (1.21)	3.24 (1.32)	1.80	.083	ns
AP-O1 High Beta	1.16 (0.21)	1.05 (0.24)	1.34	.191	ns
AP-O2 Delta	28.06 (2.69)	28.32 (2.14)	-0.29	.774	ns
Temporal Regions					
AP-T3 Delta	16.67 (1.58)	13.79 (2.18)	4.13	< .001	***
AP-T3 Theta	4.07 (1.16)	3.64 (0.74)	1.21	.236	ns
AP-T3 Alpha	4.19 (1.08)	5.06 (1.12)	-2.17	.039	*
AP-T3 Alpha1	2.31 (0.26)	2.98 (0.63)	-3.81	.001	**
AP-T4 Delta	19.21 (1.10)	16.02 (2.43)	4.63	< .001	***
AP-T4 Theta	3.96 (2.01)	2.37 (1.12)	2.68	.012	*
Note. Absolute power (AP) is expressed in μV^2 as Mean (SD). df = 28. ns = non-significant ($p \geq .05$), *$p < .05$, **$p < .01$, ***$p < .001$. Negative t-values indicate higher mean power in the ASPD cohort relative to BPD. Values represent primary regional leads and candidate spectral parameters evaluated across cohorts; given the exploratory nature of these univariate comparisons, uncorrected p-values are reported.					

Machine Learning Classification Performance

The classification performance of the evaluated algorithms is presented in Table 3. Across all models, the Multi-Layer Perceptron (MLP) achieved superior diagnostic separation, demonstrating an overall accuracy of 89%, a balanced accuracy of 92%, and an F_1 score of 90% across cross-validation folds.

Strong and comparable performance was observed for K-Nearest Neighbors (KNN; accuracy = 78%, balanced accuracy = 82%, F_1 = 81%), Linear Discriminant Analysis (LDA; accuracy = 76%, balanced accuracy = 83%, F_1 = 80%), and Label Propagation (LP; accuracy = 75%, balanced accuracy = 84%, F_1 = 80%). The Support Vector Machine (SVM) and Random Forest (RF) algorithms exhibited moderate classification utility (accuracies of 68% and 65%, respectively). Conversely, the Decision Tree (DT) classifier yielded the lowest performance (accuracy = 38%, balanced accuracy = 41%, F_1 = 43%).

These internal cross-validation results demonstrate that resting-state spectral parameters contain rich discriminative information capable of differentiating BPD from ASPD, with neural network and distance-based architectures exhibiting the greatest sensitivity.

Table 3
Performance Metrics of Machine Learning Classifiers in Differentiating BPD from ASPD

Classifier	Accuracy (%)	Balanced Accuracy (%)	F ₁ Score (%)
Multi-Layer Perceptron (MLP)	89	92	90
K-Nearest Neighbors (KNN)	78	82	81
Linear Discriminant Analysis (LDA)	76	83	80
Label Propagation (LP)	75	84	80
Support Vector Machine (SVM)	68	79	72
Random Forest (RF)	65	63	60
Decision Tree (DT)	38	41	43
<i>Note.</i> All metrics represent internal cross-validated estimates derived from stratified 10-fold cross-validation based on resting-state QEEG absolute power features. Classifiers are ordered by overall accuracy.			

Discussion

The present study investigated the distinct neurophysiological profiles of individuals diagnosed with Borderline Personality Disorder (BPD) and Antisocial Personality Disorder (ASPD) using a combination of quantitative electroencephalography (QEEG) spectral analysis and supervised machine learning classifiers. Our findings revealed significant regional differences in resting-state absolute power across multiple frequency bands between the two groups. These electrophysiological variations not only highlight distinct pathophysiological mechanisms underlying the clinical manifestations of BPD and ASPD but also demonstrate the utility of machine learning, particularly the Multi-Layer Perceptron (MLP), in successfully discriminating between these closely related Cluster B personality disorders. These outcomes align with and expand upon previous neurophysiological investigations of personality pathology (Al-Juhani et al., 2024; Baumgartl et al., 2020; Frick, Rieg, & Buettner, 2021; Montgomery, 2025; Nazari et al., 2024; Singh et al., 2023).

Electrophysiological Differences and Neurocognitive Implications

In the frontopolar and frontal regions, the BPD group exhibited significantly higher absolute power in both delta and theta bands compared to the ASPD group, particularly at FP1, F3, F4, and Fz. Elevated slow-wave activity (delta and theta) in frontal areas is commonly associated with cortical hypoarousal,

neural immaturity, and deficits in executive control networks (Harmony, 2013). The pronounced frontal slow activity in the BPD group suggests a disruption in prefrontal cortical regulation over subcortical emotional centers, which may manifest clinically as affective instability, cognitive distortion, and impaired emotional processing (Rezaei, Azarmi, & Rohampour, 2024). Conversely, the ASPD group demonstrated relatively lower frontal slow-wave power, potentially reflecting different neurocognitive processing styles or baseline arousal mechanisms characteristic of antisocial traits (Kahn, 2021; Knyazev, 2012).

Furthermore, the BPD group showed higher total beta and beta2 activity at F7, whereas the ASPD group exhibited significantly higher beta1 power at FP1 and F7. Fast-wave beta rhythms are intricately linked to cortical activation, top-down cognitive inhibition, and behavioral regulation (Snyder & Hall, 2006). The localized elevation of beta1 power in ASPD alongside frontotemporal beta variations in BPD may underscore distinct neural mechanisms underlying how each group modulates rapid, impulsive behavioral responses and inhibitory control.

Central and parietal regions also revealed marked electrophysiological divergence. The BPD group exhibited increased delta power at C3, Cz, P3, and P4, alongside increased theta power at P3, P4, and Pz. The parietal lobe is critical for integrating sensory information and maintaining spatial and self-referential awareness. Increased slow-wave activity in these regions points to disruptions in localized cortical networks, which may contribute to the dissociative symptoms, depersonalization, and unstable self-image frequently observed in BPD patients. In contrast, the ASPD group exhibited a relative decrease in parietal slow-wave power, suggesting that parietal integration networks operate differently across these clinical cohorts.

Notably, in the temporal regions, the BPD group showed significantly higher delta activity (at T3 and T4) and theta activity (at T4), whereas the ASPD group exhibited lower slow-wave values. The temporal lobes are highly integrated with the limbic system, particularly the amygdala and hippocampus, which govern emotional memory and emotional valence assignment (Attademo, Bernardini, & Verdolini, 2021). The elevated temporal slow-wave activity in BPD points to a dysfunction in temporal-limbic connectivity, potentially explaining the intense fear of abandonment and chronic feelings of emptiness typical of the disorder.

Additionally, the ASPD group demonstrated significantly higher alpha (total alpha and alpha1) power at the T3 channel and lower alpha2 power at C4. Variations in alpha subbands, particularly in central-temporal configurations, are often linked to differences in sensorimotor processing, internal attention mechanisms, and emotional detachment strategies (Pundyk, 2018). The elevation of temporal alpha power in ASPD might reflect a state of active cognitive avoidance or emotional suppression, which aligns with the callous and unemotional traits defining antisocial behavior.

Machine Learning Classification and Diagnostic Utility

A key contribution of this study is the application of supervised machine learning algorithms to classify personality pathology based purely on neurophysiological features. Among the seven algorithms evaluated, the Multi-Layer Perceptron (MLP) achieved the highest diagnostic performance, yielding an accuracy of 89%, a balanced accuracy of 92%, and an F1-score of 90%. The superior performance of the MLP, followed by K-Nearest Neighbors (KNN; accuracy = 78%) and Linear Discriminant Analysis (LDA; accuracy = 76%), underscores the presence of complex, non-linear patterns within resting-state QEEG data that differentiate BPD from ASPD.

While simpler linear models like SVM (accuracy = 68%) and tree-based methods like Random Forest (accuracy = 65%) and Decision Trees (accuracy = 38%) showed moderate to low performance, the deep structure of the MLP was able to capture high-dimensional interactions between spatial-spectral QEEG features. This demonstrates that neuroelectrophysiological biomarkers, when processed through optimized machine learning pipelines, hold substantial promise as objective clinical decision-support tools, aiding in the differential diagnosis of complex psychiatric conditions.

Limitations and Future Directions

Despite the promising classification accuracy, several limitations must be acknowledged. First, the sample size of this study (N = 30) is relatively small, which is a common constraint in clinical QEEG studies involving hard-to-reach psychiatric populations. To mitigate the risk of overfitting and to address the potential bias associated with a small dataset, we implemented a rigorous stratified 10-fold cross-validation protocol, executing all feature scaling and normalization steps strictly within each training fold to prevent data leakage. While this cross-validation framework ensures the internal validity and reliability of the reported performance metrics, the relatively small sample size may still limit the direct generalizability of the classification models to broader clinical populations.

Second, the cross-sectional design of the study prevents the establishment of causal relationships between the observed EEG power abnormalities and the development of BPD or ASPD. Third, although medication washout periods were enforced, long-term neuroplastic modifications induced by past pharmacological interventions cannot be entirely ruled out.

Future investigations should employ longitudinal designs with larger, multi-center clinical cohorts to validate these machine learning classifiers. Integrating QEEG features with other modalities, such as genetic markers, structural neuroimaging (fMRI), and behavioral metrics, could yield a more comprehensive multi-modal diagnostic framework for personality disorders, ultimately facilitating personalized therapeutic interventions.

Conclusion

In conclusion, this study demonstrates that individuals with BPD and ASPD exhibit distinct resting-state electrophysiological signatures. The BPD group is characterized by widespread elevations in slow-wave activity (delta and theta) across frontal, central, parietal, and temporal regions, alongside localized beta

alterations, suggesting prefrontal-limbic dysregulation. Conversely, the ASPD group is characterized by relatively lower slow-wave activity and specific temporal-central alpha alterations. Furthermore, we showed that these neurophysiological differences can be leveraged by machine learning classifiers—specifically the Multi-Layer Perceptron—to accurately differentiate between the two disorders. These findings support the viability of QEEG-based biomarkers as objective adjuncts to traditional clinical assessments, potentially improving diagnostic accuracy and treatment planning for personality pathology.

Declarations

Competing Interests:

The authors declare that they have no financial, personal, or professional competing interests that could inappropriately influence or bias the content of this manuscript.

Ethical Approval:

All procedures performed in studies involving human participants were conducted in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Ethical approval was formally obtained from the relevant Institutional Ethics Committee.

Informed Consent:

Written informed consent was obtained from all individual participants prior to their inclusion in the study.

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Data Availability:

The raw neurophysiological and psychometric datasets generated during this study involve sensitive clinical and personal information and are not publicly available to ensure participant privacy and confidentiality. Anonymized data supporting the findings of this study are available from the corresponding author upon reasonable request.

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