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Neurofeedback for Alcohol Use Disorder: Implications for Single-Case Research Design and Examining Craving Desire

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Abstract

Substance use disorder (SUD) and alcohol use disorder (AUD) persist as a significant concern in the United States despite increasing treatment options. Effective interventions to reduce cravings and prevent relapse are still sought after. During the pandemic, drinking behaviors and cravings exacerbated among individuals with AUD. Neurofeedback shows documented promise in addressing AUD, yet studies often lack comprehensive data on craving. In this quantitative study, participants with AUD received 12 neurofeedback sessions using the Peniston protocol as inspiration for session designs. Four research questions guided the study, examining pre–post qEEGs; pre, post, and follow-up AUDIT scores; and neurofeedback sessions data. The study also tracked changes in self-reported craving levels over time. Hypotheses predict improvement in post-qEEGs, posttreatment craving scores, and neurofeedback session averages following each neurofeedback session. The discussion will focus on the implications for neurofeedback for AUD, cravings, and single-case research designs.

Keywords: neurofeedback; single-case research design; alcohol use disorder

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Alcohol use disorder (AUD) presents a significant and pervasive challenge in the United States (Edwards et al., 2015). Defined in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*) as "a problematic pattern of alcohol use leading to clinically significant impairment or distress" (American Psychiatric Association [APA], 2013, p. 490), it stands as a valid target for intervention (Dehghani-Arani et al., 2013). Recognizing the importance of addressing ambivalence towards sobriety, the added criteria of "craving" in the *DSM-5* underscores its significance. Additionally, Schlauch et al. (2019) strongly encourages researchers to measure craving over time versus pre–post measurements.

Many treatment options exist for those with AUD; however, an alternative modality is the brain-based intervention known as neurofeedback or EEG-biofeedback (Demos, 2019). Neurofeedback has emerged as a promising approach in addressing addiction symptoms (Dehghani-Arani et al., 2013; Dousset et al., 2020; Shepard, 2015; Sokhadze et al., 2008) with recent calls for more robust studies that may include refined or innovative methodologies to further understand its efficacy (Omejc et al., 2019). Thus, this paper entails quantitative electroencephalogram (qEEG) data while also demonstrating innovative methodologies and analyses of neurofeedback session-to-session data and craving data, which will ideally inform clinicians with valuable insights and present future research options. Further, the methodical approach of single-case research designs (SCRDs) using neurofeedback data for SUD/AUD may offer insights into session-to-session brain wave patterns over

time, along with measured self-report craving desires.

Methods

La Vaque et al. (2002) acknowledge the importance of adhering to best practices in neurofeedback methodologies and studies. For this study, the recommendation of interest is encouraging researchers to incorporate multiple observations (La Vaque et al., 2002). Integrating multiple observations into research studies encompasses various methodologies, including SCRDs, which are also referred to as time series designs and allow participants to serve as their baseline (Kazdin, 2021). Key characteristics of SCRDs include (a) repeated dependent variable measures; (b) measurement across time; and (c) designation of the "case" as an individual, organization, or other type of group (Kazdin, 2021; Lobo et al., 2017). Researchers employing SCRD can also use multiple baselines (where participants start the intervention at different times), reversal designs, and multiple treatment designs based on their desired data results and research objectives. For instance, the A phase serves as the baseline with repeated measures but no intervention, while the B phase incorporates the intervention with the same repeated measurements as the A phase. The fundamental aim is to evaluate whether an intervention has any effect on the independent variable.

Given that variations of SCRDs offer diverse strengths for assessing intervention effects, the literature underscores the importance of researchers exercising caution when analyzing their data. A similar mindset may also prove beneficial for neurofeedback researchers and clinicians, given the significant disparities and complexities in subjects' individual life experiences, physiological development, and underlying brain patterns. Hence, the present study's research questions include the SCRD-based questions and additional questions comparing participants' pre- and post-qEEG data, and their pre–post and follow-up data using the Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993). The research questions guiding this study were as follows:

1. Is there a change in a participant's *z*-scores from pre- to postneurofeedback intervention of normative database comparison qEEG data?
2. Is there a change over time during the neurofeedback treatment sessions in participants' alcohol craving thoughts as measured by the Craving Desire scale (CDS; Ciraulo et al., 2013)?
3. Is there a change over time in neurofeedback session-to-session data for participants' mean magnitude of their respective brain wave frequencies in band 1, band 2, and band 3?
4. Is there a change over time in participants' alcohol use according to the pre, post, and follow-up scores of the AUDIT (Saunders et al., 1993)?

Clinicians

The present study engaged student clinicians, comprising master's level students in clinical mental health from a nationally accredited program approved by the Council for Accreditation of Counseling and Related Education Programs (CACREP). These students had previously fulfilled the didactic coursework requirements for neurofeedback set by the Biofeedback Certification International Alliance (BCIA) and were supervised by a certified and licensed supervisor during data collection and the administration of neurofeedback sessions. Furthermore, volunteer clinicians with neurofeedback training, such as faculty or alumni, were also involved in the study.

Measures

Demographic Questionnaire. The demographic questionnaire included gender, age, ethnicity, family alcohol use, family drug alcohol use, and a current list of medications. Additionally, the form contained questions about the participant's age of first alcohol use, any diagnosis of a mental health disorder, the state of their liver, and if they felt motivated for neurofeedback treatment.

CDS. Researchers Kavanagh et al. (2013) suggest that although a researcher may ask a single question of "Are you craving right now?" for the repeated dependent variable, that internal consistency may improve with an assessment that includes more than a single question. Hence, the postneurofeedback session, self-report measurement for craving in this study was the CDS (Ciraulo et al., 2013). The CDS, developed by Ciraulo et al. (2013), consists of three items assessing the current desire for alcohol. These items are "I do want a drink right now," "I crave a drink right now," and "I have a desire for a drink right now." Responses are rated on a 7-point Likert scale ranging from "very strongly agree" to "very strongly disagree." Ciraulo et al. (2013) specifically designed the CDS for use in AUD studies and for repeated postintervention measurements. The CDS minimum

score is a 3 with the maximum being a 21. Participants were asked after every session to self-report their craving level. All participants reported their CDS scores at 16 time points. Two were completed prior to neurofeedback treatment, 12 were completed after every neurofeedback session, and the last two were collected around 1–3 weeks poststudy. For the purpose of the study and exploring craving change, we computed the CDS scores into Phase A and Phase B.

AUDIT: Self-Report Version. AUDIT (Saunders et al., 1993) serves as an assessment tool to gauge whether an individual's alcohol consumption poses harm. Developed by the World Health Organization through collaboration among six countries, the AUDIT aims to screen drinking behavior and related issues (Saunders et al., 1993). Comprising 10 items, the questionnaire utilizes a range of responses for items 1–8, spanning from 0 to 4 to indicate the frequency of alcohol consumption (0 = *Never*, 1 = *Monthly or less*, 2 = *Two to four times a month*, 3 = *Two to three times a week*, 4 = *Four or more times a week*). A sample question is "How often do you have a drink containing alcohol?" Questions 9 and 10 employ a 3-point Likert scale (1 = *No*, 2 = *Yes*, *but not in the last year*, 3 = *Yes, during the last year*), with an example item being “Have you or someone else been injured as a result of your drinking?” The questionnaire’s structure allocates items 1–3 for assessing alcohol consumption, items 4–6 for alcohol dependence, and items 7–10 for alcohol-related issues. A score of 8 or more for males (7 or more for females) indicates harmful alcohol use (Saunders et al., 1993), while a score of 20 or more suggests alcohol dependence. The maximum score achievable on the questionnaire is 40 (Saunders et al., 1993). Internal consistency of the AUDIT, as demonstrated among 1,888 individuals, yielded mean values of 0.93 for drinking behavior and 0.81 for adverse psychological reactions (Saunders et al., 1993). Validity was assessed through comparison with known alcohol users and nondrinkers. Among alcohol users, 99% scored 8 or higher, with 98% scoring 10 or more. Conversely, only three nondrinkers (0.5%) scored 8 or more.

Instrumentation

Quantitative Electroencephalography. Before commencing neurofeedback treatment, a qEEG was conducted to analyze an individual’s baseline brainwave patterns and pinpoint areas for potential improvement through conditioning. It was recommended that clients refrain from consuming nonessential substances for at least 24 hr prior to the qEEG recording, unless instructed otherwise by a medical professional. Any medically prescribed substances were taken into consideration during the interpretation of the qEEG results. Medications were also considered for the development of treatment protocols as well as the Peniston protocol and the Scott-Kaiser modification (Dousset et al., 2020; Peniston & Kulkosky, 1989, 1990; Scott & Kaiser, 1998).

The 19-channel qEEG recordings were obtained using one of two systems: (a) the BrainMaster Discovery 24 high-impedance amplifier with NeuroGuide software (BrainMaster Technologies, Inc., Bedford, OH) or (b) the Mitsar BT 201 high-impedance amplifier with WinEEG software (Mitsar Co. Ltd., St. Petersburg, Russia). Recordings were conducted in both eyes-closed and eyes-open conditions, utilizing appropriately sized Electro-Caps (Electro-Cap International, Inc., Eaton, OH) fitted according to manufacturer guidelines, along with ear-clip leads. Electrode preparation procedures were carried out to ensure impedance levels remained at or below 5K ohms (Jones, 2015).

Neurofeedback. During the neurofeedback sessions, clinicians employed the BrainMaster Atlantis two-channel amplifiers (BrainMaster Technologies, Inc., Bedford, OH) along with BioExplorer software (Cyberevolution, Inc., Seattle, WA). Electrode site preparation involved cleaning the site, ground, and reference locations with rubbing alcohol and gently abrading them using PDI sterile alcohol prep pads and Nuprep skin prep gel. Gold-plated electrodes were then affixed to the clients using 10-20 conductive paste. Impedance measurements were carefully taken to ensure that interelectrode impedance remained below 5K ohms (Jones, 2015).

Participants

The specific characteristics and inclusion criteria encompassed individuals diagnosed with AUD who were aged 18 years or older. Exclusion criteria comprised active psychosis, current intoxication, advanced liver cirrhosis, and failure to meet the inclusion criteria. Participants were not restricted based on race, gender, ethnicity, or any other demographic variable. Prior to participant recruitment, the study obtained approval from the Institutional Review Board. Recruitment of participants involved reaching out to local counselors working with AUD clients, as well as outpatient facilities, through the distribution of flyers and emails. Additionally, social media platforms were utilized for recruitment purposes. Upon

expressing interest and contacting the Principal Investigator, potential participants received an email containing detailed study information and the Informed Consent document. All neurofeedback services were provided to participants free of charge, and they also received a nominal payment for their participation.

Data Analysis

For the pre–post qEEG data, we first de-identified participant data. Utilizing WinEEG, initial qEEG data underwent frequency domain analysis utilizing the fast fourier transform (FFT) technique as per Beauchamp (1973) and Congedo and Lubar (2003). WinEEG software facilitated this analysis by computing FFT and subsequently determining absolute power, relative power, and mean frequency for each electrode placement on the scalp (Congedo & Lubar, 2003). Next, using NeuroGuide software, participant data is compared with that of healthy individuals from the Lifespan Normative database, enabling clinicians to identify deviations from the norm which are typically expressed in *z*-scores. We also used NeuroGuide for artifacting all participants' qEEG data for EC and EO conditions. The common qEEG montage of LE = linked ears and AVE = average reference was applied. Data reports consist of AVE absolute power *z*-scores.

AUDIT scores consisted of collecting pre (around the initial qEEG), post (during the post qEEG), and follow-up (Qualtrics) measurements for each participant. Simple change score computations were calculated using *Statistical Package for the Social Sciences (SPSS) software version 26* (SPSS, 2019). The AUDIT scores function as the participants' self-report data. Self-report data is highly suggested by Wigton and Krigbaum (2015) to collect and compare with physiological data. The AUDIT pre, post, and follow-up data for all participants is reported in a single chart.

For the SCRD analyses, we initially inputted data into Excel to generate graphical representations depicting the participants' data alongside resulting trend lines. Subsequently, our analysis utilized the nonoverlap of all pairs (NAP) method pioneered by Parker and Vannest (2009). Unlike methods reliant on trend lines or averages, NAP is commonly employed in SCRD and favored in AB Phase designs. While some researchers have criticized NAP analysis for its perceived inability to distinguish between phases (Manolov & Solanas, 2018), it is pertinent to note that in neurofeedback sessions, participants continuously receive the intervention rather than distinct treatment and no-treatment phases. NAP scores are derived by comparing all data points across the two phases (Fielenbach et al., 2019). In our study, Phase A encompasses the initial defined group of neurofeedback sessions, while Phase B comprises the final or successive defined group of sessions. The resulting NAP scores yield effect sizes categorized as follows: 0.00–0.65 indicating a weak effect, 0.66–0.92 suggesting a medium effect, and 0.93–1.0 denoting a large effect (Parker & Vannest, 2009).

To enhance the robustness of the NAP findings, we employed simulation modeling analysis (SMA), a software program provided by Clinical Research Solutions (2021), which is freely downloadable and designed for SCRD involving fewer than 30 time points (Borckardt, 2006). This software enables the control of autocorrelation, assessment of session data slopes and trend lines, and conducts a 5,000-simulation test to identify the most fitting trend line or the most correlated model. The analysis offers five distinct models: (a) Model 1 proposes an increase in outcome measure during Phase A followed by a decrease in Phase B; (b) Model 2 suggests a stable Phase A followed by an increase in Phase B; (c) Model 3 indicates an increase in Phase A followed by stabilization during Phase B; (d) Model 4 proposes a continuous increase from Phase A into Phase B; and (e) Model 5 reveals an increase in Phase A, an immediate decrease, and a subsequent increase in Phase B.

SMA provided valuable insights into participants' neurofeedback session data, allowing for the prediction of subtle changes within the data and offering potential trajectories of participant response had the intervention been continued by clinicians.

Participant 1

Participant 1 (P1) reported having family members with drug and alcohol issues. P1 also stated he began drinking at age 15, identified as a Caucasian male, and when he began the study was 55 years old. P1 was taking doctor-prescribed medication for blood pressure, and an anti-depressant, an Antabuse (i.e., a medication that causes adverse effects with alcohol consumption). P1 reported he was motivated for neurofeedback treatment. Clinicians conducting neurofeedback sessions informed us of P1's elevated anxiety states during his first few sessions.

QEEG Findings. Analyzing P1's initial and final scores (Table 1), it is evident that there was a decrease in theta activity (4–8 Hz) across both EC

and EO conditions. Moreover, there was a significant reduction in higher beta activity, particularly notable in the EC condition. Additionally, in other channels observing EC beta activity (such as Fz, Cz, F3, and P3), initial pre *z*-scores mostly ranged from $z \geq 2.00$. Following the intervention, post scores for these channels exhibited a consistent trend toward the mean with $z \geq 1.00$. P1's individual protocol included downtraining 4–8 Hz, increasing 8–10 Hz, and downtraining 20–25 Hz at Pz with EC.

CDS. P1's mean values across different phases were as follows: Phase A (M = 10.6), Phase B (M = 7.6), and overall (M = 9.2). P1's test for level change yielded $R = -0.43$, $p = .20$; while the test for slope change showed $R = -0.27$, $p = .44$, indicating a decreasing slope vector during both Phase A and Phase B (Figure 1). To further examine the change in trend, we utilized the simple moving average (SMA) descriptive output for ordinary least squares (OLS), revealing an OLS Slope of $m = -0.45$, b = 13.03, 95% CI [7.88, 11.13]. Subsequently, we employed the SMA function of bootstrapped autocorrelation for OLS using the residuals of the fitted model, resulting in N = 16, lag-1 = −0.12, $p = .42$. Additionally, for Phase A, the values were n = 8, lag-1 = −0.48, $p = .09$; and for Phase B, n = 8, lag-1 = 0.003, $p = .30$. Results displayed in Figure 1.

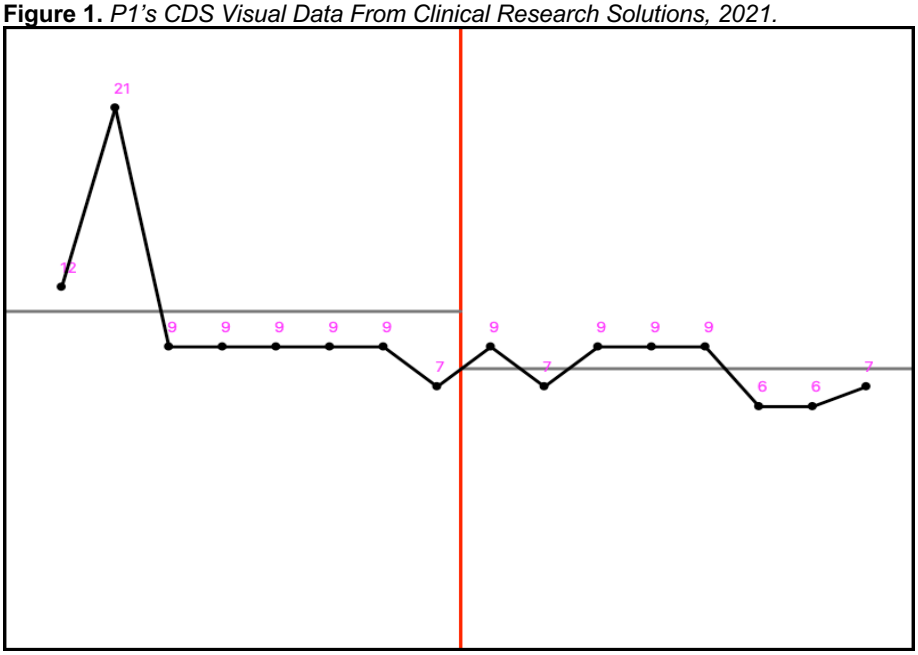
Table 1

Pre/Post qEEG Z-Score Data for P1

	EC Pre	EC Post	EO Pre	EO Post
4–8 Hz	0.89	0.09	0.75	0.17
8–10 Hz	0.57	−0.45	0.15	−0.31
20–25 Hz	4.41	1.54	4.81	2.06

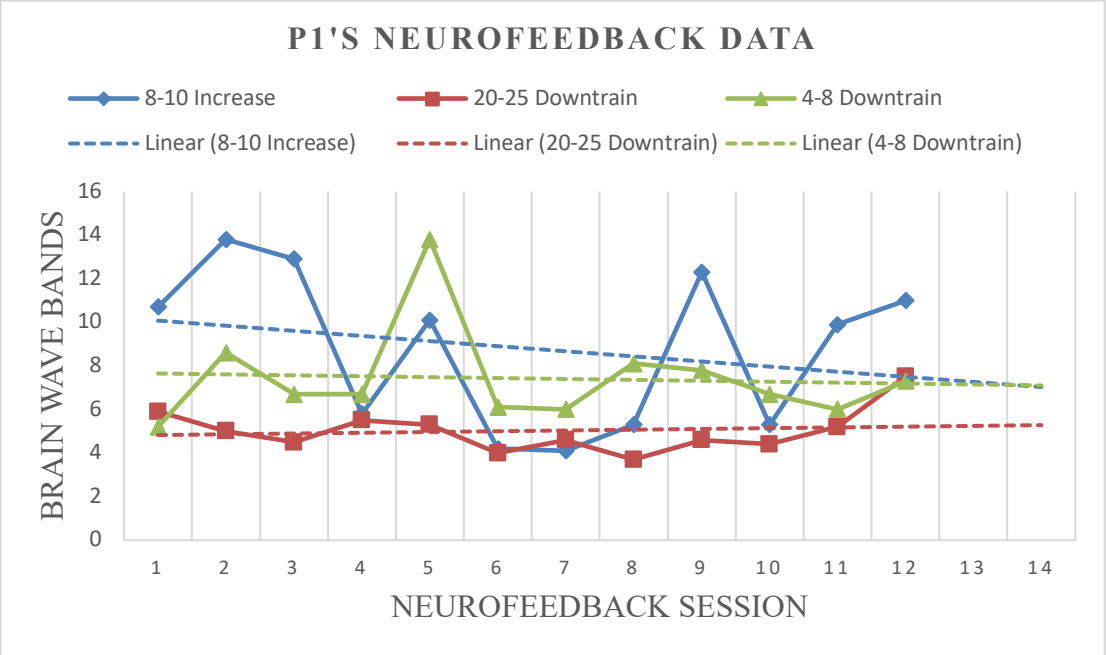
Note. EC = eyes closed; EO = eyes open.

Figure 1. *P1's CDS Visual Data From Clinical Research Solutions, 2021.*



Neurofeedback Session Data. Upon reviewing the visual representation of P1's data (Figure 2), it becomes apparent that the trend lines for both the 8–10 Hz and 20–25 Hz bands are moving in the opposite direction to the desired outcome. However, there is a slight decrease observed in the 4–8 Hz band, suggesting a potential trend toward achieving the protocol goal. This graphical representation serves as the SCRD visual analysis. 12 neurofeedback sessions were categorized into Phase A (n = 6) and Phase B (n = 6) for the analysis of NAP scores. These scores are instrumental in determining the effect size. P1's visual for neurofeedback data are in Figure 2 and the NAP results are detailed in Table 2.

Figure 2. *P1's Visual of Neurofeedback Data.*



According to his NAP scores, P1's data did not reveal any medium or large effects. Additionally, we used the SMA to further examine any unseen or minute changes. In P1's 4–8 Hz band, the SMA models indicated no significant change, with all partial correlations falling within the weak range (i.e., 0.1 to 0.3). Conversely, the 8–10 Hz band exhibited the most favorable fit with Model 1 (R = −0.65, p = .04), signifying a decrease in the outcome measure during Phase A followed by an increase in Phase B, aligning well with the established protocol. Similarly, the change effects observed in P1's 20–25 Hz band were best represented by Model 1 (R = −0.6, p = .03).

Table 2

Nonoverlap of All Pairs Statistical Outcomes for P1

	S	Pairs	NAP	VARs	*z*	*p*	90% CI
4–8 Hz	−2	36	0.472	156	−0.16	.873	[−0.626, 0.515]
8–10 Hz	−12	36	0.333	156	−0.96	.337	[−0.904, 0.237]
20–25 Hz	−8	36	0.389	156	−0.64	.522	[−0.793, 0.349]

Note. S = distribution; Pairs = total pairs comparisons; NAP = nonoverlap of all pairs effect sizes; VARs = variance; *z* = *z*-score; *p* = *p*-value (p = .05); CI = confidence interval.

Participant 2

Participant 2 (P2) self-identified as a 28-year-old Latino male in his Qualtrics demographic form. He indicated that someone in his family struggled with alcohol and drug abuse. P2 disclosed that his own struggle with alcohol began in 2014 at the age of 21. He reported not taking any medications and denied being diagnosed with a mental health disorder. His highest level of education was a college degree, and he expressed satisfaction with his level of social support. P2 expressed motivation for AUD treatment. Clinicians noted his exceptional commitment to neurofeedback sessions and punctuality in keeping his appointments. Throughout the neurofeedback interventions, P2 appeared externally content. Additionally, he was concurrently attending outpatient treatment, which ceased around his ninth neurofeedback session.

QEEG Findings. We artifacted data for both EC and EO conditions. Based on P2's pre- and postscores (see Table 3), there was an increase in the 4–8 Hz band and the 18–25 Hz band, contrary to the intended inhibition and decrease settings for his protocol. However, there was an increase in the 12–15 Hz band from pre to post in both EC and EO conditions. P2's neurofeedback protocol was inhibiting 4–8 Hz, increasing 12–15 Hz, and downtraining 18–25 Hz at Cz with EO.

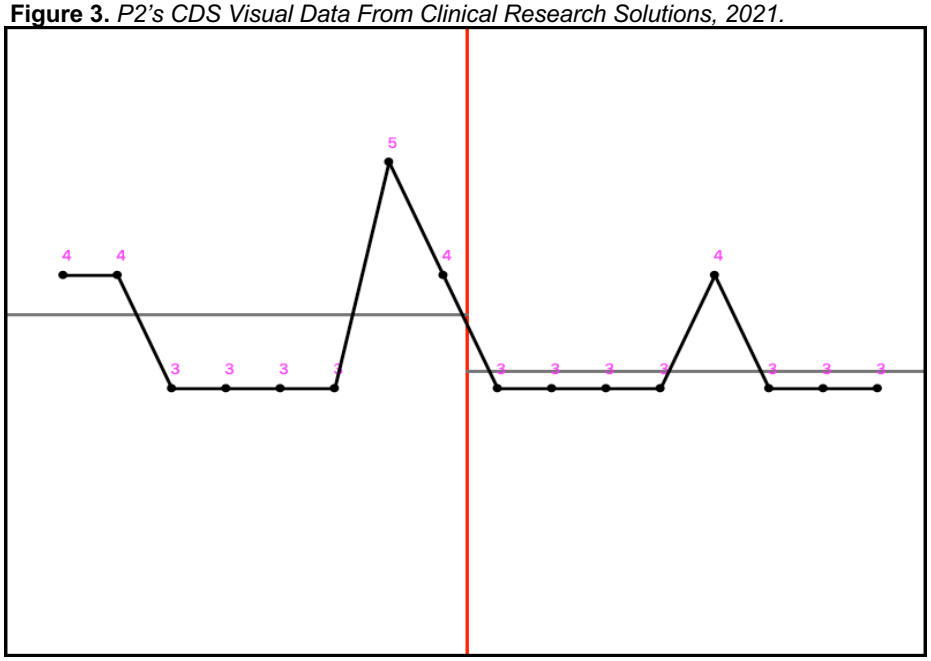
Table 3
Pre/Post qEEG Z-Score Data for P2

	EC Pre	EC Post	EO Pre	EO Post
4–8 Hz	0.66	2.07	0.03	1.01
12–15 Hz	0.96	1.64	0.57	1.20
18–25 Hz	1.98	2.45	1.71	2.89

Note. EC = eyes closed; EO = eyes open.

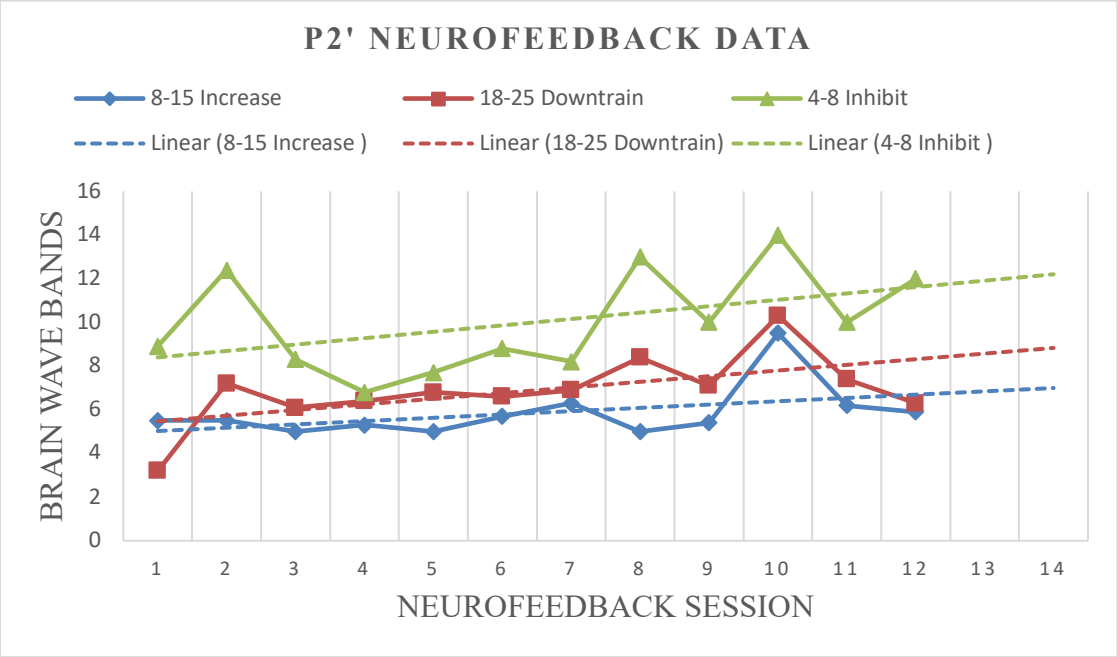
CDS. The mean scores for P2's phases (see Figure 3) were as follows: Phase A (M = 3.6) and Phase B (M = 3.1). The combined mean for both phases was (M = 3.4), which represents the equivalent of level change. Autocorrelation was programmed into all data points for both phases at .183 for lag-1. P2's test for level change yielded R = −0.42, p = .17. The test for slope change resulted in (R = .09, p = .77). For the OLS analysis, the OLS Slope resulted in m = −0.04, b = 3.7, 95% CI [3.13, 3.69]. Additionally, the bootstrapped autocorrelation was utilized for OLS with the residuals of the fitted OLS model, yielding results of N = 16, lag-1 = .15, p = .19. Phase-specific results indicated autocorrelation for Phase A (n = 8, lag-1 = .16, p = .17) and Phase B (n = 8, lag-1 = −.19, p = .35). Running the raw data and removing phase effects for the bootstrapped autocorrelation models revealed no significant effects.

Figure 3. *P2's CDS Visual Data From Clinical Research Solutions, 2021.*



Neurofeedback Session Data. While not the primary focus of his protocol, P2's 4–8 Hz band exhibited an increase rather than the desired inhibition of the wave. However, the 8–15 Hz band showed an increase according to the visual trend line. Given the proximity of these bands and their shared use of the 8 Hz data, some of the observed increase in the 8–15 Hz band may be influencing the 4–8 Hz data. This could potentially account for part of the increase in the 4–8 Hz band. Despite clinicians' emphasis on reducing the 18–25 Hz band, the trend line indicates the opposite effect. Figure 4 provides a visual representation of this analysis.

Figure 4. *P2's Visual of Neurofeedback Data.*



To reiterate, NAP scores ranging from .00 to .65 indicate a weak effect, .66 to .92 suggest a medium effect, and .93 to 1.0 signify a large effect. P2's data exhibited medium NAP score effects across all three brain wave bands (see Table 4). However, none of the *p* scores were significant, although the 18–25 Hz band approached significance, albeit in the opposite trend desired (i.e., increasing instead of decreasing). Furthermore, we analyzed the data using SMA and assessed the fit of five models. P2's 4–8 Hz band trend was best represented by Model 2, indicating a stable Phase A and an increase during Phase B (R = 0.56, p = .07). While P2's 8–15 Hz band did not yield significant findings in the SMA models, it also aligned well with Model 2 (R = 0.46, p = .16), partially supporting his desired trend. Conversely, the change effects for P2's 18–25 Hz band were best captured by Model 3 (R = 0.65, p = .03), demonstrating significance. Model 3 suggests an increase during Phase A followed by a stable or leveling-out Phase B, possibly indicating P2's initial achievement of his protocol goal followed by maintenance of that goal.

Table 4
Nonoverlap of All Pairs Statistical Outcomes for P2

	S	Pairs	NAP	VARs	*z*	*p*	90% CI
4–8 Hz	22	36	0.806	156	1.76	.078	[0.040, > 1]
8–15 Hz	20	36	0.778	156	1.60	.109	[−0.015, > 1]
18–25 Hz	24	36	0.833	156	1.92	.055	[0.096, > 1]

Note. S = distribution; Pairs = total pairs comparisons; NAP = nonoverlap of all pairs effect sizes; VARs = variance; *z* = *z*-score; *p* = *p*-value (p = .05); CI = confidence interval.

Participant 3

Participant 3 (P3) completed the demographic form indicating male gender, 57 years of age, and Latino ethnicity. He mentioned no familial history of alcohol or drug addiction. P3 recognized his initial alcohol-related issue at 17 years old. His current medication regimen included naltrexone, Seroquel, a blood pressure medication, and an antidepressant, prescribed for anxiety. His highest level of education is a master's degree. P3 expressed contentment

with his social support network and exhibited readiness for AUD treatment. During interactions, P3 displayed signs of anxiety through fidgeting, sweating, and body tension. Clinicians observed his restlessness during neurofeedback sessions, occasionally accompanied by yawning and drowsiness. Clinicians offered short breaks to this client. P3 took a 1-week hiatus from neurofeedback sessions due to a work-related commitment.

QEEG Findings. For P3, we used manual artifacting for both EC and EO conditions due to participant movement and tension. The individualized neurofeedback protocol for P3 was downtraining 4–10 Hz, increasing 12–15 Hz, and downtraining 25–30 Hz at Fz with EO. P3's outcomes revealed slight alterations in both the 4–10 Hz and 25–30 Hz bands (see Table 5). Notably, the latter exhibited a favorable shift towards the mean. In the 12–15 Hz band, there was a decrease during EC sessions but an increase during EO sessions. Consequently, the increase in the EO 12–15 Hz band was in accordance with the protocol and deemed beneficial.

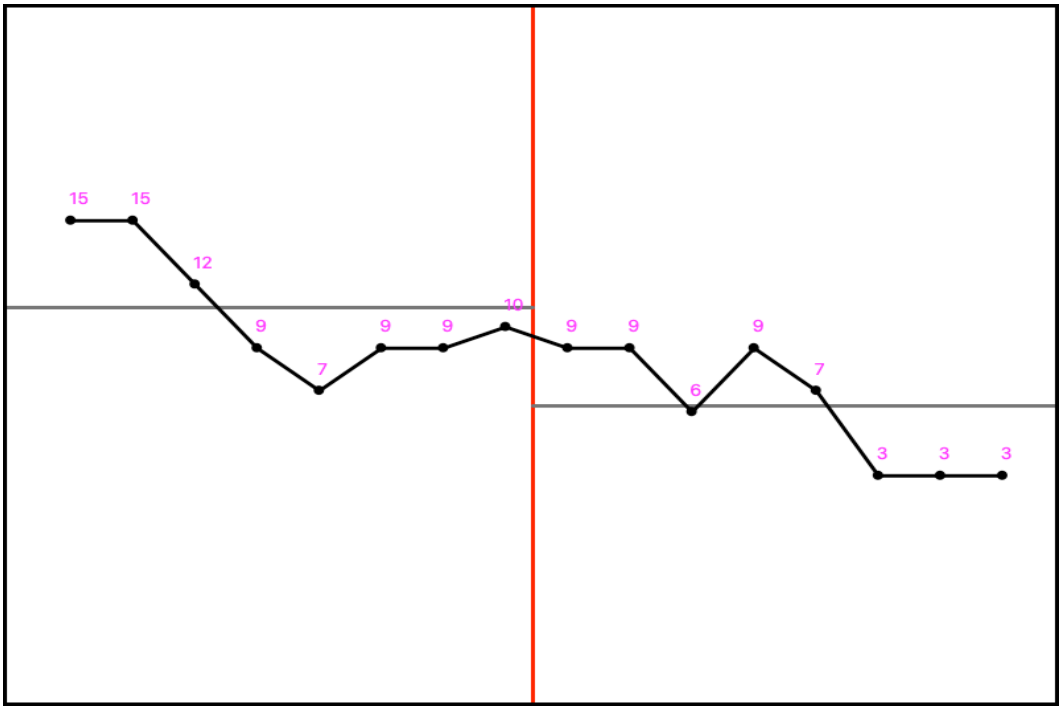
CDS. P3's phase (see Figure 5) means were calculated as follows: Phase A (M = 10.8) and Phase B (M = 6.1). The overall mean across all phases with a sample size of 16 was (M = 8.4). Utilizing the SMA and conducting tests for level change, P3's data yielded R = −0.65, p = .18. Additionally, the test for slope change resulted in R = 0.03, p = .95. In the ordinary least squares (OLS) analysis, the descriptive analysis function was employed to determine the OLS Slope, resulting in m = −0.99, b = 10.57, 95% CI [4.25, 7.86]. The OLS analysis indicated significant results for the entire sample (N = 16, lag-1 = .43, p = .02), as well as for Phase A (n = 8, lag-1 = .47, p = .01), but not for Phase B (n = 8, lag-1 = −.23, p = .38).

Table 5
Pre/Post qEEG Z-Score Data for P3

	EC Pre	EC Post	EO Pre	EO Post
4–10 Hz	0.28	0.79	−0.22	0.61
12–15 Hz	2.00	1.00	0.08	1.38
25–30 Hz	−0.58	−0.29	−0.63	−0.10

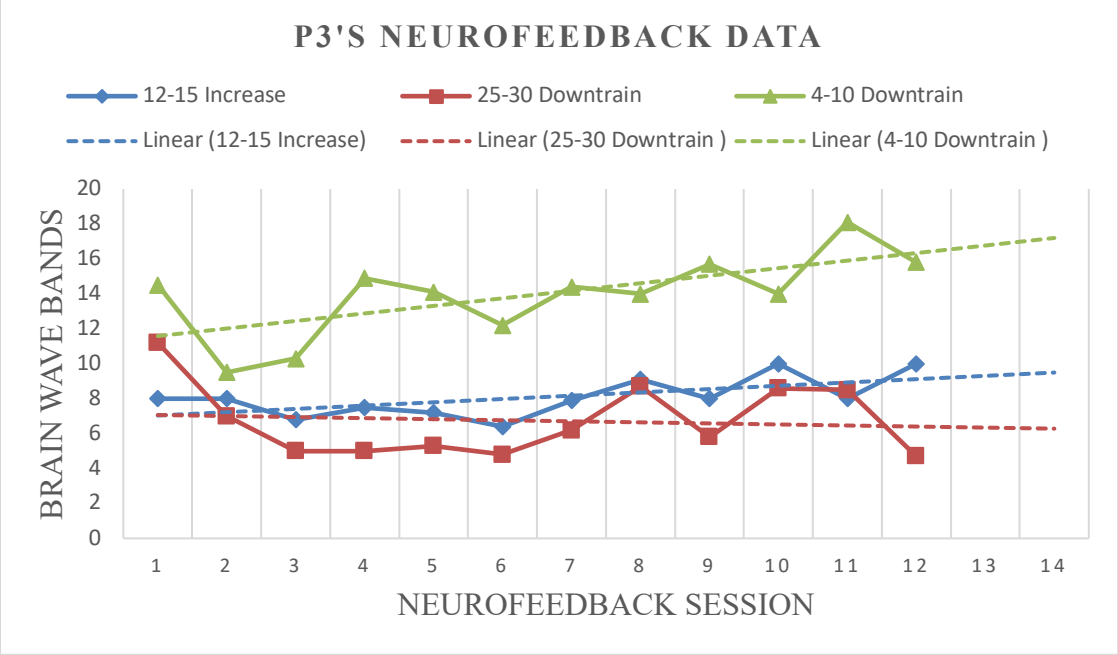
Note. EC = eyes closed; EO = eyes open.

Figure 5. *P3's CDS Visual Data From Clinical Research Solutions, 2021.*



Neurofeedback Session Data. Following P3's protocol, the visual representation (see Figure 6) of his training bands (12–15 Hz and 25–30 Hz) suggests a slight positive trend in the desired direction. However, the 4–10 Hz band does not exhibit a visual trend in the desired direction. It's plausible that artifacts, such as altered data due to P3's movements during sessions, could influence his data, particularly in the higher band range or alter the 25–30 Hz band. Nonetheless, there is a visual decrease in his 25–30 Hz band, aligning with protocol objectives. To delve deeper into the analysis, we utilized the NAP scores derived from P3's resulting brain bands. The NAP scores, presented in Table 6 are utilized to determine effect size.

Figure 6. *P3's Visual of Neurofeedback Data.*



Based on the NAP scores, all of P3's brain wave bands exhibited a medium effect. Notably, the 12–15 Hz band showed a significant change in the desired direction for his personalized protocol, approaching a large effect size. Further analysis involved examining P3's brain wave bands using the SMA. For the 4–10 Hz band, Model 4 yielded the best fit (R = 0.66, p = .07), indicating a progressive increase throughout both Phase A and Phase B, aligning with the observed trend. P3's 12–15 Hz band demonstrated optimal fit with Model 2 (R = 0.74, p = .02), depicting stability during Phase A followed by an increase during Phase B, in accordance with his protocol. Lastly, P3's 25–30 Hz band aligned most closely with Model 5 (R = −0.52, p = .09), illustrating a decrease during Phase A, followed by an immediate increase and subsequent decrease during Phase B.

Table 6

Nonoverlap of All Pairs Statistical Outcomes for P3

	S	Pairs	NAP	VARs	*z*	*p*	90% CI
4–8 Hz	20	36	0.778	156	1.60	.109	[−0.015, > 1]
12–15 Hz	28	36	0.889	156	2.24	.025	[0.207, > 1]
25–30 Hz	10	36	0.639	156	0.80	.423	[−0.293, > 1]

Note. S = distribution; Pairs = total pairs comparisons; NAP = nonoverlap of all pairs effect sizes; VARs = variance; *z* = *z*-score; *p* = *p*-value (p = .05); CI = confidence interval.

Participant 4

Participant 4 (P4) completed the demographic form, identifying herself as female, 59 years old, and of white ethnicity. P4 disclosed a family history of alcohol abuse but not drug abuse, with her first experience of alcohol abuse dating back to the age of 8. She reported being prescribed medication for thyroid gland issues, panic attacks and/or sleep (benzodiazepine), blood pressure, heartburn, and anti-nausea. Additionally, P4 acknowledged a diagnosis of anxiety and held a degree in accounting as her highest level of education. P4 expressed feeling "very satisfied" with her social support and exhibited motivation for AUD treatment. Clinicians noted P4's mild anxiety during most sessions, along with her perception that time passed quickly at the end of each neurofeedback session. Despite this, P4 generally maintained a content demeanor and consistently attended all scheduled sessions, displaying dedication according to clinicians' observations.

QEEG Findings. Manual artifacting was used for P4's EC pre data due to muscle tension with the remaining data being ran through automatic artifacting. Her protocol involved inhibiting 4–7 Hz, increasing 9–11 Hz, and inhibiting 25–30 Hz, specifically at the Oz site. However, P4's data presented an additional challenge as the neurofeedback program did not encompass training at the Oz site. Given that Oz is situated between O1 and O2, an additional step was necessary to incorporate data from both sites. This involved combining and averaging the data from O1 and O2 locations. In P4's 4–7 Hz band, there was a slight increase rather than the desired inhibition. Both her EC and EO data in the 9–11 Hz band showed a minor increase, consistent with her protocol. However, in the 25–30 Hz band, P4's data showed an approximate 1 standard deviation increase during EC, contrary to her protocol. Conversely, during EO, her 25–30 Hz band decreased by approximately 2 standard deviations, aligning with her protocol (see Table 7).

Table 7

Pre/Post qEEG Z-Score Data for P4

	EC Pre	EC Post	EO Pre	EO Post
4–7 Hz	1.09	1.51	0.01	0.68
9–11 Hz	0.16	0.18	0.18	0.49
25–30 Hz	0.95	2.40	4.38	2.29

Note. EC = eyes closed; EO = eyes open.

CDS. Like with every participant, neurofeedback clinicians prompted P4 to evaluate her current craving level. P4 consistently expressed how her recent outpatient program and neurofeedback had greatly reduced her craving thoughts. In each of the 16 data points, P4 consistently rated her cravings at the lowest level of 3. Consequently, we opted not to analyze her CDS data, as it would simply show a flat line graphically.

Neurofeedback Session Data. The visual trend lines (see Figure 7) for all P4's data pose challenges for visual analysis. To restate, P4's protocol involved inhibiting brainwave bands within the range of 25–30 Hz and 4–7 Hz. P4's bands being inhibited is somewhat reflected in the visual charts. Ideally, P4's 9–11 Hz band should show an increase over time, but the trend in the visual data is unclear.

None of P4's NAP scores (see Table 8) revealed a notable effect or significant change. P4's 4-7Hz band exhibited the strongest fit with Model 3 (R = −0.36, p = .17), albeit the correlation was weak. Similarly, P4's 9–11 Hz band, also displaying a weak correlation, demonstrated the closest fit with Model 1 (R = −0.31, p = .27), indicating a decrease in Phase A followed by an increase in Phase B. While this change is minor, the upturn in Phase B corresponds with the desired trend for P4's protocol. Conversely, the 25–30 Hz band did not exhibit a significant effect or change, aligning most closely with Model 1 (R = −0.32, p = .19).

Participant 5

The fifth participant (P5) identified as a 54-year-old male of white ethnicity. P5 noted that no one in his family had struggled with alcohol or drug abuse. He disclosed beginning alcohol use at the age of 15 and currently takes medications for blood pressure, cholesterol, blood thinning, depression, and naltrexone. While P5 hasn't received a formal diagnosis for a mental health disorder, he expressed grappling with feelings of depression and anxiety. Despite accumulating university credits, P5 did not complete his degree. He indicated feeling "satisfied" with his current level of social support. P5 demonstrated charisma and enthusiasm for neurofeedback sessions. However, due to his local job commitments, he faced challenges attending certain session times, leading to fluctuations in mood influenced by work stress. Additionally, P5 recently completed an outpatient program.

Figure 7. *P4's Visual of Neurofeedback Data*.

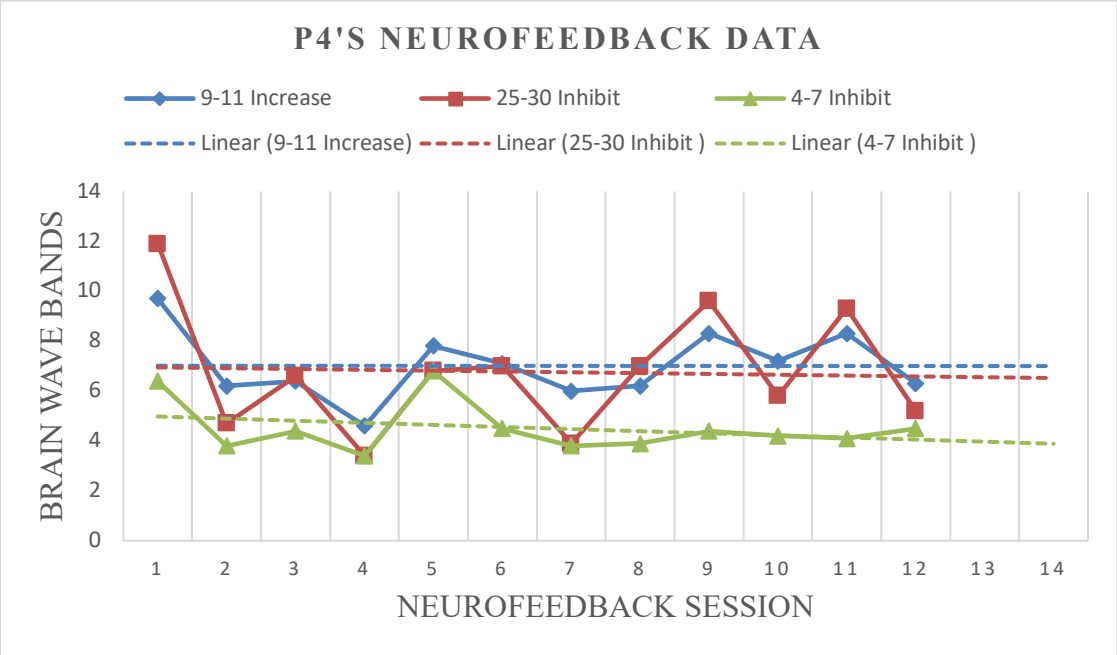


Table 8

Nonoverlap of All Pairs Statistical Outcomes for P4

	S	Pairs	NAP	VARs	*z*	*p*	90% CI
4–7 Hz	−9	36	0.375	156	−0.72	.471	[−0.821, 0.321]
9–11 Hz	1	36	0.514	156	0.08	.936	[−0.543, 0.599]
25–30 Hz	3	36	0.542	156	0.81	.810	[−0.487, 0.654]

Note. S = distribution; Pairs = total pairs comparisons; NAP = nonoverlap of all pairs effect sizes; VARs = variance; *z* = *z*-score; *p* = *p*-value (*p* = .05); CI = confidence interval.

QEEG Findings. We employed automatic artifacting for all P5's qEEG data, except for his post-EO data (see Table 9). Due to muscle tension issues, we opted for manual artifacting in this instance. P5's personalized neurofeedback protocol involved EO with the site location set at Cz, targeting the decrease of 4–10 Hz, increase of 12–15 Hz, and decrease of 20–30 Hz. Below are P5's *z*-scores. Reviewing P5's qEEG data, it appears he managed to marginally reduce his 4–10 Hz band during both EO and EC conditions, as well as his EC 20–30 Hz band. However, there was no significant change observed in his 12–15 Hz band. Notably, P5's pre- and post-qEEG data exhibited *z*-scores that did not raise any concerns and remained consistent with the norm.

CDS. P5's averages indicate Phase A (*M* = 6.13) and Phase B (*M* = 3), with a total mean of (*M* = 4.56) across all 16 sessions, reflecting changes in levels (see Figure 8). Furthermore, P5's test for level change yielded *R* = −0.72, *p* = .07, while the test for slope change resulted in *R* = −0.42, *p* = .35. Descriptive statistics for P5's data using OLS showed a slope of m = -0.39 and an intercept of b = 7.9, with a 95% confidence interval of [3.56, 5.69]. Bootstrapped autocorrelation for OLS utilizing the residuals revealed *N* = 16 with lag-1 = .17, *p* = .16. Phase results with the OLS residuals indicated a significant lag-1 of −0.71, *p* = .01 for Phase A (*n* = 8) and lag-1 of .00, *p* = .0001 for Phase B (*n* = 8). Thus, the overall OLS line showed no significance, both phase levels displayed a significant change.

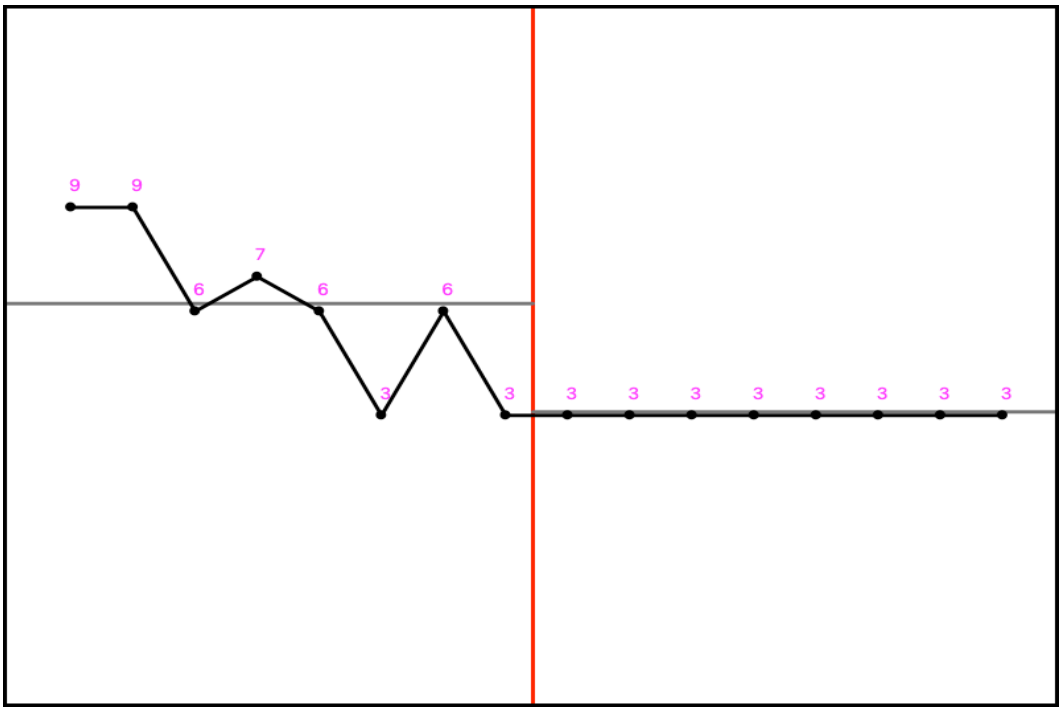
Table 9
Pre/Post qEEG Z-Score Data for P5

	EC Pre	EC Post	EO Pre	EO Post
4–10 Hz	0.72	0.23	0.22	−0.005
12–15 Hz	0.07	−0.08	0.07	0.06
20–30 Hz	0.28	−0.10	0.09	0.82

Note. EC = eyes closed; EO = eyes open.

Neurofeedback Session Data. From a visual standpoint, P5's data reveals coherent trend lines (see Figure 9). Following P5's protocol, the trend lines depicting the increase in 12–15 Hz and decrease in 20–30 Hz frequencies seem to show a positive trajectory. Throughout the sessions, P5 exhibited occasional jaw tension and minor movements. Table 10 presents an analysis of his session data using NAP scores for further examination.

Figure 8. *P5's CDS Visual Data from Clinical Research Solutions, 2021.*



P5's NAP scores for the 4–10 Hz and 20–30 Hz frequency bands showed weak or minimal effects, lacking significant values. Although the 12–15 Hz band displayed a medium NAP score aligning with the intended protocol trend, the associated p-value did not reach significance. Moving forward, we delved into analyzing P5's neurofeedback session data using SMA modeling. Notably, the 4–10 Hz band demonstrated the strongest fit with SMA Model 3 (R = 0.57, p = .04), characterized by an increase in Phase A followed by stabilization in Phase B. Similarly, P5's 12–15 Hz band data showed the closest fit with Model 3 (R = 0.40, p = .06). Conversely, his 20-30Hz band data aligned with Model 4 (R = −0.32, p = .18), suggesting a preferred decrease throughout the sessions.

AUDIT Results

All participants' AUDIT pre-post and follow-up data were composed into a single graph which is displayed below in Figure 10. Pre-time point data was collected during the participants' qEEG session, post was collected following their final neurofeedback session, and follow-up was collected 3–4 weeks after the neurofeedback sessions had concluded.

Figure 9. *P5's Visual of Neurofeedback Data*.

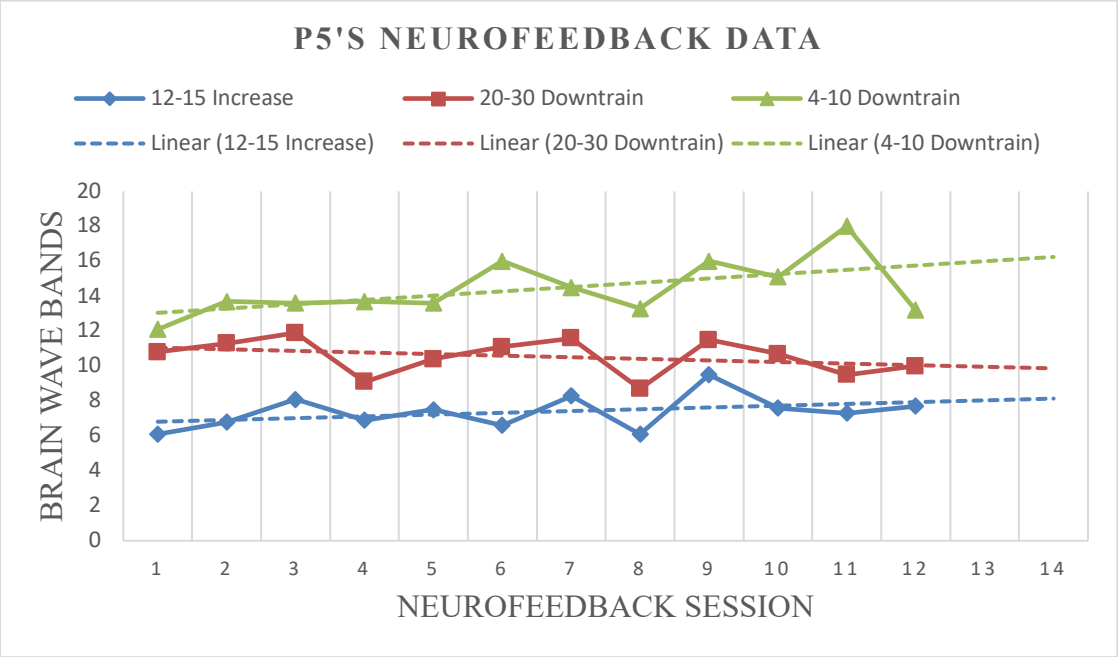


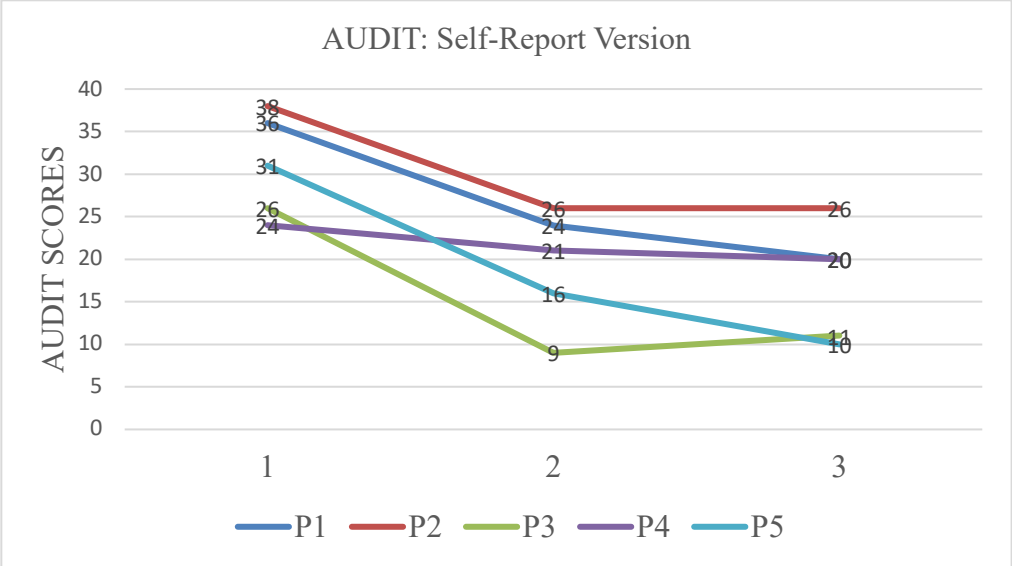
Table 10

Nonoverlap of All Pairs Statistical Outcomes for P5

	S	Pairs	NAP	VARs	*z*	*p*	90% CI
4–10 Hz	11	36	0.653	156	0.88	.379	[−0.265, 0.876]
12–15 Hz	17	36	0.736	156	1.36	.174	[−0.099, > 1]
20–30 Hz	−8	36	0.389	156	−0.64	.522	[−0.793, 0.349]

Note. S = distribution; Pairs = total pairs comparisons; NAP = nonoverlap of all pairs effect sizes; VARs = variance; *z* = *z*-score; *p* = *p*-value ($p = .05$); CI = confidence interval.

Figure 10. *Participants' Self-Report AUDIT Scores (Pre, Post, Follow-Up).*



Discussion

The main objective of this study was to investigate the efficacy of neurofeedback in curbing cravings and enhancing self-regulation through a combination of self-report evaluations and physiological measurements. Comparing pre and post qEEG data across participants revealed diverse outcomes. P1 experienced a desirable slight decrease in theta (4–8 Hz) activity and an undesirable decrease in alpha (8–10 Hz). However, P1 also exhibited a significant decrease in his beta (20–25 Hz) EC/EO conditions by 2 standard deviations. P2 achieved notable success in elevating his sensorimotor rhythm (SMR) by approximately 1 standard deviation. Similarly, P3 demonstrated effective results by enhancing their EO SMR by about 1 standard deviation. P4 managed to marginally increase alpha (9–11 Hz) and decrease EO beta (25–30 Hz), aligning with their prescribed protocol. P5 succeeded in slightly reducing theta (4–10 Hz) and EC beta (20–30 Hz).

Considering participants' neurofeedback sessions, outcomes also exhibited a spectrum of variability. Each participant was administered tailored neurofeedback protocols. While certain individuals displayed subtle shifts aligning with intended objectives, others evidenced notable changes characterized by moderate to substantial protocol goals. Furthermore, certain participants evinced indications of prospective enhancement in neurophysiological regulation contingent upon sustained participation in neurofeedback sessions. For neurofeedback session data, we employed SCRD methodology which enabled us to scrutinize individual transformations over the course of neurofeedback treatment comprehensively. This approach facilitated a nuanced understanding of shifts by analyzing data points from diverse vantage views. For example, P5's visual analysis exhibited promising trends, demonstrating alignment with his protocol. Notably, SMA revealed for his SMR (12–15 Hz) band a best fit with Model 3 (R = 0.40, p = .06), suggesting a Phase A increase followed by a stable Phase B, consistent with the prescribed protocol and but potentially indicating a learning plateau. Furthermore, P5's 20–30 Hz band demonstrated a consistent decrease across sessions, aligning well with Model 4 (R = −0.32, p = .18). Without supplementary analyses or the application of SCRD, discerning these subtleties might have proven challenging.

The CDS served as a pertinent instrument for self-reported assessment of craving intensity. Three out of four participants conveyed a discernible attenuation in alcohol cravings, a phenomenon persisting beyond the cessation of neurofeedback session. Conversely, P1's data indicated a marginal escalation in craving intensity during the concluding phase of the assessment. Subsequent scrutiny of pre, post, and follow-up evaluations employing the AUDIT unveiled that four out of five participants registered either diminished or static scores, indicative of a protracted reduction in overall alcohol consumption. Despite the diversity observed in participants' qEEG data and neurofeedback session outcomes, it was their self-reports of craving and alcohol use that yielded more illuminating insights.

Limitations and Implications for Research

The neurofeedback sessions took place in an academic environment rather than in a dedicated research facility. It is pertinent to note potential factors such as variations in session administration by students, including differences in threshold settings and varying levels of proficiency in neurofeedback techniques. While efforts were made to monitor sessions for electrode pops and other potential artifacts, it's important to acknowledge that session averages remained uncorrected for artifacts, which could potentially distort data. Additionally, many participants had either completed or had a few remaining outpatient addiction treatment sessions prior to their involvement in the current study.

The utilization of SCRD in the context of neurofeedback session data constitutes a novel methodological approach, meriting the attention from future scholars. Researchers may find it advantageous to either emulate the format employed in this study or explore alternative SCRD methodologies and analytical techniques. A notable attribute of SCRD methodologies lies in their capacity to discern subtle fluctuations in participant data across temporal dimensions (Lenz, 2015), thereby furnishing neurofeedback practitioners with valuable insights into requisite protocol modifications or instances of reaching learning plateaus. This tailored examination of individual physiological responses to interventions holds considerable potential for enriching the efficacy of neurofeedback services, particularly for professionals within counseling or psychological domains who seek to ascertain meaningful indices of client progress.

Conclusion

Our study explored neurofeedback for AUD using pre and post qEEGs, pre/post/follow-up AUDIT scores, and assessing craving desire over time. Five participants completed the study, with outcomes resulting in varied changes in their qEEG and neurofeedback session averages. We also utilized SCRD methods and analyses for recognizing individualized protocols and examining discrete complexities and trends in neurofeedback session averages. Repeated assessment of the CDS and AUDIT scores displayed promising results through self-reports of reduction in craving desire and alcohol use.

Author Declaration

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