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Psycho-neuro-biological Correlates of Beta Activity

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Abstract

Chronic stress and anxiety in everyday life can lead to sympathetic hyperactivity. This can be observed as behavioral, chemical, and neurological changes, including increased rumination, anxiety, and depression, and chemical changes in biological markers like homocysteine. In the EEG, increased beta (13–30 Hz) wave activity, especially high beta (> 20 Hz) has long been noted in anxiety states. However, recent research indicates that low beta waves (13–20 Hz) may play a role as well. The current paper presents a pilot study that assessed the Neurocycle's efficacy as a nonpharmacological mind-management therapy for people who struggle with anxiety and depression. We assessed psychometrics, blood-serum homocysteine levels, and quantitative electroencephalography (qEEG). Efficacy of the Neurocycle was demonstrated by improved psychometric self-assessment over the study. We observed a positive correlation between subject's low beta relative power and homocysteine levels. The findings validate the Neurocycle's efficacy for improving mental health as measured by behavioral, chemical, and neurological measures. Altogether, these findings support low beta's role in stress/anxiety manifestation given that its modulation significantly correlated with stress biomarkers in patients' blood samples and stress and anxiety self-assessments. Future work should expand these findings with larger datasets to confirm the ranges of healthy and maladaptive low beta.

Keywords: qEEG; beta; stress; anxiety; homocysteine

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Introduction

Increasing evidence suggests a correlation between resting-state electroencephalography (EEG) activity and anxiety symptoms in patients. Specifically, an increase of beta (13–30 Hz) and a decrease in alpha (8–12 Hz) waves have been associated with higher states of anxiety (Hammond, 2005; Ribas et al., 2018; Tharawadeepimuk & Wongsawat, 2014; Thompson & Thompson, 2007). Furthermore, studies evaluating methods of reducing anxiety have found that a decrease in beta activity is directly correlated with lower anxiety levels (Sherlin et al., 2010; Walker, 2010). These results have been consistently verified across multiple clinical conditions (i.e., PTSD, anxiety spectrum disorders), as well as across diverse anxiety treatment methods—from neurofeedback therapy to SSRI treatments to mindfulness and meditation—overall, confirming the relationship between beta wave activity and anxiety factors. However, which ranges of beta specifically play a role in this interrelation have still not been confirmed or normed in the literature. While many studies, including Díaz et al. (2019), have correlated high beta (which they defined as 22–30 Hz) with anxiety factors (Díaz et al., 2019; Tarrant et al., 2018; Tas et al., 2015; Walker, 2010), increased low beta (13–20 Hz) and overall beta activity (13–30 Hz) have also been correlated with anxiety, stress, and fear factors (Ribas et al., 2018). Thus, the current pilot study seeks to contribute to the field's developing

knowledge of the relationship between beta wave activity and anxiety to improve understanding of how beta modulation can be integrated into therapy modalities in the treatment of anxiety and depression-related mental health struggles.

Interrelation Between Beta Activity and Anxiety

The past decade of EEG studies has confirmed early seminal research of a relationship between beta wave activity—overall, low, and high—and an umbrella of anxiety factors. Recently, Ribas et al. (2018) provided percentage ranges at which beta activity and anxiety risk factors correlate; measuring at T3 and T4, their qEEG assessments identified levels of overall beta wave activity greater than 17% and high beta wave activity greater than 10% with subjects' fear, panic, insecurity, phobia, and anxiety. Though high beta wave activity is typically associated with anxiety and stress issues (Díaz et al., 2019; Tarrant et al., 2018; Tas et al., 2015; Walker, 2010), findings such as those from Ribas et al. (2018) help to clarify how both low beta and high beta are related to anxiety factors and an increased percentage of either can be correlated to increased anxiety factors. Direct modulation of beta wave amplitude via EEG-based biofeedback (neurofeedback [NFB]) therapy (21–30 Hz) for decreasing anxiety levels has also confirmed the interrelationship of beta wave activity and anxiety factors. Walker (2010) demonstrated how reductions in beta wave amplitude yielded statistically significant reductions in self-reported anxiety, indicating that decreased beta wave activity decreased anxiety symptoms. Moreover, heightened beta amplitudes have been correlated with anxiety in its manifestations in other mental health disorders, including posttraumatic stress disorder (PTSD; Roohi-Azizi et al., 2017) as well as bipolar disorder, schizophrenia, and addiction (Kesebir & Yosmaoğlu, 2020).

Therapy modalities for anxiety have also yielded important findings as to how beta activity and anxiety are related. Clinical studies of how pharmacological anxiety spectrum disorder treatments impact beta wave activity reveal that decreases in anxiety via methods such as SSRI treatment significantly correlate with reductions in prefrontal and frontal beta as well as high beta 6 months post-SSRI treatment (Tas et al., 2015). Another pilot study evaluating the effects of virtual reality on reducing anxiety found that relative power high beta activity decreased while low beta increased after sessions of treatment to decrease anxiety (Tarrant et al., 2018). Traditional holistic therapy modalities for anxiety such as yoga, meditation, and breathing techniques have also demonstrated reduced overall beta wave amplitude alongside improved mental state (Kaushik et al., 2020). As each study's reduction in anxiety via said therapeutic methods resulted in a change of beta wave activity, the multiple modalities of therapies used in these studies all validate the interrelationship between beta wave activity and anxiety factors.

The Search for Consistency in Defining the Beta-Anxiety Relationship

While numerous studies have focused on overall beta and high beta activity, there is a lack of consistency across the definitions of low versus high beta amongst different researchers using varying cutoff frequencies and a lack of consistency regarding which beta wave range is associated with anxiety factors. Between low and high beta, Díaz et al. (2019) suggests that low beta is associated with quiet and introspective thinking, which they termed the "healthy range" of beta. The researchers found that low beta (13–20 Hz) reduced in global coherence (a measurement of interhemispheric comodulation) from 55% to 15–20% when transitioning from a resting state to a demanding task, indicating that coherence within the lower beta frequencies was more closely associated with rest and could be differentiated from higher beta, which can be implicated in anxiety symptoms (Díaz et al., 2019). However, Milner et al. (2020) reported that amongst patients with high tinnitus-related distress, higher-amplitude low beta (13–20 Hz) activity was observed, indicating an association between increased low beta and ruminating cognitive-emotional processing. This type of internally focused thinking is associated with increased low beta and can result in more negative thinking types like rumination (Apazoglou et al., 2019) and anxiety in excess. Some of the most recent research has identified that high-amplitude low beta waves are related to a persistent sympathetic hyperactivity state that influences mental stress (Kopańska et al., 2022). These associations of differing aspects of anxiety with different ranges of Beta frequencies show how the neighboring frequency bands can interact with or be impacted by anxiety levels in distinct manners, and relationships must be assessed across the spectra to understand how anxiety manifests in the qEEG and can therefore be addressed therapeutically.

The Need for a Psycho-Neuro-Biological Approach

The World Health Organization (WHO; 2022) has reported that there has been a 13% increase in

mental health conditions and substance abuse disorders since 2019. However, despite this increase, current pharmacological treatments do not offer lasting treatment or resolution for these disorders (Ivanov & Schwartz, 2021), and the aforementioned lack of standardization across the field regarding the relationship between neural frequencies and mental health conditions has necessitated more research. Specifically, Newson and Thiagarajan (2019) called for researchers to contribute to the creation of a large qEEG database that could be assessed to inform and standardize norms of neurological function and related mental health outcomes. The need for more direct application of such neuroscientific research to the development of clinical practices and the treatment of mental illness has also been established (Ivanov & Schwartz, 2021). Given these identified gaps in the research and treatment of mental health conditions and their associated brain wave manifestations, a melding of neurophysiological, psychosocial, and biomedical streams of research are necessary to produce methods of jointly assessing biological and psychosocial measures and tailoring interventions in a patient-centered approach.

As such, the current pilot study takes a novel psycho-neuro-biological approach to the study of beta activity, their association with high/low stress and anxiety, and the effective management and resolution of anxiety symptomatology. To circumvent the lack of established cutoff low and high beta frequency definitions, a unique approach was taken that combines psychological, neurological, and biological measurements of participants' stress and anxiety levels to confirm the relationship between a reduction in anxiety and stress and its corresponding reduction in low beta wave activity for participants. Implementing the Neurocycle—a nonpharmacological, mind-management, and mind-directed neuroplasticity therapy modality for mental health improvement and anxiety and depression-related symptoms reduction—our study aimed to assess whether the Neurocycle intervention has a substantively positive impact on psychological and neurophysiological measures in a population of subjects with mental health and neurological symptoms. The following hypotheses were generated:

> H1: There will be change in the subjects' neurophysiological functioning, as measured by qEEG analysis of low beta relative power throughout the Neurocycle program.
>
> H2: There will be change in the subjects' biophysical anxiety symptoms throughout the completion of the Neurocycle program, as measured by blood serum homocysteine levels.
>
> H3: There will be positive change in the subjects' psychological well-being after the completion of the Neurocycle program, as measured by psychometric assessments of stress and anxiety.

Altogether, this psycho-neuro-biological approach will provide the more detailed neurophysiological data called for by Newson and Thiagarajan (2019) through a mapping of the psychological, neurological, and biological identifiers of anxiety, helping to describe low beta neural activity and its relationship with mental health conditions within the nexus of their neurophysiological, biological, and psychosocial tripartite nature.

Materials and Methods

Study Design

A double-blind randomized clinical trial (RCT) pilot study was selected for its suitability in determining if an intervention has a meaningful effect on key outcome measures of interest and its ability to establish high confidence in causal claims (Spieth et al., 2016). The study design, instruments, and protocol were approved by the Sterling Institutional Review Board (approval ID no. 7281-RPTurner). A total of 14 participants were recruited based on power analysis of convenience sampling; *a priori* power analysis was conducted using G*Power 3.1.9.2, and, assuming a moderate to high effect size ($f = 0.30$, power $[1 - \beta] = 0.80$) and alpha (α) of 0.05 for a between-within subjects analysis of variance (ANOVA) with two groups and six repeated measures, the necessary sample size was verified as 12 to detect a significant effect in the population (Cohen, 1988; Erdfelder et al., 1996; Faul et al., 2007) and an additional two participants for potential attrition during the study period. Participants for this study were recruited from patients and employees of Network Neurology and from additional flyers for this clinical trial posted around Network Neurology and at local colleges within a 15-mile radius of the Network Neurology office. To ensure participants met the recruitment criteria of preexisting anxiety and/or depression, the research team recruited a total initial pool of 30 recruits in a prescreening phase to reach the desired sample size of 14 participants for the pilot study given the current prevalence of depression and generalized anxiety

disorders in clinical settings (70–80% [> 14 on the HAM-D; Trivedi et al., 2006] and 50% [> 18 on the HAM-A; Ruiz et al., 2011], respectively).

To select the 14 participants from the initial 30 recruits, inclusion and exclusion criteria were applied. The inclusion criteria for this study consisted of: (a) consent to participate in the study; (b) 18 years of age or older; (c) a score of 14 or above on the HAM-D depression scale; (d) a score of 18 or above on the HAM-A anxiety scale; and (e) completion of the pilot study. The exclusion criteria for this study consisted of: (a) prior experience or familiarity with Dr. Leaf's books, applications, or teachings (due to possible study bias); (b) concurrent diagnosis of epilepsy or refractory depression (due to complexity of comorbid diagnoses); (c) current prescription of more than 3 psychotropic medications (due to confounding factors in brain analysis and masking of symptoms); (d) a score of less than 14 on the HAM-D depression scale; (e) a score of less than 18 on the HAM-A anxiety scale; and/or (f) incomplete study participation.

After the final 14 participants were selected, they were provided with an Informed Consent explaining the purpose and background of the study, its procedures, its duration (including their right to cease participation at any point during the study), the risks and discomfort associated with the assessments (e.g., potential discomfort from blood draw and qEEG procedures), potential benefits to the participants, costs (none) and compensation for the study (access to the Neurocycle app), protection of their privacy, and contact information for the study personnel. The subjects were randomly assigned to the "treatment" group (n = 7), the Neurocycle, or the "control" group (n = 7), which received no special attention beyond the standard of care of their physician. During the study, attrition occurred following baseline measurements in both groups (control: attrition of n = 1, for a final total of n = 6; treatment: attrition of n = 2, for a final total of n = 5). Replacement of missing data was not a possible strategy for addressing attrition given that individualized brain mapping could not be replaced by random values. However, attrition bias was avoided by removing any partial data from participants who dropped out from the final dataset as these participants violated the inclusion criteria of completing the pilot study. Therefore, their entire profiles were removed from the final samples, and data integrity was maintained.

Materials

The intervention utilized the Neurocycle program hosted on the Neurocycle app. The Neurocycle (Leaf, 1997, 2021) is a 63-day mind-directed self-help mental health program created by Dr. Caroline Leaf that is implemented in three phases of 21 days for a total of 63 consecutive days. These three phases are administered through the Neurocycle app, in which participants are directed via daily audio and video recordings through the five-step Neurocycle process of Gather Awareness, Reflect, Write, Recheck, and Active Reach, which provide a scientifically validated framework for participants to identify, face, process, and manage intrusive toxic thoughts that cause distress, including symptoms of anxiety and depression (Idris, 2020; Leaf, 1997, 2021). This approach acknowledges that individuals can reconceptualize and take control of their mental health through mind-management and provides development in the required skills to actualize the benefits of mindfulness: self-regulation, resilience, reconceptualization, and exposure (Shapiro et al., 2006).

Measurements, Instruments, and Data Collection

The psycho-neuro-biological effects of the program were assessed using a novel three-phase structure in a pilot study to test the effectiveness of the Neurocycle. The psychological effects of the Neurocycle were measured by the Leaf Mind Management (LMM) scale and triangulated with the Hospital Anxiety and Depression Scale Anxiety and Depression subscale (HADS-A & HADS-D; Bjelland et al., 2002) and the BBC Subjective Well-Being Scale (BSC; Pontin et al., 2013). The neurophysiological effects of the Neurocycle were assessed using surface qEEG functional analysis. The psychological and neurophysiological effects were then confirmed in bloodwork analysis to measure participants' homocysteine levels, which are known to increase alongside stress, anxiety, and depression (Kevere et al., 2014). This combined approach was designed to address criticisms in the field of psychology that self-assessments are inherently flawed measurement tools on their own due to biases that can be beneath our consciousness or socially motivated (Chen et al., 2013; Karpen, 2018). Additionally, the tripartite approach addresses the lack of consensus in the field of electroencephalography regarding what constitutes high and low beta frequencies and their exact relationship with stress and anxiety in brain function by providing a third measurement to confirm a change in anxiety and stress. The assessments were administered in a staged format that captured key insight into the changes in participants' stress

and anxiety across six distinct time periods: preintervention (Day 0), on Days 7, 14, 21, and 42, and postintervention on Day 63. The schedule of assessment administration is provided in Table 1 below, and descriptions of each assessment phase follow.

Table 1
Mean and Standard Errors of Confirmation Measures and Correlations for Treatment Group

Measure	Pre-Screen	Day 0	Day 7	Day 14	Day 21	Day 42	Day 63
Clinical Anxiety (HAM-A)	X						
Clinical Depression (HAM-D)	X						
Psychological Effects (BBC-SWB)		X	X	X	X	X	X
Self-Report Anxiety (HADS-A)		X	X	X	X	X	X
Self-Awareness and Mind Management of Stress and Anxiety (LMM)		X	X	X	X	X	X
Neurophysiological Effects (qEEG)		X			X		X
Bloodwork (Homocysteine)		X			X		X

Neurophysiological Assessment

Participants underwent three qEEG sessions for neuroimaging analysis on Days 0, 21, and 63 to assess neural activity changes from baseline to the completion of the first phase of the intervention (Day 21) and then from this phase to the completion of the entire program (Day 63). For each recording, subjects were seated in a quiet, comfortable room and allowed to relax in a comfortable armchair. Nineteen electrode sites were located according to the international 10-20 system, cleaned using a mild abrasive gel (Nu-Prep), and electrodes tested to obtain impedances below 5 kΩ. Subjects were instructed to sit quietly without movement while EEG was recorded at a 250 Hz sampling rate (Mitsar EEG-201). Subjects were prompted to relax to reduce muscle artifact if noted by the researcher at time of recording. Participants' qEEG was recorded for 10 minutes with their eyes open and another 10 minutes with their eyes closed. Only eyes-open data are reported on in this paper.

Psychological Assessment

Self-assessment of psychometric indicators was provided by participants during all six key stages of the intervention's administration: Days 0, 7, 14, 21, 42, and 63. The primary assessment tool implemented was the LMM scale, which was designed by the principle investigator (PI) to assess autonomy, awareness, toxic thoughts and isolation, toxic stress and anxiety, barriers and challenges, and empowerment and life satisfaction. The LMM has shown strong structural validity and reliability in testing (publication pending); Cronbach's alphas for subfactors ranged from .62 to .90 with an overall factor that ranged from .77 to .80. The LMM measures subjects' changes in awareness, processing, reconceptualization, and control of reactions and responses to the circumstances of life that cause feelings of anxiety and depression. As such, it is a tool for assessing participants' mindfulness of their mental health and the necessary mediators—self-regulation, resilience, reconceptualization, and relived experience—to respond healthily to stress and anxiety. Improvements in stress and anxiety can be measured by increases in the autonomy, awareness, and empowerment subscales alongside decreases in the toxic thoughts, toxic stress, and barriers subscales. To validate the LMM assessment in this study, traditional measures of anxiety, stress, and depression were also administered, including the HADS-A, HADS-D (Bjelland et al., 2002), and BBC-SWB (Pontin et al., 2013) instruments. The HADS-A and HADS-D are 4-point Likert scale each with seven items possessing strong validity and reliability with Cronbach alphas that range from .68 to .89 (Bjelland et al., 2002). Likewise, the BBC-SWB is a 5-point Likert scale with 24 items and has been found both a reliable and valid instrument that also possesses strong Cronbach alphas that range from .74 to .95, indicating very strong reliability (Pontin et al., 2013). By administering these instruments across six time periods, the evolution of change in the participants' well-being, depression, and stress and anxiety levels could be tracked

alongside and between qEEG and blood measurements, filling in the qualitative explanation of the participants' mental health changes.

Biological Assessment

Participants were sampled for blood-measured homocysteine levels, elevated levels of which are known to be associated with an individual's elevated stress and anxiety levels and direct neurotoxic effects (Aghayan et al., 2020; Chung et al., 2017) given that this sulphurated amino acid is responsible for mediating methylation, which is critical for nervous system balance and health (Kennedy, 2016). This assessment was performed in three parts. Blood samples were drawn by a contracted phlebotomist in 10 mL vials preintervention on Day 0, after the initial phase of the intervention on Day 21, and postintervention on Day 63. Blood amino acid analysis for homocysteine levels was then performed by a contracted lab and reported to the researchers as follows: normal range: 5–15 mcmol/L; moderately elevated range: 15–30 mcmol/L; intermediately elevated range: 30–100 mcmol/L; and severely elevated range: < 100 mcmol/L (Haldeman-Englert et al., 2022).

The qEEG data for each subject was preprocessed using the Harvard Automated Preprocessing Pipeline for Electroencephalography (HAPPE; Gabard-Durnam et al., 2018) to remove artifactual contributions to the data such as eye, muscle, electrical, and movement-related artifacts. The resulting data was analyzed using a sliding window FFT to obtain power spectral density estimates for each electrode site. Then, relative power was calculated for each frequency band relative to the total power in the 1–80 Hz range. Relative power was used for analyses to allow direct comparison from one subject to another, controlling for interpersonal differences in overall EEG amplitude. In this study, all-electrode-averaged low beta relative power (13–20 Hz) was analyzed.

The data gathered from the qEEG, bloodwork, and psychometric assessments were analyzed altogether using IBM SPSS v27. Overall study analysis was examined with the original planned mixed (between-within subjects) ANOVA with the two groups (treatment and control) over six repeated measures (the pretest and five follow-up measures) was performed. The overall main effects of group, time, and the interaction of group and time were assessed to determine if the effects of the intervention had an impact on the study outcomes. Pairwise group comparisons over time were calculated using the Bonferroni method to adjust for multiple comparisons. To examine the specific hypotheses outlined in this paper, linear multiple regression models and simple regressions were conducted to examine the relationships among the specific variables of interest, as well as nonparametric correlations to assess potential triangulating relationships. The alpha (α) level for this pilot study was set at .10.

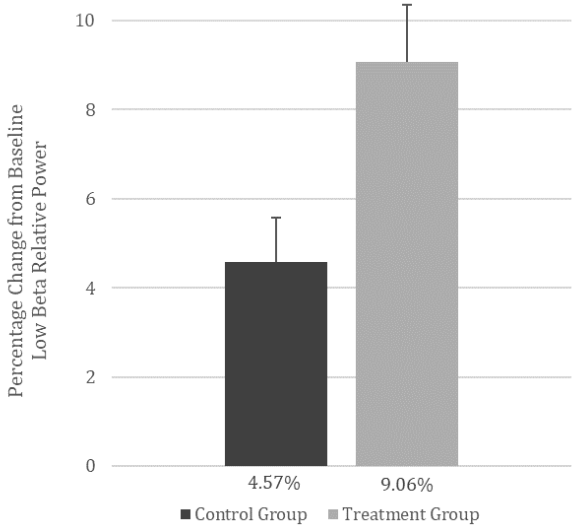
Results

Our multivariate linear regression model showed that the LMM toxic-stress subscale and homocysteine levels were significant predictors and accounted for 41.4% of the variance of global average low beta relative power changes from Day 1 to Day 63, $F = 4.49$, $p < .05$, $R^2 = 41.4\%$. Looking at the individual predictors of the model, we can see that the strongest indicator was the change in homocysteine with a beta coefficient (standardized) of .613 ($p = .036$). Additionally, the LMM toxic stress subscale change was meaningful (moderate) at .395 ($p = .142$). These results indicate that the greater change in homocysteine was a prime predictor of change in average low beta relative power. Furthermore, within participants' change in the LMM toxic stress over the course of the study, greater change in toxic stress was related to greater change in average low beta relative power regardless of homocysteine levels.

These results confirmed H1, H2, and H3. Overall, participants' average low beta relative power changes correlate with the trajectory of change in neurophysiological functioning during the Neurocycle. At baseline there was no statistically significant difference in low beta relative power between the treatment and control group, $t(5.89) = 1.60$, $p = .118$, but by Day 21 we observe a statistically significant difference between the groups, $t(9) = 1.71$, $p = .089$, see Figure 1.

The neurophysiological improvement is confirmed in the correlations of decreased LMM Toxic Stress subscale scores with decreased HADS-A Anxiety ($\rho = .894$, $p < .001$) and HADS-D Depression ($\rho = .592$, $p = .046$) subscale scores for intervention participants. Together, these correlations validated H1.

Figure 1. *Low Beta (13–20Hz) Relative Power Percentage Change From Baseline to Day 21 for the Treatment and Control Groups.*



H2 was confirmed through the corresponding correlation between average low beta relative power and blood serum homocysteine levels (ρ = .755, p = .007). Given that homocysteine and average low beta relative power wave activity decreased from Day 21 to 63, as self-reported anxiety and depression improved as evidenced by the lowered HADS-A and LMM Toxic Stress scores (ρ = .894, p-value < .001), biophysical anxiety symptoms were clearly lessened. Thus, positive change occurred, confirming H2.

Additionally, the same psychometric assessments confirmed that intervention participants experienced a reduction in their anxiety from Day 21 to Day 63 of the program. Analyses confirmed a statistically significant change in participants' low beta relative power (M_{diff} = .0052, SE = .003, t = 1.75, p = .078). Due to low sample sizes in the pilot study, multivariate correlational analyses by group were not possible; however, there are corresponding relationships of percent change low beta relative power with change in homocysteine levels (ρ = .852, p = .033), and the psychometric tests of depression and anxiety via lowered HADS-A (ρ = .866, p = .067) and LMM Toxic Stress scores (ρ = .689, p = .099), see Table 2. Thus, H3 was confirmed.

Table 2
Mean and Standard Errors of Confirmation Measures and Correlations for Treatment Group

Measure	Day 21 Mean	Day 21 SE	Day 63 Mean	Day 63 SE	% Change Low Beta Correlation
Low Beta Relative Power	0.118	0.005	0.112	0.007	-
Bloodwork (Homocysteine)	187.80	23.64	173.69	17.06	.852*
Self-Report Anxiety (HADS-A)	7.25	3.25	7.00	3.03	.866*
Self-Awareness and Mind Management of Stress and Anxiety (LMM)	6.00	.32	5.25	.37	.689*

Note. *Significant correlation (ρ) with percent change from baseline low beta relative power, p < .10.

Discussion

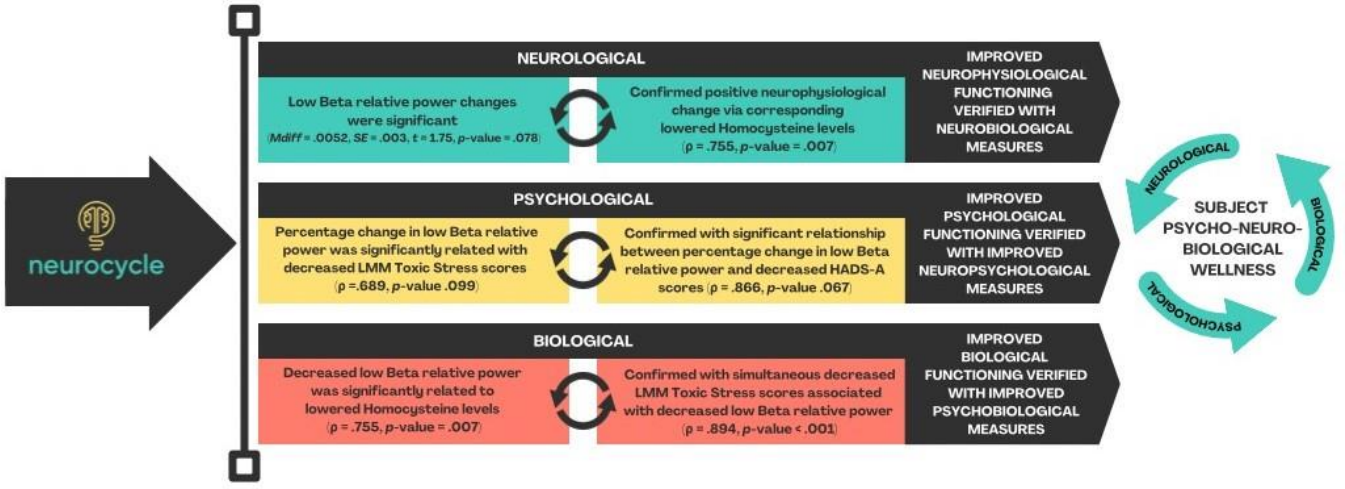
Though low beta has historically been associated with positive mental state aspects, such as focused energy (Abhang et al., 2016; Díaz et al., 2019; Tarrant et al., 2018), the musing thought capability of this wavelength can become detrimental if too high a relative power is reached (Apazoglou et al., 2019). For instance, abundant high-amplitude low beta wave activity is related to persistent sympathetic hyperactivity that influences mental stress (Kopańska, 2022). Thus, the relative power of low beta appears to be a factor in the modulation between the self-monitoring and internal focus capabilities of beta and more toxic applications of reflective thought, such as rumination (Apazoglou et al., 2019). Figure 2 displays how the psycho-neuro-biological results of the current study support this understanding of low beta wave relative power modulated in relation to overall subject wellness.

The current study's results suggest that changes in global average low beta relative power and blood serum homocysteine levels are associated with

participants' anxiety and stress, as indicated by the HADS-A and LMM Toxic Stress scale. The results from the three different manners of measurement—neurological measurement with the qEEG, psychological measurement with the LMM and HADS-A, and biological measurement with homocysteine—show interactive, statistically significant relationships that validate the data through each field. While all subject's low beta increased from the beginning of the study to Day 21, that increase was significantly stronger in the treatment group as they engaged in the treatment process, as shown in Figure 1. It is important to acknowledge that improving mental health is not a linear process from start to end and experiencing an increase in symptoms before they get better is common across many therapeutic modalities. Throughout the rest of the study from Day 21 to 63, each independent measurement modality was verified by two other modalities, as described in Figure 2. The positive neurophysiological change resulting from the decrease in global average low beta relative power from Day 21 to Day 63 of the study was supported by the biological measurement of the participants' decreased homocysteine levels. Following, the improved biological functioning resulting from the decreased blood homocysteine levels was verified with psychosocial assessments of participants' decreased stress and anxiety. Coming full circle, this improved psychological functioning was then verified by correlating both sets of significant results from the two psychosocial assessments—the HADS-A and LMM Toxic Stress scale—with their significant association with global average low beta relative power. While the finding of association between global low beta, homocysteine, and psychometric measure of stress was found over the entire set of participants, only the treatment group showed a significant reduction in symptoms as shown by the decrease in the HADS-A and LMM Toxic Stress scale. It is important to note that the qEEG recordings were made during an at-rest condition that was not designed to elicit any specific emotional response, which may account for some of the differences in beta frequencies engaged between this study and other findings in the qEEG literature featuring studies that utilized varied levels of stressors (Díaz et al., 2019; Ribas et al., 2018; Tharawadeepimuk & Wongsawat, 2014).

Answering Newson and Thiagarajan's (2019) call for more qEEG contributions toward the understanding of neurological function and related mental health outcomes, these tripartite statistical relationships have therefore shown that low beta is involved in the management of anxiety. Furthermore, the current data indicates that lower low beta relative power may be associated with improved perspectives of subjects' stress and anxiety. This finding emphasizes the importance of addressing low beta when dealing with anxiety and mental well-being, thereby emphasizing the significance of the Neurocycle as a mindfulness tool that directly interfaces with low beta wave activity. As this was a

Figure 2. *Summary of the Psycho-Neuro-Biological Impact of the Neurocycle Program (Days 21–63): Global Average Low Beta Relative Power, Homocysteine, and Psychosocial Measurements.*



Note. LMM = Leaf Mind Management Scale; HADS-A = Hospital Anxiety and Depression Scale: Anxiety Subscale.

pilot study, future research should confirm these relationships with larger data sets and longitudinal studies to provide normative ranges for understanding low beta's involvement in anxiety magnification and mitigation. Such ranges could inform therapy modalities and improve patient care with treatments that directly address the manifestation of anxiety at its psycho-neuro-biological roots (Ivanov & Schwartz, 2021).

Conclusion

The present pilot study was conducted to assess the efficacy of the Neurocycle for improving the psycho-neuro-biological wellness of participants as measured by global average low beta relative power, homocysteine blood levels, LMM Toxic Stress subscale scores, and HADS-A scores. Neurophysiological changes were observed as an indicator of improved mental wellness through improved psychosocial state as indicated by decreased LMM Toxic Stress subscale scores and decreased HADS-A anxiety scores. Neurological and mental improvement was validated with measurement of decreased homocysteine and low beta levels, from Day 21 to Day 63 of the study, coinciding with decreased self-report of symptoms of stress and anxiety. The correlation of these results provides novel support for the connection between low beta and poor mental health indicators such as rumination or active anxious focus.

Though high beta is typically associated with stress and anxiety, the reduction of low beta wave amplitude in the current results was significantly associated with lowered participant stress and anxiety, revealing that both low and high beta are involved in the mind management of stress and anxiety. Altogether, this study's psycho-neuro-biological approach provides evidence for the efficacy of the Neurocycle for mind management and stress and anxiety reduction. Continued work should expand the data from this pilot with larger-scale and longitudinal research to establish the exact ranges of beneficial versus maladaptive low beta.

Author Declarations

Dr. Caroline Leaf has ownership in Switch on Your Brain and the Neurocycle and financially benefits from royalties for the intellectual property that is subject to evaluation or improvement through the research presented here. Financial interest concerns were addressed through the adoption of a double-blind research design and involvement of a third-party research consultation firm. There are no conflicts of interest or grant support to disclose.

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