

Efficacy of an Alpha Neurofeedback Training in the Treatment of Anxiety and Depression of a Group of Patients: A Pilot Study

Alexandra Glink^{1,2,3*}, Maria Eugenia Gras^{1,2}, and Montserrat Planes^{1,2}

¹University of Girona, Quality of Life Research Institute, Girona, Spain

²University of Girona, Department of Psychology, Girona, Spain

³Neuroon Clinic Psychology Center, Empuriabrava, Girona, Spain

Abstract

Background. Anxiety and depression are highly prevalent in the general population and primary care. While alpha rhythm (8–12 Hz) stimulation has been shown to reduce anxiety, its impact on broader emotional well-being, including depressive symptoms, is less studied. **Objective.** This exploratory study examined the effects of alpha neurofeedback training on anxiety and depression in adults. **Methods.** Fourteen female participants with anxiety and depressive symptoms were randomly assigned to an intervention group ($n = 7$) or a waitlist control group ($n = 7$). Psychological symptoms and alpha brainwave activity were assessed before and after the intervention. After the initial phase, the waitlist participants also received the training, forming a quasi-experimental design. **Results.** Ten sessions of alpha neurofeedback significantly reduced anxiety in both experimental and quasi-experimental phases. Depressive symptoms decreased notably only in the quasi-experimental phase, when all participants received the intervention. Alpha amplitude increased, and improvements in anxiety and depression were correlated, though not statistically significant. **Conclusions.** These preliminary findings suggest that alpha neurofeedback may be an effective nonpharmacological intervention to reduce anxiety and depression in adults. Results are exploratory, highlighting the need for larger, diverse samples and follow-up assessments to confirm the durability of effects.

Keywords: alpha; neurofeedback; anxiety; depression

Citation: Glink, A., Gras, M. E., & Planes, M. (2026). Efficacy of an alpha neurofeedback training in the treatment of anxiety and depression of a group of patients: A pilot study. *NeuroRegulation*, 13(2), 119–126. <https://doi.org/10.15540/nr.13.2.119>

***Address correspondence to:** Dr. Alexandra Glink, Neuroon Clinic Psychology Center, C/Pla de Roses 15, E-17487 Empuriabrava, Girona, Spain. Email: alexandraglink@gmail.com

Copyright: © 2026. Glink et al. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC-BY).

Edited by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA

Reviewed by:
Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA
Randall Lyle, PhD, Mount Mercy University, Cedar Rapids, Iowa, USA

Introduction

Depression and anxiety disorders are among the most prevalent psychological conditions in both the general population and primary care settings. These highly disabling disorders result not only in substantial human suffering and loss of health but also in significant economic costs due to reduced productivity and healthcare burden (Demertzis & Craske, 2006; Kroenke et al., 2007). Given their strong association with morbidity and mortality (Kessler et al., 2007; Richards, 2011), early identification and effective treatment of both conditions are crucial.

In addition to traditional therapeutic approaches such as psychotherapy and pharmacotherapy, neurofeedback training has emerged as a promising complementary intervention for individuals diagnosed with anxiety and depression. These disorders are characterized by abnormal patterns of electrical activity within neural networks involved in emotion regulation and behavior (Menon, 2011).

Neurofeedback is a form of biofeedback that provides real-time information about cortical activity through electroencephalography (EEG). Using auditory or visual feedback, participants learn to voluntarily modulate specific EEG frequency components (Lubar, 1997). These components may

include individual brainwave frequencies, ratios between them, or measures of coherence. The ultimate goal of neurofeedback training is to enable individuals to optimize their brain activity, thereby promoting beneficial changes in emotional regulation, cognition, and behavior (Niv, 2013).

Early studies of neurofeedback focused on enhancing alpha activity in individuals with anxiety disorders, as increased alpha power has been associated with greater relaxation and reduced arousal (Hardt & Kamiya, 1978). Subsequent research has provided accumulating evidence supporting the efficacy of neurofeedback in the treatment of anxiety and depression. Reviews by Hammond (2006a, 2006b) and Moore (2000) summarized multiple studies demonstrating clinically significant improvements following neurofeedback interventions. Typically, protocols targeting parietal alpha enhancement are used for anxiety, whereas frontal alpha asymmetry training is commonly applied in the treatment of depression (Baehr et al., 2001; Choi et al., 2011).

In primary care, depressive and anxiety disorders frequently co-occur, and patients often present with somatic rather than psychological complaints, such as back pain, chest tightness, palpitations, sleep disturbances, appetite changes, or fatigue. This overlap underscores the urgent need to develop and evaluate new, effective, and noninvasive interventions targeting both disorders simultaneously.

The aim of the present study was to examine the effects of an alpha enhancement neurofeedback protocol—typically employed in the treatment of anxiety—on individuals presenting with both anxiety and depressive symptoms.

Materials and Methods

Participants

This study was conducted at a psychological center. Fifteen patients aged between 25 and 60 years with anxiety and/or depression were initially considered for participation. Only one male contacted the center to participate, and he was excluded to maintain intergroup similarity. After applying the exclusion criteria, 14 female participants (mean age = 37.85 years, $SD = 11.07$) were included in the analysis.

Ethics Statement

This exploratory study was conducted at a psychological center with adult participants who provided written informed consent prior to participation. Although the study did not receive formal approval from an Institutional Review Board (IRB), all procedures were performed under academic supervision and in accordance with the ethical standards of the Helsinki Declaration (World Medical Association, 1996). Participants were treated respectfully, their privacy was ensured, and data were collected and stored confidentially. The study represents preliminary, hypothesis-generating research in the field of neuroregulation.

Procedure

Written informed consent was obtained from all participants. A pro forma data sheet specifically designed to gather psychosocial information was used. Participants were divided into two groups by allocating consecutive patients alternately.

Anxiety and depression levels were initially assessed (Phase 1) using the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). The HADS authors propose three cut points for each subscale (anxiety and depression): normal (0–7), doubtful disorder (8–10), and clinically relevant problem (≥ 11).

Initial alpha brainwave amplitude was measured and recorded. One group of seven participants (intervention group) received alpha neurofeedback training twice per week for 5 weeks. The other group served as a waitlist control and did not receive training during this period. In Phase 2, after the 5-week treatment period, anxiety, depression, and alpha amplitude were reassessed in both groups. In Phase 3, the waitlist control group received the same neurofeedback training as the intervention group and data were reassessed, resulting in a combined intervention group of 14 participants.

Electroencephalography

EEG data were collected at the P4 electrode (parietal cortex), using the International 10/20 electrode system. Gold cup electrodes were placed in a monopolar montage, with the ground electrode on the right ear and the reference electrode on the left ear. The scalp was prepared with NuPrep skin prep gel, and electrodes were adhered with Ten20 conductive paste (both from Weaver and Company, Aurora, CO).

Table 1
Distribution of Participants According to Group, Sociodemographic, and Clinical Variables

Sociodemographic Variables		Group	
		Intervention (<i>n</i> = 7)	Control (<i>n</i> = 7)
Gender	Male	0% (0)	0% (0)
	Female	100% (7)	100% (7)
Age	Between 25 and 60 years	100% (7)	100% (7)
Clinical Variables			
Anxiety or depression	Yes	100% (7)	100% (7)
	No	0% (0)	0% (0)
Symptoms	Muscular pain	42.9% (3)	14.3% (1)
	Insomnia	0% (0)	42.9% (3)
	Gastrointestinal discomfort	42.9% (3)	28.6% (2)
	Headache	14.3% (1)	14.3% (1)
Medication or substance that may influence the nervous system	Yes	0% (0)	0% (0)
	No	100% (7)	100% (7)

Note. Percentages and frequencies are presented by row.

Data were collected using the Nexus-10 MKII device and Biotrace+ software (Mind Media BV, Echt, NL) at a sampling rate of 256 Hz, ensuring skin impedance levels were below 10 k Ω . Raw data were processed using a Butterworth bandpass filter, and average peak-to-peak amplitudes (in μ V) were calculated for the alpha frequency band (8–12 Hz). These amplitudes were used to guide neurofeedback training.

Neurofeedback

Participants underwent 10 sessions of alpha-enhancement neurofeedback over 5 weeks. Individual training sessions included two 15-min feedback screens (Smiley face and Waterfall). A threshold of 10 μ V was set, with feedback screens pausing if alpha amplitude fell below this threshold. Positive reinforcement was provided when alpha amplitude exceeded the threshold. Mean alpha amplitude during training was monitored using a digital counter within the Biotrace+ software. A digital counter system within the Biotrace+ software

was employed to determine the mean alpha amplitude that was occurring during training.

Statistical Analysis

All parametric statistical analyses were performed using SPSS, Version 14.0 for Windows. Due to the small sample size, nonparametric tests (Mann-Whitney, Wilcoxon, and Friedman) were used. For clarity, mean values of the variables are also reported.

Results

Anxiety and Depression Levels Pretreatment and Posttreatment

All participants were assessed using the HADS before and after neurofeedback training. Tables 2–5 present the mean scores and standard deviations for anxiety and depression subscales in both the experimental and quasi-experimental designs.

Table 2

Mean Scores and Standard Deviation (in Brackets) of the HADS Anxiety Subscale by Group, Before and After the Intervention, With Results of the Mann-Whitney U Test

Experimental Design	Pre x (dt)	Post x (dt)
Intervention group (n = 7)	11.29 (3.86)	7.43 (2.64)
Control group (n = 7)	12.00 (3.79)	12.23 (3.60)
Z (p)	-0.06 (.95)	2.20 (.03)

Table 3

Mean Scores and Standard Deviation (in Brackets) of the HADS Anxiety Subscale, Before and After the Intervention, With Results of the Wilcoxon T Test

Quasi-Experimental Design	Pre x (dt)	Post x (dt)
Intervention group (n = 14)	11.79 (3.62)	8.00 (2.75)
Z (p)		-3.194 (.001)

Table 4

Mean Scores and Standard Deviation (in Brackets) of the HADS Depression Subscale by Group, Before and After the Intervention, With Results of the Mann-Whitney U Test

Experimental Design	Pre x (dt)	Post x (dt)
Intervention group (n = 7)	9.57 (4.86)	6.14 (3.76)
Control group (n = 7)	9.29 (3.82)	9.57 (3.05)
Z (p)	-0.13 (.90)	-1.74 (.082)

Table 5

Mean Scores and Standard Deviation (in Brackets) of the HADS Depression Subscale, Before and After the Intervention, With Results of the Wilcoxon T Test

Quasi-Experimental Design	Pre x (dt)	Post x (dt)
Intervention group (n = 14)	9.57 (3.90)	7.07 (3.85)
Z (p)		-2.840 (.005)

The intervention group showed a significant reduction in anxiety levels compared to the control group after the neurofeedback training. When analyzing all 14 participants together, the decrease in anxiety remained statistically significant.

Regarding depression, no significant differences were observed between the intervention and control groups, although a notable reduction was evident. When considering all participants together, the

reduction in depression reached statistical significance, albeit smaller than for anxiety.

Given these overall results, it was clinically relevant to examine individual changes in anxiety and depression (Tables 6 and 7).

Participants with normal anxiety did not show a reduction, whereas those with doubtful anxiety decreased by 40.89%, and participants with clinically relevant anxiety decreased by 29.05%.

Table 6*Mean Pre- and Posttest HADS Anxiety Scores and Percentage Change for Each Participant*

Subject	Anxiety		% Change
	Pretest	Posttest	
1	10	4	-60.00%
2	9	6	-33.33%
3	17	11	-35.29%
4	9	7	-22.22%
5	9	6	-33.33%
6	12	11	-8.33%
7	13	10	-23.07%
8	13	10	-23.07%
9	14	9	-35.71%
10	17	10	-41.18%
11	5	5	0%
12	17	12	-29.41%
13	11	7	-36.36%
14	9	4	-55.56%
	Pretest x	Posttest x	% Change
	11.78	8.00	-32.09%

Table 7*Mean Pre- and Posttest HADS Depression Scores and Percentage Change for Each Participant*

Subject	Depression		% Change
	Pretest	Posttest	
1	16	9	-43.75%
2	5	4	-20.00%
3	10	2	-80.00%
4	12	9	-25.00%
5	7	5	-28.57%
6	14	15	7.14%
7	15	13	-13.33%
8	9	10	11.11%
9	10	6	-40.00%
10	9	5	-44.44%
11	2	3	50.00%
12	11	9	-18.18%
13	7	5	-28.57%
14	7	4	-42.86%
	Pretest x	Posttest x	% Change
	9.57	7.07	-26.12%

Participants with normal depression decreased by 14%, those with doubtful depression decreased by 38.33%, and participants with clinically relevant depression decreased by 18.62%.

Alpha Amplitude Pretreatment and Posttreatment

Tables 8–10 display mean alpha amplitude before and after neurofeedback training.

Table 8

Mean Scores and Standard Deviation (in Brackets) of Alpha Amplitude at Baseline According by Group, Before and After the Intervention, With Results of the Mann-Whitney U Test

Experimental Design	Pre x (dt)	Post x (dt)
Intervention group (n = 7)	7.47 (1.09)	13.48 (6.04)
Control group (n = 7)	6.88 (1.80)	7.00 (1.76)
Z (p)	-0.32 (.75)	-3.00 (.003)

Table 9

Average Scores and Standard Deviation (in Brackets) of Alpha, Before and After the Intervention, With Results of the Wilcoxon T Test

Quasi-Experimental Design	Pre x (dt)	Post x (dt)
Intervention group (n = 14)	7.02 (1.38)	12.79 (4.62)
Z (p)		-3.296 (.001)

Table 10

Mean Pre- and Posttest Alpha Amplitude and Percentage Change for Each Participant

Subject	Alpha Amplitude		% Change
	Pretest	Posttest	
1	7.44	9.31	25.13%
2	4.89	9.29	89.97%
3	7.81	25.73	229.44%
4	7.24	12.57	73.61%
5	5.82	10.76	84.87%
6	4.32	8.22	90.27%
7	6.99	8.61	23.17%
8	7.17	10.60	47.83%
9	8.00	13.70	71.25%
10	8.14	15.28	87.71%
11	9.31	16.26	74.65%
12	5.90	12.55	112.71%
13	6.90	9.98	44.63%
14	8.37	16.18	93.30%
	Pretest x	Posttest x	% Change
	7.02	12.79	82.19%

Table 11
Spearman's Correlations Between Changes in Alpha Amplitude, Anxiety, and Depression

		Alpha-Anxiety Correlation r_s (p)	Alpha-Depression Correlation r_s (p)	Anxiety-Depression Correlation r_s (p)
Experimental Design	Intervention group ($n = 7$)	-0.073 (.877)	-0.148 (.751)	0.962 (.001)
Quasi-Experimental Design	Control group ($n = 7$)	-0.316 (.490)	-0.128 (.784)	-0.488 (.267)

Table 8 shows that while groups were similar at baseline, alpha amplitude significantly increased in the intervention group posttraining. These differences were confirmed in the quasi-experimental design, including the control group once they received training.

Overall, 64.28% of participants increased their alpha amplitude above 10 μ V (mean pretest = 7.02 μ V; mean posttest = 12.79 μ V), representing an average increase of 82.19%. No systematic relationship was observed between initial alpha amplitude and percentage increase.

In the intervention group, the expected inverse relationship between alpha amplitude and anxiety/depression was observed, although correlations were not statistically significant. However, a strong, significant correlation between anxiety and depression improvements was found in the intervention group and quasi-experimental design, indicating that reductions in anxiety were closely linked to reductions in depression.

Discussion

This exploratory study suggests that 10 sessions (5 hr) of alpha neurofeedback training can increase alpha amplitude and reduce anxiety and depression symptoms in adult female participants. Both the experimental and quasi-experimental designs indicated statistically significant improvements after training, despite the small sample size.

These results are consistent with previous studies demonstrating alpha neurofeedback's ability to enhance alpha amplitude (Dempster & Vernon, 2009; Hardt & Kamiya, 1978; Zoefel et al., 2011). Research generally indicates that more training sessions yield better results, though a ceiling effect may occur after a certain number of sessions (Cho et al., 2008; Nowlis & Wortz, 1973).

From a clinical perspective, our results suggest that short-term alpha neurofeedback may effectively reduce anxiety. Participants with doubtful or clinically relevant anxiety demonstrated meaningful decreases in symptoms, consistent with early findings by Hardt and Kamiya (1976, 1978) and other studies (Hammond, 2006a, 2006b; Moore, 2000). Similarly, reductions in depression were observed, echoing findings from Linden et al. (2012) and Choi et al. (2011), who reported improvements in mood and executive function after neurofeedback training.

Correlation analyses indicated that larger increases in alpha amplitude tended to coincide with greater reductions in anxiety and depression, though these associations were small and nonsignificant. Notably, anxiety and depression improvements were strongly correlated, highlighting the intertwined nature of these symptoms.

Limitations and Future Directions

Given the exploratory nature of this study, results should be interpreted cautiously:

- (a) sample size and composition: only 14 female participants were included, limiting generalizability, particularly to males,
- (b) exploratory design: findings are preliminary and hypothesis-generating rather than confirmatory,
- (c) training duration: Although 5 hr of neurofeedback produced measurable effects, optimal session number and duration remain uncertain, and
- (d) follow-up data: long-term maintenance of changes was not assessed.

Future research should replicate these findings with larger, more diverse samples, systematically examine optimal training parameters, and include follow-up assessments to determine the durability of neurofeedback effects.

In conclusion, this exploratory study provides preliminary evidence that short-term alpha neurofeedback may increase alpha amplitude and reduce anxiety and depressive symptoms, supporting its potential clinical utility while highlighting the need for further research.

Author Declaration

The authors confirm that the study was conducted without any commercial or financial relationships that could be construed as potential conflicts of interest. No grants or external funding were received for this work. The authors used AI tools solely for limited grammatical and language editing. No AI was used in the creation of the manuscript's content, analysis, or conclusions, and the authors take full responsibility for the work.

References

- Baehr, E., Rosenfeld, J. P., & Baehr, R. (2001). Clinical use of an alpha asymmetry neurofeedback protocol in the treatment of mood disorders. *Journal of Neurotherapy*, 4(4), 11–18. https://doi.org/10.1300/J184v04n04_03
- Cho, M. K., Jang, H. S., Jeong, S.-H., Jang, I.-S., Choi, B.-J., & Lee, M.-G.T. (2008). Alpha neurofeedback improves the maintaining ability of alpha activity. *NeuroReport*, 19(3), 315–317. <https://doi.org/10.1097/wnr.0b013e3282f4f022>
- Choi, S. W., Chi, S. E., Chung, S. Y., Kim, J. W., Ahn, C. Y., & Kim, H. T. (2011). Is alpha wave neurofeedback effective with randomized clinical trials in depression? A pilot study. *Neuropsychobiology*, 63(1), 43–51. <https://doi.org/10.1159/000322290>
- Demertzis, K. H., & Craske, M. G. (2006). Anxiety in primary care. *Current Psychiatry Reports*, 8(4), 291–297. <https://doi.org/10.1007/s11920-006-0065-4>
- Dempster, T., & Vernon, D. (2009). Identifying indices of learning for alpha neurofeedback training. *Applied Psychophysiology and Biofeedback*, 34(4), 309–328. <https://doi.org/10.1007/s10484-009-9112-3>
- Hammond, D. C. (2006a). Quantitative electroencephalography patterns associated with medical conditions. *Biofeedback*, 34(3), 87–94.
- Hammond, D. C. (2006b). What is neurofeedback? *Journal of Neurotherapy*, 10(4), 25–36. https://doi.org/10.1300/J184v10n04_04
- Hardt, J. V., & Kamiya, J. (1976). Conflicting results in EEG alpha feedback studies: Why amplitude integration should replace percent time. *Biofeedback Self-Regulation*, 1(1), 63–75. <https://doi.org/10.1007/bf00998691>
- Hardt, J. V., & Kamiya, J. (1978). Anxiety change through electroencephalographic alpha feedback seen only in high anxiety subjects. *Science*, 201(4350), 79–81. <https://doi.org/10.1126/science.663641>
- Kessler, R. C., Angermeyer, M., Anthony, J. C., De Graaf, R., Demeyttenaere, K., Gasquet, I., de Girolamo, G., Gluzman, S., Gureje, O., Haro, J. M., Kawakami, N., Karam, A., Levinson, D., Medina Mora, M. E., Oakley Browne, M. A., Posada-Villa, J., Stein, D. J., Tsang, C. H. A., Aguilar-Gaxiola, S., ... Üstün, T. B. (2007). Lifetime prevalence and age-of-onset distributions of mental disorders in the World Health Organization's World Mental Health Survey Initiative. *World Psychiatry*, 6(3), 168–176.
- Kroenke, K., Spitzer, R. L., Williams, J. B. W., Monahan, P. O., & Löwe, B. (2007). Anxiety disorders in primary care: Prevalence, impairment, comorbidity, and detection. *Annals of Internal Medicine*, 146(5), 317–325. <https://doi.org/10.7326/0003-4819-146-5-200703060-00004>
- Linden, D. E., Habes, I., Johnston, S. J., Linden, S., Tatini, R., Subramanian, L., Sorger, B., Healy, D., & Goebel, R. (2012). Real-time self-regulation of emotion networks in patients with depression. *PLoS ONE*, 7(6), Article e38115. <https://doi.org/10.1371/journal.pone.0038115>
- Lubar, J. F. (1997). Neocortical dynamics: Implications for understanding the role of neurofeedback and related techniques for the enhancement of attention. *Applied Psychophysiology and Biofeedback*, 22(2), 111–126. <https://doi.org/10.1023/a:1026276228832>
- Menon, V. (2011). Large-scale brain networks and psychopathology: A unifying triple network model. *Trends in Cognitive Sciences*, 15(10), 483–506. <https://doi.org/10.1016/j.tics.2011.08.003>
- Moore, N. C. (2000). A review of EEG biofeedback treatment of anxiety disorders. *Clinical Electroencephalography*, 31(1), 1–6. <https://doi.org/10.1177/155005940003100105>
- Niv, S. (2013). Clinical efficacy and potential mechanisms of neurofeedback. *Personality and Individual Differences*, 54(6), 676–686. <https://doi.org/10.1016/j.paid.2012.11.037>
- Nowlis, D. P., & Wortz, E. C. (1973). Control of the ratio of the midline parietal to midline frontal EEG alpha rhythms through auditory feedback. *Perceptual and Motor Skills*, 37(3), 815–824. <https://doi.org/10.1177/003151257303700329>
- Richards, D. (2011). Prevalence and clinical course of depression: A review. *Clinical Psychology Review*, 31(7), 1117–1125. <https://doi.org/10.1016/j.cpr.2011.07.004>
- Zigmond, A. S., & Snaith, R. P. (1983). The hospital and depression scale. *Acta Psychiatrica Scandinavica*, 67(6), 361–370. <https://doi.org/10.1111/j.1600-0447.1983.tb09716.x>
- Zoefel, B., Huster, R. J., & Herrman, C. S. (2011). Neurofeedback training of the upper alpha frequency band in EEG improves cognitive performance. *NeuroImage*, 54(2), 1427–1431. <https://doi.org/10.1016/j.neuroimage.2010.08.078>

Received: November 9, 2025

Accepted: November 25, 2025

Published: June 29, 2026