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Evaluation of Neurofeedback for Posttraumatic Stress Disorder Related to Refugee Experiences Using Self-Report and Cognitive ERP Measures

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Abstract

Background. Neurofeedback holds promise as an intervention for the psychophysiological dysfunction found in posttraumatic stress disorder (PTSD). Few empirical studies have assessed the efficacy of neurofeedback for PTSD, and none in individuals with refugee trauma. A proposed mechanism for neurofeedback efficacy in PTSD is through remediating deficits in cognitive control. We assessed pre- and postchanges in symptoms and neurocognitive functioning of refugee clients participating in a neurofeedback intervention for PTSD. *Methods*. Clinical data for 13 adult refugees with chronic PTSD who participated in neurofeedback combined with trauma counseling (NFT) was compared with 13 adult refugees placed on a waitlist to receive neurofeedback. Waitlist clients continued to receive trauma counseling alone (TC). NFT was additionally assessed pre- and posttherapy for changes in event-related potentials (ERPs) and behavioral indices of cognitive control using a visual continuous performance task (VCPT). Comparison VCPT data from healthy controls (HC) was available from the Human Brain Index database. *Results*. Posttherapy, NFT had significantly lower symptoms of trauma, anxiety, and depression compared with TC. NFT demonstrated an increased P3 amplitude and improved behavioral performance suggesting a normalization of cognitive control. *Conclusions*. These preliminary observations are consistent with a possible benefit of neurofeedback for remediating PTSD. This may be achieved at least partially by an improvement in cognitive control. Further confirmation of the effectiveness of the treatment now requires a randomized controlled trial that considers issues such as placebo response, nonspecific therapist effects, and duration of treatment.

Keywords

neurofeedback, posttraumatic stress disorder, refugee trauma, event-related potentials, cognitive control

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The psychological distress experienced by refugees involves cumulative and severe experiences of torture, traumatic events and loss, as well as postmigration stressors such as immigration detention, temporary protection status and the struggle of adapting to a resettlement country.[1-6] Refugees experience elevated rates of psychological disorders, particularly posttraumatic stress disorder (PTSD), which is estimated to occur up to 10 times more often.[6,7] Comorbidity of PTSD with depression, anxiety, somatic disorders, and prolonged grief[8,9] is common among refugees and affects functioning and quality of life.[10,11] Because of the high incidence of torture experiences, prevalence of traumatic brain injury in this population is also high (for review, see Steel et al[6]).

The complex nature of refugee trauma, means that adequate therapy is likely to require a specialized and multifaceted approach in addition to standard cognitive behavioral therapy and pharmacotherapy. Neurofeedback is a noninvasive intervention that aims to address symptoms via retraining brain activity[12] through operant conditioning.[13] A trainee's ongoing brainwave activity is measured using electroencephalography (EEG), and is shown to them in real time via a simple stimulus (visual or auditory) as reinforcement indicating when their brain is producing the desired activity. With training, the brain more easily and frequently produces the desired brainwave

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Table 1. Previous Trials Evaluating Neurofeedback (NF) Interventions for Posttraumatic Stress Disorder (PTSD).

Study	Population n = NFT/Control	Neurofeedback Protocol	Control Intervention	Outcomes[a]
Peniston and Kulkosky (1991)[26]	Army veterans; n = 15/14	30 sessions; occipital, alpha (8-12 Hz)/theta (4-8 Hz)	Trauma counseling	↓ PTSD symptoms, depression and nightmares; even at 30-month follow-up
Peniston et al (1993)[27]	Army veterans with comorbid alcohol abuse, n = 20/—	20 sessions; alpha/theta and "abreactive therapy"	—	↑theta ↓alpha synchrony; no relapse after 26 months in n = 16
van der Kolk et al (2016)[25]	Community treatment resistant, n = 28/24	24 sessions; right temporal (T4); alpha (13-15 Hz)	Waitlist (treatment as usual)	↓ PTSD symptoms
Gapen et al (2016)[28]	Community treatment resistant n = 17/—	40 sessions; T3-T4 or T4-P4; alpha (12-15 Hz)	—	↓ PTSD symptoms; improved affect regulation (questionnaire)

[a]Outcomes describe NF group postintervention relative to preintervention or control group where relevant; "↓" indicates decrease and "↑" indicates increase.

activity[14] (for evidence of this in PTSD see Nicholson et al[15] and Kluetsch et al[12]).

EEG measured event-related potentials (ERPs) are an objective and reliable measure of information processing that can be useful for assessing the changes relating to neurofeedback in addition to symptoms[16] (test-retest = 0.9 over 1 year[17]). A robust ERP marker of PTSD is a reduced "P3b" (oddball) reflecting controlled attention.[18,19] Abnormal cognitive control has also been reported in PTSD, demonstrated as a reduced "P3" amplitude during inhibition of emotionally distracting words[20] and in poorer behavioral performance.[21] Neurofeedback has been shown to improve symptoms and normalize ERP indices of cognitive control.[22] The same mechanism may underlie the effect of neurofeedback in PTSD.[23,24] There are no studies, to our knowledge, that have assessed changes in ERPs related to neurofeedback treatment for PTSD.

To date, the evidence for the efficacy of neurofeedback as an intervention for adults with PTSD is modest with only 4 published studies[25-28] (Table 1) (for reviews see Reiter et al[29] and Graap and Freides[30]). There are no studies that have examined neurofeedback in chronic trauma related to refugee experiences, nor any that have investigated the underlying brain mechanisms at work. This study will assess the efficacy of neurofeedback integrated with trauma counseling in treating refugees with chronic PTSD (NFT) compared with a waitlist control group receiving trauma counseling alone (TC). The Visual Continuous Performance Task (VCPT) was used to probe cognitive control during simultaneous measurement of ERPs and behavioral performance.[16] We hypothesized that NFT compared with TC would show a greater reduction of symptoms, and that NFT would exhibit normalization of the P3 ERP for cognitive control.

Methods

Participants

Data were collated retrospectively for 26 adults with chronic PTSD who had been seen for treatment at the Service for the Treatment and Rehabilitation of Torture and Trauma Survivors (STARTTS) located in Sydney, Australia (http://www.startts.org.au). PTSD was defined by a score of greater than 2.5 on the Harvard Trauma Questionnaire (HTQ)[31] and inclusion criteria allowed for comorbid depression or anxiety. Exclusion criteria included a current substance use disorder, severe neurological condition, developmental delay, or need for urgent medical care. All clients were initially seen for trauma counseling, but because of poor treatment response were referred for neurofeedback.[31] We report on the first 13 consecutive clients who received neurofeedback and trauma counseling (NFT) (4 female, mean age ± SD = 46.5 ± 11.5 years) and compared these clients with the next 13 clients waiting. All waiting clients continued to receive trauma counseling (TC) (5 female, 43.2 ± 8.3 years). Client allocation to NFT was based on a first come–first served basis. The average number of sessions of trauma counseling that clients received prior to their assessment at time point 1 was 26 ± 35 for NFT and 12 ± 17 for TC, $F(1, 25) = 1.32$, $P = .263$).

Clients gave informed consent for their data to be used for clinical research at the time of their referral to the neurofeedback program. The Sydney South West Human Research Ethics Committee reviewed and approved this study (HREC NO. LNR/15/LPOOL/369).

Procedure

Symptom and quantitative EEG assessments were completed before and after individualized therapy sessions, on a separate day. Only NFT had completed pre- and post-EEG assessments. NFT received 27 ± 12 sessions over 46 ± 32 weeks (Table 2). TC received 17 ± 8 sessions over 45 ± 24 weeks.

Neurofeedback Training Protocols. Neurofeedback protocols were selected for each client based on their presenting levels of hyperarousal and instability and were adjusted across sessions depending on a client's response. To address hyperarousal (ie, anxiety, fear, and insomnia), protocols began by rewarding sensory motor rhythm (SMR, 12-15 Hz) across the sensory motor

Table 2. Demographics of Neurofeedback (NF) and Treatment as Usual (TAU) Groups.

	NF (n = 13)	TAU (n = 13)	Significance (*P*)
Demographics			
Male[a]	9 (69)	8 (62)	—
Age (years)[b]	46.5 ± 11.5	43.0 ± 7.8	—
Years of education[b]	11.0 ± 3.6	9.3 ± 3.3	—
Permanent residency[a]	10 (77)	8 (62)	—
Years in Australia[b]	11.3 ± 10.8	2.1 ± 1.3	.005
Trauma history			
Early life[a]	4 (31)	3 (23)	—
Torture[a]	7 (54)	7 (54)	—
Witnessed[a]	13 (100)	12 (92)	—
General[a]	13 (100)	13 (100)	—
Treatment			
Sessions before intervention	26 ± 35	12 ± 17	—
Treatment sessions[b]	27 ± 12	17 ± 8	.028
Weeks between assessments[b]	46 ± 32	45 ± 24	—
Interpreter used[a]	8 (62)	13 (100)	.013
Psychotropic medication[a]	5 (38)	8 (62)	—

[a]Chi-square test: total number (%) given.
[b]*T* test: mean ± SD given.

Table 3. Baseline and Posttherapy Change Scores in Symptoms of Neurofeedback (NFT) and Treatment as Usual (TAU) Groups (Mean ± SD).

	Baseline			CHANGE (post − Pre)		
	NFT	TAU	Sig.[a] (*P*)	NFT	TAU	Sig.[a] (*P*)
Posttraumatic stress (PTS)[b] total	2.8 ± 0.4	3.2 ± 0.5	—	−0.9 ± 0.5	−0.1 ± 0.6	.002
Intrusion	2.8 ± 0.6	3.3 ± 0.7	—	−1.0 ± 0.6	−0.1 ± 0.6	.004
Numbing	3.0 ± 0.7	3.0 ± 0.8	—	−1.4 ± 0.9	−0.1 ± 1.1	.010
Anxious arousal	3.0 ± 0.7	3.1 ± 0.4	—	−0.9 ± 1.1	−0.3 ± 0.5	—
Dysphoric arousal	3.0 ± 0.5	3.3 ± 0.5	—	−0.7 ± 0.7	−0.2 ± 0.7	—
Avoidance	2.9 ± 0.7	3.1 ± 0.7	—	−1.0 ± 0.9	−0.2 ± 0.9	—
Anxiety[c]	2.8 ± 0.7	3.0 ± 0.5	—	−1.0 ± 0.8	−0.3 ± 0.7	.030
Depression[c]	2.8 ± 0.6	2.9 ± 0.4	—	−1.1 ± 0.6	−0.1 ± 0.6	.001

[a]Sig. indicates Significant at $P < .05$.
[b]Posttraumatic stress (PTS) measured using Harvard Trauma Scale.
[c]Anxiety and Depression measured using Hopkins Symptom Checklist–25).

region (Cz or C4)[32,33] and progressed to training of lower frequencies (8-10 Hz or 6-9 Hz) at parietal[26,27] or temporal[25] locations, to lower the level of arousal as clinical improvement occurred. The right hemisphere is generally targeted, that is, T4-P4 placement, supported by the finding that right parietal areas are implicated in the maintenance of PTSD.[34] However, if instability is present,[35(pp108-109)] an interhemispheric, or symmetrical, placement was chosen, that is, T3-T4. For clients who were still complaining of problems with attention we would train frontal alpha training. Quantitative EEG was primarily used to check of gross neurological problems and to fine tune the starting frequency for training. Clients were scheduled for 1 to 2, 1-hour sessions per week, involving 20 minutes of neurofeedback integrated with trauma counseling.[36]

Clinical Measures. PTSD symptoms were measured using the Harvard Trauma Questionnaire (HTQ).[31] Symptoms of anxiety and depression were assessed using the Hopkins Symptom Checklist–25 (HSCL-25).[37] These measures are available in multiple languages.

Symptoms—Analysis. Symptom difference scores were calculated for each individual by subtracting their baseline from posttherapy score. NFT and TC were compared on difference scores from trauma, depression and anxiety scales. A single analysis of covariance (ANCOVA) included group (2) as a between subjects variable and symptom type (3) as a repeated measure, controlling for number of sessions and using a significance threshold of $P < .05$. Follow-up analyses used ANCOVAs to compare groups on HTQ symptom subscales. NFT and TC were assessed for baseline differences on demographic data using *t* tests and chi-square tests for categorical variables (Table 3).

The Visual Continuous Performance Task. Cognitive control was measured using the Visual Continuous Performance Task (VCPT), designed by Kropotov et al[16] to probe several basic cognitive processes during simultaneous measurement of ERPs and behavioral performance. Clients were asked to watch for animal stimuli and press a button if a second animal stimulus follows (A-A), that is, the "GO" condition, or withhold responding if a plant stimulus follows (A-P), that is, the "NOGO" condition. The withholding of a response requires cognitive control. Each stimulus was presented for 100 ms, with an interstimulus interval of 1100 ms and intertrial interval of 3100 ms. One hundred A-P trials were presented in a pseudorandom order among equal numbers of A-A trials, and 2 other trial types beginning with plants, that is, ignore conditions.[16]

EEG Acquisition and Processing. Clients completed the EEG recording seated in front of a computer monitor in a sound attenuated room. Data were acquired from 19 symmetrical scalp electrode sites (Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, and O2) placed according to the international 10-20 system and referenced to linked ears, using a 21-channel "Mitsar-201" system (CE 0537, PC-controlled, manufactured by Mitsar Co, Ltd, Russia, www.mitsar-medical.com) amplified (bandpass 0.5-30 Hz) and sampled at 250 Hz. Impedance was kept below 10 kohm. The VCPT was presented using Psytask 2.x computer code.

EEG data were further processed offline using WinEEG software (designed for Mitsar systems, version 2.103.70).[38] Recordings were re-referenced to a common average montage. Electro-oculogram artifact was identified through an independent component analysis and corrected by zeroing the activation curves of individual components.[39,40] Intervals with residual artefacts were identified and excluded based on an absolute threshold of ± 100 μV. Trials were excluded for artifact and incorrect responses.

VCPT ERP—Quantification and Analysis. Waveforms were epoched by condition from 300 ms before stimulus 1 to 900 ms after stimulus 2. The primary component of interest was the P3 NOGO reflecting cognitive control and was quantified by peak amplitude within a latency window of 250 to 450 ms post–stimulus 2 in A-P trials of the grand average waveform at Cz, identified by visual inspection.[16] The mean number of NOGO trials available per participant was 78.8 ± 20.2 for pre- and 80.9 ± 19.5 for postneurofeedback assessments. Changes in P3 NOGO pre- to post- were analyzed at the group level using a paired-samples t test at a significance threshold of $P < .05$.

Age- and gender-matched VCPT ERP data were available from the Human Brain Institute's (HBI) normative database[16,41] to evaluate the direction of any significant change in the client group (Figure 1). The HBI has an average 43 ± 12 healthy controls (HC) per age window of ≤5years.

VCPT Behavioral Data—Analysis. Behavioral performance was quantified in terms of commission errors (failure to suppress a response, A-P or "NOGO" trials), omission errors (failure to sustain attention, A-A or "GO" trials), reaction time for correct responses (A-A trials) and reaction time variability. Responses to GO trials were considered incorrect if given outside 200 to 2000 ms post onset of the target stimulus. Group differences in behavioral indices were examined in one repeated-measures ANCOVA controlling for number of therapy sessions at significance threshold $P = .05$.

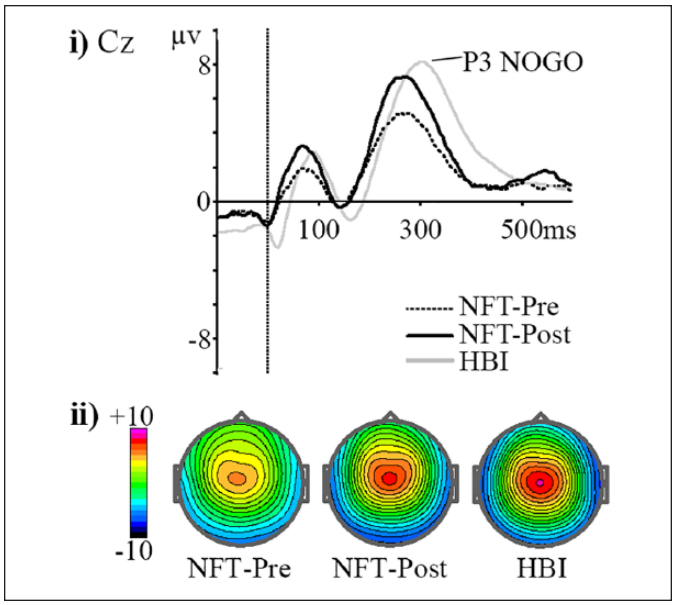

Figure 1. Normalization of the P3 component of the NOGO event-related potential (ERP) in the treatment group. (i) ERPs for NOGO trials of the Visual Continuous Performance Task for 13 clients pre– (dotted line) and post– (black line) neurofeedback therapy (NFT) and in healthy controls (gray line) from the Human Brain Institute's (HBI) normative database. (ii) Maps for the P3 NOGO peaks for NFT pre- and posttherapy, and HBI. This figure is available in color online.

Results

Demographics

NFT did not differ from TC on age, number of females, years of education, permanent residency status, trauma and torture experiences, or treatment with psychotrophic medication (Table 2). NFT had been in Australia for longer than TC (Table 2). NFT originated from Afghanistan (n = 3), Cambodia (n = 2), Sri Lanka (n = 2), Chile, Croatia, Iran, Iraq, Kuwait, and Vietnam while TC comprised refugees from Iran (n = 6), Iraq (n = 6), Afghanistan, and Sri Lanka.

Therapy Sessions

NFT did not differ to TC on length of therapy course; however, they did receive more treatment sessions, $t(24) = 2.34$, $P = .028$ (Table 2). Standard practice for neurofeedback recommends at least 2 sessions per week to maximize learning. Number of sessions was therefore included as a covariate in

Table 4. Pre– and Post–Neurofeedback Therapy Behavioral scores (Mean ± SD).

	Pre	Post	$t(12)$	Significance (P)
Omission errors	11.7 ± 10.5	4.6 ± 4.7	2.4	.037
Commission errors	4.3 ± 7.7	1.2 ± 3.6	2.1	.065
Reaction time	397.3 ± 87.2	365.3 ± 85.4	1.4	—
Reaction time variability	13.5 ± 3.7	8.6 ± 2.8	3.8	.003

symptom analyses. A greater proportion of TC had required an interpreter in therapy, $\chi^2(1) = 6.19$, $P = .013$.

Symptoms

There were no significant symptom differences between NFT and TC at baseline (Table 3). A main effect of group indicated significantly larger improvement in NFT than TC, holding number of sessions constant, $F(1, 23) = 14.1$, $P = .001$. There was no main effect of symptom kind or interaction with group. Compared with TC, NFT had a greater reduction of symptoms of posttraumatic stress, $F(1, 25) = 13.0$, $P = .002$; anxiety, $F(1, 25) = 5.4$, $P = .030$; and depression, $F(1, 24) = 16.5$, $P = .001$, posttherapy, holding number of sessions constant (Table 3). ANCOVAs on HTQ-subscales identified an NFT advantage specifically for intrusion, $F(1, 24) = 8.0$, $P = .002$, and numbing, $F(1, 24) = 5.7$, $P = .010$. Twelve of the NFT compared with 1 TC reduced their PTSD symptom levels to below the clinically significant score of 2.5 posttherapy. Eight NFT and 5 TC were on psychotropic medication at the time of their first assessment. Posttherapy, no NFT and 5 TC were on psychotropic medication.

VCPT—P3 NOGO ERP

The mean P3 NOGO peak amplitude increased from pre– to post–neurofeedback therapy, $t(12) = 2.5$, $P = .026$ (Figure 1). This was in a direction reflecting normalization when visually compared with the P3 NOGO generated from the HBI's normative database.[16,41]

VCPT—Behavioral Performance

NFT had reduced reaction time variability, $t(12) = 3.8$, $P = .003$, post–neurofeedback therapy and showed a trend improvement of fewer omission errors, $t(12) = 2.4$, $P = .037$ (Table 4). The reductions in commission errors and reaction time from pre– to post–neurofeedback therapy were not statistically significant.

Correlations With Symptoms

There were no significant correlations between ERP, behavioral, or symptom changes from pre– to post–neurofeedback therapy (.05 divided by 15 tests = .003). We note that there was a strong correlation between decreased omission errors and increased P3 NOGO ($r = -0.83$, $P < .001$).

Discussion

This study found that neurofeedback as an adjunct to trauma counseling in adult refugees with PTSD is associated with a reduction of PTSD symptoms to below diagnostic threshold in 12 of 13 participants. This was seen in only one TC participant. This is a significant outcome in a group of people with chronic PTSD. The symptom improvements were paralleled by a normalization of the P3 NOGO ERP and associated behavioral performance, which suggests improved cognitive control. Following from our case reports,[36,42] this pilot study increases our confidence that neurofeedback training may be a promising adjunct to trauma counseling for chronic PTSD, and further that the mechanism of effect may involve remediation of cognitive control processes.

Our results contribute to a literature that has found a positive effect of neurofeedback training in some[26,28] though not all studies[43] (for a critical review see Graap and Freides[30]). Symptom improvement is reported in the majority of studies, despite the chronicity of the condition, speaking to a powerful therapeutic effect. However, there are important differences between these studies including type and chronicity of posttraumatic symptoms and the type and consistency of the neurofeedback intervention. This makes it more difficult to be confident about the effect size of the improvement or the indications for the different neurofeedback techniques.

We hypothesized that cognitive control would be improved with neurofeedback and consistent with this, we observed a normalization of the P3 NOGO. This is noteworthy, given the modest sample size. In normal controls, the VCPT ERP has excellent test-retest stability with little change over time or practice effects.[17] However, until we can compare the changes with a suitable control group, we also cannot rule out the possibility that other elements of the therapy, other than neurofeedback, relate to the normalization of the P3 NOGO ERP. We did not examine other ERP components given the small size and nonrandomized design of our study.

In this study, the improved P3 NOGO was observed across clients receiving different training protocols, which suggests that there may be different pathways to improved cognitive control. A lack of cognitive control limits the ability of traumatized individuals to disengage from traumatic memories or to modulate their emotional responses and arousal, causing them to use avoidance and numbing to withdraw from situations in which executive control is required.[24] The shift in P3 NOGO in clients who also remitted from trauma symptoms is consistent with the hypothesis that an improvement in cognitive control underlies recovery from PTSD.[23,24] There is further evidence from studies using functional magnetic resonance imaging for a P3-NOGO abnormality in PTSD, specifically reporting reduced activation of frontal executive regions.[23,44] However, the P3 in GO/NOGO tasks is seldom reported and results have been mixed as to the direction or presence of a difference associated with PTSD.[45-47]

We did not observe a strong relationship between symptom improvements and psychophysiological measures. This lack of a relationship may relate to the small sample size and possible confounding factors such as the change in medication from baseline to follow-up testing, or to the indirect and possibly nonlinear relationship between cognitive control and psychopathology. Finally, PTSD may relate to more than one underlying mechanism. The change that was observed could not be compared to change in TC as they did not take part in this section of the research. Ongoing research will investigate other factors known to relate to symptom reduction in PTSD, such as the attenuation of arousal, to help understand the underlying mechanisms important to remediation of the disorder so as to better target treatment such as neurofeedback.

Our research extends previous studies in PTSD[26,28] by focusing on a refugee sample. These individuals are characterized by chronic and complex presentations and current exposure to post-migration stress. Chronic insomnia, body pain, grief, and chronic depression are also common features of their presentation. In the experience of STARTTS, effective treatment for refugee trauma requires a multimodal approach combining trauma counseling, psychiatric treatment, body-oriented therapies, and family assistance.[48] Neurofeedback may be an important tool for remediating a facet of this trauma. If replicated, the current findings would be generalizable to other cases of chronic PTSD.

Limitations and Future Directions

This pilot study was based on data gathered during routine clinical work. Although this facilitates the generalization of the results to the real world, it limits conclusions due to sample size and the non-random way that treatment was allocated. Randomization is likely to address group differences in current stress, duration of time spent in Australia, prior exposure to treatment and need for an interpreter in therapy. Groups had received different amounts of trauma counseling which, although controlled for in the analysis, may have affected the results. The lack of appropriate controls for the ERP assessment limits our ability to interpret changes in ERPs over the course of treatment, as they may be the effect of learning or result from factors in therapy other than neurofeedback. Important clinical factors such as a past history of head trauma, starvation, medication exposure and variation in counselling practice are not accounted for because of the nature of the data collection and the acceptance of service users with a broad range of pre-existing conditions.

Conclusions

This study found that neurofeedback therapy was a potent intervention for refugees suffering from PTSD who had not responded to trauma counselling alone. After therapy, NFT had significantly lower symptoms of posttraumatic stress, anxiety and depression compared TC. In line with our hypothesis, NFT also normalized the P3 amplitude and corresponding reduction in omission errors, reflecting improved cognitive control. These preliminary observations are consistent with the possible benefit of neurofeedback for remediating PTSD, which may be achieved at least partially by an enhancement in cognitive control. Studies of the efficacy of neurofeedback for PTSD are needed that are randomized, controlled, adequately powered and take into account the diversity of neurofeedback protocols.[29]

Authors' Note

Mirjana Askovic, Anna J. Watters and Anthony W. F. Harris are also affiliated with Psychiatry, Sydney Medical School at Westmead Hospital, Sydney, Australia.

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Author Contributions

Mirjana Askovic: Conceptualization, Methodology, Data collection and curation, Writing – original draft
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Mariano Coello: Conceptualization, Funding Acquisition, Writing – review & editing
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Juri Kropotov: Conceptualization, Methodology, Supervision, Formal analysis, Writing – review & editing

Declaration of Conflicting Interests

The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: Dr Anthony W. F. Harris has received consultancy fees from Janssen Australia and Lundbeck Australia. He has been on an advisory board for Sumitomo Dainippon Pharma. He has received payments for educational sessions run for Janssen Australia, Servier and Lundbeck Australia. He is the chair of One Door Mental Health.

Ethical Approval

The Sydney South West Human Research Ethics Committee reviewed and approved this study (HREC NO. LNR/15/LPOOL/369).

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Informed Consent

Clients gave informed consent for their data to be used for clinical research at the time of their referral to the neurofeedback program.

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