



Frontal alpha asymmetry neurofeedback for the reduction of negative affect and anxiety



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ARTICLE INFO

Article history:

Received 8 September 2016
Received in revised form
9 February 2017
Accepted 15 February 2017
Available online 20 February 2017

Keywords:

Neurofeedback
Frontal alpha asymmetry
Right prefrontal cortex
Negative affect
Anxiety
EEG

ABSTRACT

Frontal alpha asymmetry has been proposed to underlie the balance between approach and withdrawal motivation associated to each individual's affective style. Neurofeedback of EEG frontal alpha asymmetry represents a promising tool to reduce negative affect, although its specific effects on left/right frontal activity and approach/withdrawal motivation are still unclear. The present study employed a neurofeedback training to increase frontal alpha asymmetry (right - left), in order to evaluate discrete changes in alpha power at left and right sites, as well as in positive and negative affect, anxiety and depression. Thirty-two right-handed females were randomly assigned to receive either the neurofeedback on frontal alpha asymmetry, or an active control training (N = 16 in each group). The asymmetry group showed an increase in alpha asymmetry driven by higher alpha at the right site ($p < 0.001$), as well as a coherent reduction in both negative affect and anxiety symptoms ($ps < 0.05$), from pre-to post-training. No training-specific modulation emerged for positive affect and depressive symptoms. These findings provide a strong rationale for the use of frontal alpha asymmetry neurofeedback for the reduction of negative affect and anxiety in clinical settings.

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1. Introduction

Several research lines converge in indicating the existence of two fundamental motivational systems in the mammalian brain (Davidson, 1992; Dickinson & Dearing, 1979; Gray & McNaughton, 1982; Konorski, 1967; Lang & Bradley, 2013; Lang, 2010; Lang, Bradley, & Cuthbert, 1997): 1) an *appetitive/approach* system, that drives approach behaviors toward rewarding stimuli, and 2) a *defensive/withdrawal* system, associated with avoidance from aversive stimuli. The balance between appetitive and defensive dispositions has been referred to as the *affective style* of the individual (Davidson, 1998a, 1998b, 2004; Davidson, Jackson, & Kalin, 2000), which prompts responses to emotional stimuli, dispositional mood and vulnerability to psychopathology. On one hand, the motivational systems are embedded in evolutionary ancient brain structures which subserve similar functions in humans and in other mammals. On the other hand, humans further developed control over primary motivational impulses, mostly subtended by the prefrontal cortex (Damasio & Carvalho, 2013; Damasio et al.,

2000; Ochsner & Gross, 2005).

In this sense, the dorsolateral prefrontal cortex acts as a moderator of the primary motivational and emotional responses, through its anatomical and functional connections with core limbic structures such as the amygdala, the basal ganglia, the anterior cingulate and the orbitofrontal cortex (Spielberg et al., 2012). Furthermore, a hemispherical specialization for the prefrontal cortex in motivation and affect has been reported: prevalent activity in the right compared to the left prefrontal areas has been related to withdrawal behaviors and to the experience of negative emotions, while the opposite pattern (i.e., greater left vs. right activity) accompanies approach behaviors and positive affect (Davidson, 1988, 1998b; Harmon-Jones, Gable, & Peterson, 2010; Papousek, Reiser, Weber, Freudenthaler, & Schultze, 2012; Papousek et al., 2014). Accordingly, it has been advocated that 1) the left prefrontal cortex has a role in organizing limited resources toward goal-oriented behaviors, sustaining approach and positive affect; 2) the right prefrontal cortex mediates vigilance for threat and sensitivity to punishment, thus promoting avoidance and withdrawal (Gray & McNaughton, 1982; Sutton & Davidson, 1997). Therefore, the differential activity between the right and left prefrontal lobes is commonly considered a measure of affective style (Coan & Allen, 2004; Davidson, 1992).

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Since electroencephalographic (EEG) alpha power is an inverse index of cortical activity (Cook, O'Hara, Uijtdehaage, Mandelkern, & Leuchter, 1998; Davidson, Chapman, Chapman, & Henriques, 1990), the asymmetry in frontal alpha power is thought to reflect the balance between the activity of the right and left prefrontal lobes (Allen, Coan, & Nazarian, 2004). Accordingly, reduced alpha at right compared to left frontal sites (i.e., greater right-sided activity) has been associated with withdrawal motivation (Sutton & Davidson, 1997), negative affect (Jacobs & Snyder, 1996; Schaffer, Davidson, & Saron, 1983; Tomarken, Davidson, Wheeler, & Doss, 1992), as well as reports of more intense negative emotions after unpleasant film viewing (Papousek et al., 2014; Wheeler, Davidson, & Tomarken, 1993). On the other hand, reduced alpha at left compared to right frontal sites (i.e., greater left-sided activity) has been related to approach motivation (Harmon-Jones & Allen, 1998, 1997; Sutton & Davidson, 1997), trait positive affect (Tomarken et al., 1992), rates of pleasantness after positive film viewing (Wheeler et al., 1993), reward responsiveness (De Pascalis, Varriale, & D'Antuono, 2010), dispositional optimism (De Pascalis, Cozzuto, Caprara, & Alessandri, 2013), greater emotional flexibility (Papousek et al., 2012) and better emotional regulation (Jackson et al., 2003).

Electroencephalographic (EEG) biofeedback (*neurofeedback*) has been proposed as a tool to modulate the hemispherical asymmetry in prefrontal activity, in order to regulate affect (Rosenfeld, Cha, Blair, & Gotlib, 1995). More in general, biofeedback is a bio-behavioral technique which aims at modifying physiological activity and, in turn, improving health and/or performance. In line with the biofeedback principles, neurofeedback relies on the assumption that, by providing real-time information on brain's activity, individuals can extend their conscious control and learn how to regulate their own brain activity (Thibault, Lifshitz, & Raz, 2016). Operant conditioning is a possible mechanism of action, since the positive feedback can be conceived as a reinforcement for the desired bio-behavioral pattern (Miller & DiCara, 1967). As a preliminary step, neurofeedback studies tested whether or not it was possible to modulate frontal alpha asymmetry, as well as the effects of this modulation on reported affect. Through five sessions of frontal alpha asymmetry neurofeedback using rewarding and non-rewarding tones, Allen, Harmon-Jones, and Cavender (2001) trained two groups of healthy participants either to increase or decrease their alpha asymmetry score, computed by subtracting left (F3) from right (F4) alpha power; therefore higher scores corresponded to reduced right compared to left frontal activity. Only participants trained to decrease alpha asymmetry succeeded and neither training modified current affect, as measured by the Positive and Negative Affect Scale, state version (PANAS; Watson, Clark, & Tellegen, 1988). Recently, one comprehensive study on a sample of 60 individuals replicated Allen and colleagues' results, providing a visual feedback and adding a random-feedback control group (Quaedflieg et al., 2015); accordingly, this study showed that after 6 sessions participants were able to increase left compared to right alpha at frontal sites, but not vice versa. Further studies supported the efficacy of neurofeedback for the modulation of frontal alpha asymmetry in both directions (i.e., increasing or decreasing the asymmetry score) after three sessions of acoustic neurofeedback, with a similar design as the one from Allen and colleagues (Harmon-Jones, Harmon-Jones, Fearn, Sigelman, & Johnson, 2008), or even after one session of visual feedback (Peeters, Ronner, Bodar, van Os, & Lousberg, 2014). Again, neither positive nor negative affect were influenced by the training.

Although there is evidence for neurofeedback to be effective in modulating frontal alpha asymmetry, previous studies did not address whether observed modifications were mostly driven by changes in alpha power at right, left, or both frontal sites. From a

clinical perspective, this is of particular relevance, given that similarly altered patterns of frontal alpha asymmetry have been found in different conditions characterized by affect dysregulation, such as anxiety and depression (Beaton et al., 2008; Mennella, Messerotti Benvenuti, Buodo, & Palomba, 2015; Moscovitch et al., 2011; Stewart, Coan, Towers, & Allen, 2011, 2014). In particular, even though both anxiety and depressive symptoms have been associated with reduced frontal alpha asymmetry (right - left), in anxiety this is subtended by a dominance in withdrawal motivation and negative affect (increased right compared to left frontal activity); on the other hand, in depression the asymmetry dysregulation has been related to a reduction in approach motivation and positive affect (reduced left compared to right frontal activity) (Davidson, 1998a; Shankman & Klein, 2003). For this reason, it seems crucial establishing the specific effect of frontal alpha asymmetry neurofeedback on left and right activity, in order to provide a strong rationale for its clinical application.

The present study evaluated the effectiveness of frontal alpha asymmetry neurofeedback in increasing the alpha asymmetry index (F4 - F3). Furthermore, it was tested whether variations in asymmetry are subtended by relative changes in left (F3) and/or right (F4) frontal alpha power. Finally, the effects of the training on affect were assessed using a comprehensive battery, including the PANAS, for the evaluation of both positive and negative affect, as well as measures of anxiety and depressive symptoms.

It was hypothesized that 1) the frontal alpha asymmetry training would be effective in increasing alpha asymmetry (F4 - F3); 2) in case higher alpha asymmetry was subtended by an increase in alpha power at the right site (less dominant right-sided activity) this would be associated with a decrease in negative affect and anxiety scores; 3) if higher alpha asymmetry was driven by decreased alpha power at the left site (more prominent left-sided activity), higher positive affect scores and a reduction in depressive symptoms were expected.

2. Methods

2.1. Participants

Thirty-two healthy and free from medication undergraduate students (M age = 23.1, SD = 1.2) from the University of Padova were enrolled. The present study included only right-handed females, since asymmetrical alpha activity is influenced by handedness (Davidson, 1988), and previous studies have primarily examined female participants (Allen et al., 2001; Harmon-Jones et al., 2008; Peeters, Ronner, et al., 2014). Exclusion criteria were: previous head injury, chronic mental or neurological diseases and treatment with medications known to influence EEG, such as tranquilizers or antidepressants. Participants were randomly assigned to receive a biofeedback training designed either to increase frontal alpha asymmetry (i.e., F4 - F3; *asymmetry group*; N = 16), or to increase mid-frontal (Fz) alpha activity (*active control*; N = 16). Neurofeedback aimed at increasing alpha activity has been previously employed in order to reduce stress and anxious symptoms (Brown, 1970; Hammond, 2005; Hardt & Kamiya, 1978), due to the positive association of alpha power with states of relaxation and low arousal. Usually, this training is carried out at posterior sites, since the alpha rhythm is predominant in the occipital and parietal regions. On the contrary, in the present study the training targeted the mid-frontal site, to serve as a specific control condition for the frontal alpha asymmetry neurofeedback.

The groups were comparable with respect to sociodemographic variables (Table 1). Participants were told that at the end of the seven sessions they would receive a monetary payment proportional to their performance during training sessions (total payment

Table 1
Demographics and pre-training log-transformed alpha power at frontal sites for the Asymmetry and the Active Control groups.

Variables	Asymmetry group (N = 16)	Active control group (N = 16)
Demographics		
Age (years)	22.9 (1.2)	23.3 (1.2)
Education (years)	17.2 (1.1)	17.2 (1.0)
Alpha power ($\mu\text{V}^2/\text{Hz}$) pre-training		
F3	0.69 (0.3)	0.63 (0.3)
Fz	0.45 (0.3)	0.40 (0.3)
F4	0.62 (0.3)	0.60 (0.3)

Notes: Data are *M* (*SD*). F3 = left frontal site; Fz = mid-frontal site; F4 = right frontal site.

ranged from 10 to 25 euros). The present study was carried out with the adequate understanding and written consent of the participants in accordance with the Declaration of Helsinki. The study was approved by the Ethics Committee of the Department of General Psychology, University of Padova (Italy).

2.2. Procedure

The whole procedure was run by two psychologists expert in biofeedback, clinical assessment and EEG procedures, with the help of two trained undergraduates for data collection. The procedure consisted of 7 biweekly sessions (completed within two weeks). Each session lasted about 45 min, starting within the same 3-hr time window. During the first session, upon arrival at the laboratory, participants received general information about the experiment and read and signed an informed consent form. A brief semi-structured interview for the collection of sociodemographic status information and exclusion criteria was conducted. Then, individuals sat on a semi-reclined comfortable armchair in a sound dampened dimly lit recording room, where a psychophysiological assessment was carried out, including psychological questionnaires administration and electrophysiological recordings in resting conditions. After the initial session (*pre-training assessment*), participants were randomly assigned to the *asymmetry* or to the *active control* groups, and underwent 5 neurofeedback sessions. In the final session the initial assessment was repeated (*post-training assessment*), in order to evaluate the effects of the training.

2.3. Psychological assessment

Participants completed the following questionnaires

- 1) The Positive and Negative Affect Schedule (PANAS; Watson et al., 1988) consists of two subscales of 10 items each (i.e., negative affect (NA) and positive affect (PA)). In the version of the test used for the present study, for each item, participants are asked to rate (from 1 to 5) the extent to which they have experienced a particular emotion during the present day. The two subscales reflect dispositional dimensions, with high-NA scores characterizing subjective feelings of distress and unpleasurable engagement, and high-PA scores reflecting the extent to which an individual experiences pleasurable engagement with the environment. Reliability and validity of the PANAS as a measure of positive and negative affect are high in non-clinical samples (Crawford & Henry, 2004).
- 2) The Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) is composed of 21 items, which the participant has to evaluate on a four-point (0–3) Likert scale. The total score ranges from 0 to 63, with higher scores corresponding to higher levels of anxiety. Reliability and validity of the BAI as a measure of anxiety symptoms are high in both community samples (Beck

et al., 1988) and college populations (Creamer, Foran, & Bell, 1995).

- 3) The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996; Italian version by Ghisi, Flebus, Montano, & Sanavio, 2006). The BDI-II is a valid and reliable self-report questionnaire that evaluates the severity of depressive symptoms in the past two weeks and is composed of 21 items, which the participant has to evaluate on a four-point (0–3) Likert scale. Higher BDI-II scores correspond to more severe depressive symptoms. Importantly, reliability and validity of the BDI-II as a measure of depressive symptoms are high in both community samples (Lasa, Ayuso-Mateos, Vázquez-Barquero, Díez-Manrique, & Dowrick, 2000) and college populations (Sprinkle et al., 2002).

2.4. Electrophysiological recordings

EEG and vertical electro-oculogram (VEOG) were recorded in a standardized fashion using a computerized recording system (ProComp Infniti, Thought Technology; Montreal, Canada). Electrophysiological signals were recorded for 5 min, while participants were required to fixate a white cross on a gray background, in order to reduce eye-movements. EEG was collected from three active scalp positions using golden electrodes with a bipolar montage. The EEG sites were F3, Fz and F4, referenced online to Cz, consistently with previous neurofeedback studies (Allen et al., 2001). Using a bipolar montage, VEOG was recorded in order to detect eye-movements and eye-blinks. Electrodes were placed at the supra- and suborbit of the right eye.

All electrodes impedances were kept below 5 k Ω . Each physiological signal was amplified, band-pass filtered (1–100 Hz) and digitized at 2048 Hz with ProComp Infniti Encoder.

2.5. Neurofeedback training

Each training session began with a 5-min baseline recording at rest with eyes open, in order to establish the baseline frontal alpha asymmetry score. Mean baseline values were used to calculate the threshold for the training session, defined as mean activity +0.85 standard deviations (Allen et al., 2001). Five 5-min biofeedback training trials followed, with a 1-min inter-trial break. Each trial was segmented in 150 epochs of 2 s. Alpha power (8–13 Hz) at right (F4) and left (F3) frontal leads was extracted online using a fast Fourier transform algorithm with a Hamming window function applied to the 2-sec epoch. The difference in alpha power between right and left (F4 - F3) was then computed and compared against the threshold value established during the baseline. Participants were provided with a visual feedback consisting of a histogram reflecting the current frontal alpha asymmetry score. If the alpha asymmetry score was below the threshold, the histogram was red; when the asymmetry score exceeded the threshold (i.e., desired

state), the histogram instantly turned green. The feedback was inhibited if ocular activity exceeded $\pm 50 \mu\text{V}$.

In the control condition, participants had the same visual feedback, but it reflected the changes in alpha power recorded at Fz electrode, instead of the frontal alpha asymmetry score. Before starting the training, participants were told that biofeedback training involved modifying the activity of their brain, and that they should try to maintain the histogram green as long as they could. Every time they maintained the desired EEG state for 10 s (consecutive or not), a point was scored. It was explained to the participants that each point would correspond to 0.1 euros to add to the minimum payment of 10 euros for the participation. Participants were never told that the biofeedback was contingent on frontal alpha asymmetry and/or mid-frontal alpha.

2.6. EEG data processing

The signal was down-sampled offline at 256 Hz, and continuous EEG data correction for eyeblinks was performed through a regression-based correction algorithm (LMS regression; Gómez-Herrero, 2007). Epochs of 500 ms each were then obtained from the continuous signal and EEG chunks containing artifacts greater than $\pm 70 \mu\text{V}$ were automatically rejected (using EEGLAB plugin Darbeliai). Each EEG segment was further inspected for residual artifacts. For each accepted epoch, a Hamming windowing was applied and chunks were then overlapped by 50% to minimize loss of data. A Fast Fourier Transform (FFT) method was used to derive estimates of spectral power (μV^2) for each electrode site. Power density values ($\mu\text{V}^2/\text{Hz}$) were calculated averaging spectral power within the alpha band (8–13 Hz) for each participant at each site.

2.7. Statistics

Alpha power at right (F4), left (F3) and midline (Fz) sites was \log_{10} transformed. After transformation, data were normally distributed, as assessed with the Kolmogorov–Smirnov-test. Alpha asymmetry was calculated subtracting the logarithm of alpha power at F3 site from F4 (i.e., $F4 - F3$).

Analyses of variance (ANOVAs) with Group (Asymmetry, Active control) as a between-subjects factor were used to compare the two groups in terms of age, education, and alpha power at each site.

Changes in resting frontal alpha asymmetry were evaluated running an ANOVA with Group as a between-subjects factor and Time (pre-training assessment, post-training assessment) as a within-subjects factor.

Changes in absolute alpha power at left and right sites were evaluated running an ANOVA with Group as a between-subjects factor, Time and Lateralization (F4, F3) as within-subjects factors. An ANOVA with Group as a between-subjects factor and Time as a within-subjects factor was run to test changes in alpha power in Fz. Separate ANOVAs with Group as a between-subjects factor and Time as a within-subjects factor were conducted on each psychological measure, namely the PANAS NA and PA scores, BAI and BDI-II.

Whenever the sphericity assumption was violated, the Greenhouse–Geisser correction was applied. Corrected p-values, ϵ estimates and uncorrected degrees of freedom are reported. Tukey HSD test was used for post-hoc analyses. Main effects and interactions were considered to be significant at $p < 0.05$. Partial eta-squared (η_p^2) was reported as a measure of the effect size. The η_p^2 values considered to represent small, medium, and large effects are 0.01, 0.06, and 0.14, respectively (Cohen, 1988).

In order to evaluate the relationship between training-induced changes in frontal alpha asymmetry, alpha power at left (F3) and right (F4) sites, and subjective measures, Pearson's correlations

were performed between changes in these measures (post-training minus pre-training scores).

STATISTICA 6.1 software (StatSoft Inc, Tulsa, OK) was used for statistical analysis.

3. Results

3.1. Characteristics of the participants in the asymmetry and active control groups

ANOVAs yielded no group differences for age ($F_{(1, 30)} = 0.75$, $p = 0.40$, $\eta_p^2 = 0.02$), education ($F_{(1, 30)} = 0.11$, $p = 0.74$, $\eta_p^2 = 0.004$), pre-training alpha at left ($F_{(1,30)} = 0.27$, $p = 0.61$, $\eta_p^2 = 0.01$), midline ($F_{(1, 30)} = 0.21$, $p = 0.65$, $\eta_p^2 = 0.01$) and right ($F_{(1,30)} = 0.04$, $p = 0.83$, $\eta_p^2 = 0.001$) electrodes (i.e., F3, Fz and F4). The descriptive statistics for each group are reported in Table 1.

3.2. Effects of neurofeedback on EEG measures

From the ANOVA on frontal alpha asymmetry a significant Group \times Time interaction emerged ($F_{(1, 30)} = 4.94$, $p < 0.05$, $\eta_p^2 = 0.14$), as depicted in Fig. 1. In order to further characterize this interaction, changes in alpha power at left and right sites were analyzed, specifically, a Group by Time ANOVA on right (F4) and left (F3) alpha power was run. A significant effect for Lateralization ($F_{(1, 30)} = 8.26$, $p < 0.01$, $\epsilon = 0.99$, $\eta_p^2 = 0.22$), in the context of a Group \times Time \times Lateralization interaction emerged ($F_{(1, 30)} = 4.94$, $p < 0.05$, $\epsilon = 0.99$, $\eta_p^2 = 0.14$; see Fig. 2). Tukey post-hoc showed a significant increase in alpha power at right (F4) site from pre-to post-training in the asymmetry group ($p < 0.001$), whereas no significant difference in alpha power at right (F4) and left (F3) sites from pre-to post-training was found in the active control group ($p = 0.90$). No other main effects or interactions emerged (all p 's > 0.10). ANOVA did not show any main effect for Group and Time or Group \times Time interaction on midline alpha (Fz) activity (all p 's > 0.17).

In order to visualize the changes in frontal alpha asymmetry and alpha power at left and right sites, alpha power during baseline preceding each training session, after the first one, were extracted and plotted in Fig. 3. Although there was variability across sessions,

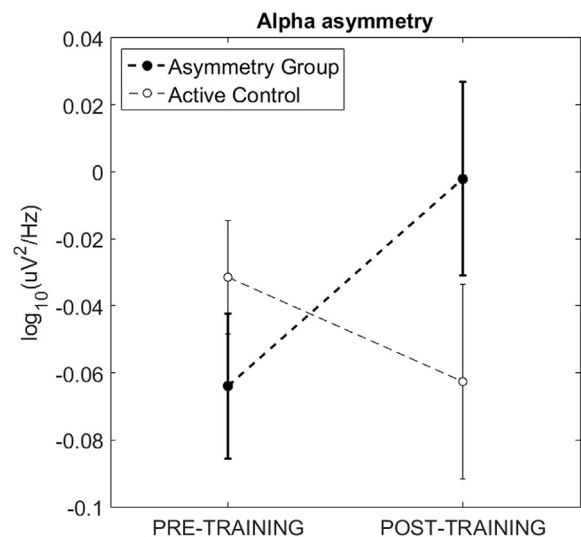


Fig. 1. Neurofeedback modulation of alpha asymmetry: the Asymmetry Group, but not the Active Control, showed a significant increase in alpha asymmetry from pre-to post-training. Error bars represent the standard error of the mean.

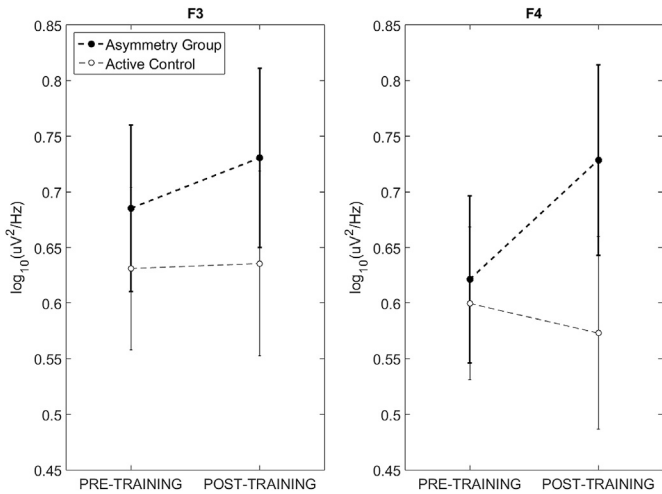


Fig. 2. Neurofeedback modulation of left and right alpha power: the Asymmetry Group, but not the Active Control, showed a significant increase in resting alpha power at F4, but not F3, from pre-to post-training. Error bars represent the standard error of the mean.

the linear trend supported the increase in frontal alpha asymmetry from pre-to post-training in the asymmetry group, and a steeper gradient of increase in alpha power at right (F4) compared to left site (F3).

As complementary analyses, separate ANOVAs with Group as a between-subjects factor, Time and Lateralization as within-subjects factors were run for power changes in delta (0.5–4 Hz), theta (4–8 Hz) and beta (13–30 Hz) bands. It emerged that the Group \times Time \times Laterality interaction was neither statistically significant for delta ($F_{(1, 30)} = 1.99, p = 0.17, \epsilon = 0.99, \eta_p^2 = 0.06$) nor for theta bands ($F_{(1, 30)} = 1.85, p = 0.18, \epsilon = 0.99, \eta_p^2 = 0.06$). On the contrary, the Group \times Time interaction was significant for the beta band ($F_{(1, 30)} = 6.75, p < 0.05, \epsilon = 0.99, \eta_p^2 = 0.18$), as determined by an increase in power from pre-to post-training at right, but not left, frontal site ($p < 0.01$).

3.3. Effects of neurofeedback on affect, anxiety and depressive symptoms

ANOVA on PANAS Negative Affect scores showed a significant main effect for Time ($F_{(1, 30)} = 6.78, p < 0.05, \eta_p^2 = 0.18$) and a Group \times Time ($F_{(1, 30)} = 4.13, p = 0.05, \eta_p^2 = 0.12$) interaction. Tukey post-hoc comparisons revealed a significant decrease in PANAS

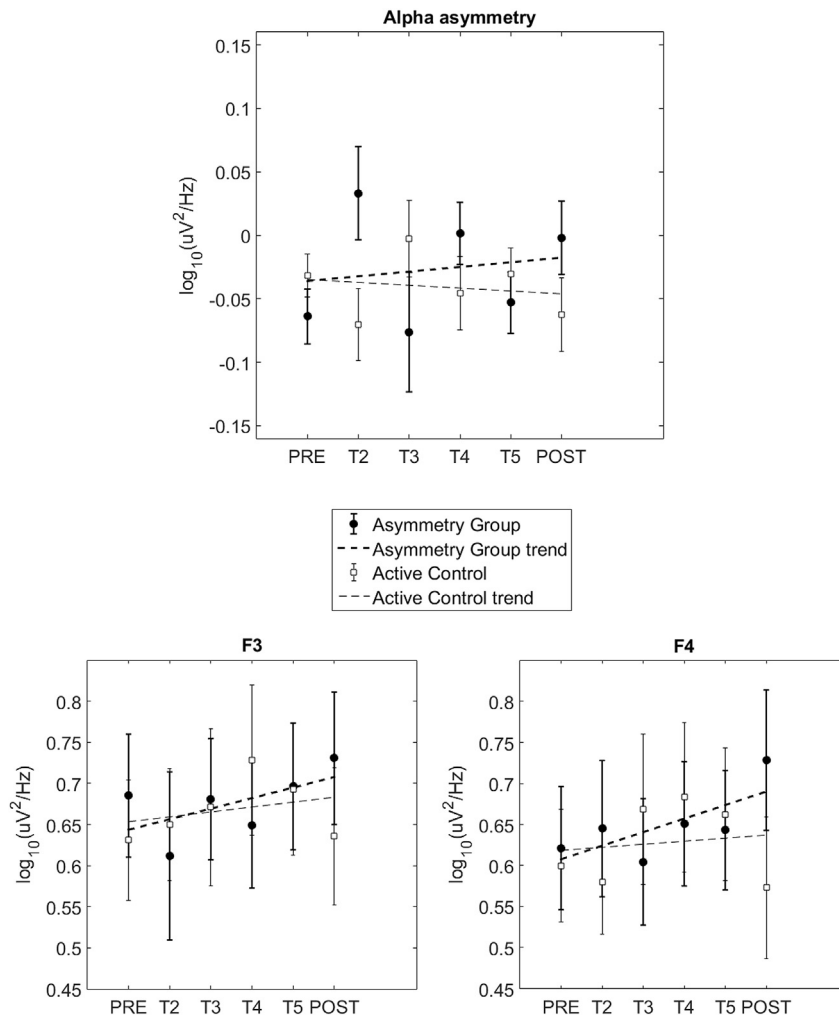


Fig. 3. Session-by-session changes in frontal alpha asymmetry and in absolute alpha power at F3 and F4: the Asymmetry Group showed a linear positive trend in alpha asymmetry during the training (top), sustained by a steeper increase in alpha power at F4 compared to controls (bottom). TN = baseline preceding neurofeedback session number N. Error bars represent the standard error of the mean.

Negative Affect scores from pre- to post-training in the asymmetry group ($p < 0.05$), whereas no significant difference in the NA-PANAS scores from pre- to post-training was found in the active control group ($p = 0.98$). On the contrary, the group by time ANOVA on PANAS Positive Affect scores did not reveal any significant effect for Group ($F_{(1, 30)} = 0.43$, $p = 0.52$, $\eta_p^2 = 0.01$), Time ($F_{(1, 30)} = 1.10$, $p = 0.30$, $\eta_p^2 = 0.04$), and Group \times Time ($F_{(1, 30)} = 0.12$, $p = 0.73$, $\eta_p^2 = 0.004$).

The Group \times Time ANOVA on BAI scores yielded a significant effect for Time ($F_{(1, 30)} = 12.27$, $p < 0.01$, $\eta_p^2 = 0.29$), further characterized by a Group \times Time interaction ($F_{(1, 30)} = 5.51$, $p < 0.05$, $\eta_p^2 = 0.16$). Tukey post-hoc comparisons showed a significant decrease in BAI scores from pre- to post-training in the asymmetry group ($p < 0.01$), whereas no significant difference in the BAI scores from pre- to post-training emerged in the active control group ($p = 0.85$).

The Group \times Time ANOVA on BDI-II scores yielded a significant effect for Time ($F_{(1, 30)} = 5.07$, $p < 0.05$, $\eta_p^2 = 0.14$). No main effect for Group or Group \times Time interaction emerged (all p 's > 0.19). All means (SD) and statistical details are reported in Table 2.

3.4. Correlational analyses

No significant correlations emerged, after multiple comparisons correction, between changes in frontal alpha asymmetry (as well as changes in left and right absolute alpha power) and changes in subjective measures from pre- to post-training (see Table 3).

4. Discussion

The present findings support the efficacy of the frontal alpha asymmetry neurofeedback in increasing the frontal alpha asymmetry index ($F4 - F3$), in line with previous research (Harmon-Jones et al., 2008; Peeters, Ronner, et al., 2014). More importantly, to our knowledge, this is the first study to report that this effect is subtended by a specific reduction in the right frontal activity from pre- to post-training, as revealed by a significant increase in alpha power at the right, but not left, scalp site. As far as the self-report measures are concerned, a significant decrease in negative affect emerged from pre- to post-training for the asymmetry group, but not for active controls, while positive affect showed no modulation. Furthermore, levels of anxiety were specifically reduced after the asymmetry neurofeedback, while no difference between groups emerged for changes in depressive symptoms.

Table 2
ANOVA on positive and negative affect, anxiety and depression scores from pre- to post-training in asymmetry group and Active control.

Variables	Pre-training	Post-training	p	η_p^2
PANAS Positive Affect Score				
Asymmetry Group	27.56 (10.35)	29.19 (6.53)	0.73 ^a	0.004 ^a
Active Control	29.94 (8.87)	30.75 (10.20)		
PANAS Negative Affect Score				
Asymmetry Group	19.25 (9.03)	14.69 (6.46)	0.05 ^a	0.12 ^a
Active Control	18.44 (6.64)	17.88 (8.58)	<0.01 ^b	
BAI				
Asymmetry Group	11.38 (9.56)	6.00 (5.56)	0.69 ^b	0.16 ^a
Active Control	10.19 (9.39)	9.13 (8.00)	<0.05 ^a	
BDI-II				
Asymmetry Group	9.75 (12.38)	6.00 (7.90)	<0.001 ^b	0.06 ^a
Active Control	8.13 (7.30)	7.19 (9.59)	0.42 ^b	

Notes: Data are M (SD). ^a = p -values and partial eta-squared referred to the Group \times Time interaction for the corresponding measure. ^b = p -values associated to post-hoc comparisons in the context of a statistically significant Group \times Time interaction (not reported for non-significant interactions). ANOVA = analysis of variance; PANAS = Positive and Negative Affect Schedule; BAI = Beck Anxiety Inventory; BDI-II = Beck Depression Inventory II.

These results align with the initial hypothesis that a decrease in right compared to left prefrontal activity would be associated with a reduction of negative affect and anxiety, which have been related to a dominance of the right over the left prefrontal cortex (Jacobs & Snyder, 1996; Schaffer et al., 1983; Tomarken et al., 1992). This is in line with the conceptualization that anxiety and negative affect manifest due to an increase in withdrawal motivation and heightened vigilance for threat, which has been shown to be subtended by prevalent right-sided frontal activity (Davidson, 1998a; Mathersul, Williams, Hopkinson, & Kemp, 2008). In the same conceptualization, depressive symptoms are thought to be associated with a deficit in approach/appetitive motivation, as revealed by a left-sided reduction in prefrontal activity, compared to the right. Accordingly, in the present study, both positive affect and depressive symptoms were unaffected by the frontal asymmetry training, and this is mirrored by the absence of changes in left frontal activity.

Nonetheless, linear correlation between changes in frontal alpha asymmetry and changes in affective measures did not emerge. Although participants who underwent frontal alpha asymmetry training showed both a decrease in right frontal activity, and a parallel reduction in subjective measures of negative affect and anxiety, these changes were not linearly related. Several studies supported the idea that these measures are linearly related during the execution of emotional tasks (Coan, Allen, & Mcknight, 2006; Mennella et al., 2015; Stewart et al., 2011), but that this relationship may not emerge in resting conditions (Elgavish, Halpern, Dikman, & Allen, 2003; Hagemann, Naumann, Becker, Maier, & Bartussek, 1998; Heller & Nitscke, 1997; Hewig, Hagemann, Seifert, Naumann, & Bartussek, 2004; Hofmann, 2007; Reid, Duke, & Allen, 1998). In particular, the capability model of individual differences in frontal alpha asymmetry (Coan et al., 2006) suggests that approach and withdrawal motivational tendencies, as revealed by frontal alpha asymmetry, are more powerfully detected during an emotional challenge compared to resting conditions. On one hand, this may be due to the fact that emotional tasks actively stimulate motivational tendencies, which subtend each individual's affective style. On the other hand, measuring frontal EEG asymmetry in response to emotional stimuli or situations has been shown to reduce the contribution of uncontrolled variance due, for instance, to the choice of the reference electrode and other confounds. Accordingly, frontal alpha asymmetry during emotional tasks has been reported to predict affect and mood better than frontal alpha asymmetry at rest (Coan et al., 2006; Stewart, Coan, Towers, & Allen, 2014). Therefore, future studies ought to investigate whether neurofeedback-related changes in EEG asymmetry, although not linearly related to unprovoked affect, predict state affect and emotional reactivity in emotional contexts. For instance, it would be worth investigating whether the observed increase in right frontal alpha would correlate with reduced negative affect in threatening situations, as well as lower self-reported unpleasantness for threatening stimuli, as suggested by the capability model.

Overall, the present results suggest that the training could have an impact in reducing anxiety symptoms in clinical populations. Anxiety symptoms have been found to be associated with increased right compared to left frontal activity, as measured by frontal alpha asymmetry (Mathersul et al., 2008); also, this cortical pattern has been related to heightened negative affect and withdrawal tendencies (Shankman & Klein, 2003). Surprisingly, so far not many studies assessed the effects of frontal alpha asymmetry neurofeedback on anxiety symptoms. Results from the present study lay the foundations for a wider testing of the frontal alpha asymmetry protocol in clinical anxiety, providing a rationale for its application. Since we observed changes in frontal asymmetry at rest and in trait

Table 3
Pearson's (r) correlation coefficients between changes in alpha power at F3, F4 and asymmetry scores and changes in Positive and Negative Affect, Anxiety, and Depression Scores from pre- to post-training in Asymmetry Group and Active Control.

		PA		NA		BAI		BDI-II	
		r	p	r	p	r	p	r	p
Asymmetry Group	F3	−0.01	0.97	−0.04	0.87	0.31	0.25	0.14	0.60
	F4	0.03	0.91	−0.25	0.36	0.16	0.56	0.13	0.62
	F4-F3	0.05	0.87	−0.25	0.34	−0.05	0.84	0.04	0.87
Active Control	F3	0.28	0.29	0.13	0.62	0.15	0.57	−0.23	0.39
	F4	0.09	0.73	0.23	0.39	0.37	0.16	−0.18	0.51
	F4-F3	−0.32	0.23	0.27	0.31	0.54	0.03	0.04	0.89

Notes: PA = Positive Affect scale of the PANAS; NA = Negative Affect scale of the PANAS; BAI = Beck Anxiety Inventory; BDI-II = Beck Depression Inventory II. $p = p$ -values (uncorrected for multiple comparisons).

anxiety, this training may be helpful especially in conditions characterized by a trait-like increase in anxiety, such as the generalized anxiety disorder. As far as state-dependent anxiety is concerned, future studies ought to test whether reducing withdrawal motivation might be beneficial for a more adaptive response to threatening stimuli or situations. Therefore, the impact of frontal alpha asymmetry neurofeedback could also be assessed in psychopathologies associated with state-dependent increases in anxiety, such as phobias or panic disorder.

On the other hand, we hypothesize that, in order to have an impact on clinical depression, neurofeedback should be aimed at increasing left compared to right prefrontal activity, thus stimulating approach motivation and positive affect. Accordingly, depressed individuals are typically characterized by a left-sided prefrontal hypo-activity, compared to right (Davidson, 1998a, 2004). In line with this conceptualization, case report studies yielded positive results in the treatment of clinical depression by inhibiting slow alpha and theta activity while reinforcing higher frequencies at left frontal sites (i.e., FP1 and F3) (Hammond, 2000, 2005). At the same time, the possibility that clinically depressed patients would benefit from the alpha asymmetry training cannot be ruled out. It is conceivable that the pre-existent left-sided hypo-activity in depressed patients would interact with the effect of the training. For instance, in depressed individuals, the same training could decrease alpha at left vs. right sites, in contrast with the pattern observed in our sample, thus being beneficial. In this regard, a small number of case studies supported the effectiveness of the frontal alpha asymmetry training in reducing depressive symptoms, although no specific information on hemispheric modulation (left or right frontal) was provided (Baehr, Rosenfeld, & Baehr, 1997, 2001; Baehr & Baehr, 1997; Choi et al., 2011; Earnest, 1999; Peeters, Oehlen, Ronner, Van Os, & Lousberg, 2014). Therefore, the training ought to be run on both anxious and depressed patients, in order to discriminate its specific effects. Importantly, there is a strong need for a control condition such as the one employed in the present work, since the vast majority of studies in the field are still uncontrolled (Baehr & Baehr, 1997; Baehr, Rosenfeld, & Baehr, 2001, 1997; Choi et al., 2011; Earnest, 1999; Hammond, 2000, 2005; Hammond & Baehr, 2009; Peeters, Oehlen, et al., 2014). Of note, in the present study, no modulation of negative affect and anxiety was observed in the control condition. It is worth highlighting that neurofeedback to increase alpha power has been traditionally used for the reduction of stress and anxious symptoms (Brown, 1970; Hammond, 2005; Hardt & Kamiya, 1978), targeting the occipital and parietal sites. Since no modulation of mid-frontal alpha emerged in the present study, neurofeedback for the increase of alpha power at mid-frontal sites seems less effective compared to posterior sites, therefore representing an ideal control condition for the frontal alpha asymmetry training.

Complementary analyses on frontal asymmetry in other EEG bands showed that the effect in alpha band extended to beta, but not delta and theta rhythms. In other words, the training was associated with an increase in both right frontal alpha and beta power. It has been previously reported that the effects of the frontal alpha asymmetry neurofeedback extended to beta band (Peeters, Ronner, et al., 2014). Intriguingly, a number of studies supported the notion that both alpha and beta bands are inversely correlated with the activation of the underlying neural structures, as measured by functional resonance imaging, both at rest (Moosmann et al., 2003) and during cognitive/motor tasks (Scheeringa et al., 2011; Yuan et al., 2010). Therefore, it can be speculated that changes in alpha and beta power reflect a similar training-induced modification. Nonetheless, some authors reported that correlation between BOLD fluctuations and alpha/beta bands are independent from each other, possibly mirroring different underlying processes (Scheeringa et al., 2011). This issue needs to be specifically addressed by future studies, looking at how training-related modifications in different EEG bands are associated to underlying BOLD fluctuations.

The present findings ought to be interpreted in light of some limitations. Firstly, self-report measures were only collected at pre- and post-training, thus not allowing for a session-by-session correlation between EEG and subjective measures of affect. This may mitigate the relationship between changes in asymmetry and changes in self-reported affect, thus potentially explaining null correlational findings. Nonetheless, our main aim was to evaluate the effectiveness of a short training on alpha asymmetry and negative affect at a group level, as a first step toward future clinical application. Evidence for training effectiveness adds to the literature in this field, suggesting that future studies ought to examine day-by-day changes in clinical populations, and to attest the stability of the effect through follow-up assessments. Secondly, regarding the method, the Cz reference has been shown not to be ideal for obtaining a measure of frontal alpha uncontaminated by volume-conducted alpha from other regions (Hagemann, 2004; Hagemann, Naumann, & Thayer, 2001; Stewart, Bismark, Towers, Coan, & Allen, 2010), although this reference scheme has been employed in previous neurofeedback studies on frontal alpha asymmetry (Allen et al., 2001; Harmon-Jones et al., 2008). The current-source-density (CSD) transform of EEG data has been reported to attenuate the risk that alpha generators in different brain regions impact on the measurements of alpha asymmetry at frontal sites, providing more consistent results concerning the association between alpha asymmetry and affective measures (Stewart et al., 2010, 2014). Therefore, future studies ought to better clarify whether the effects of the training survive when other reference schemes are used.

In summary, the present study argues in favor of the employment of frontal alpha asymmetry neurofeedback to increase right

vs. left frontal alpha activity, in order to reduce negative affect and anxiety levels, laying the foundations for testing on subclinical and clinical populations. On the other hand, we suggest that future research should investigate the effects of neurofeedback training aimed at increasing left vs. right prefrontal activity on approach motivation, positive affect and depressive symptoms.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Acknowledgements

The authors thank Sara Anastasia Contino and Agustín Jaime for their important help in data collection.

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