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Validation of a neurofeedback paradigm: Manipulating frontal EEG alpha-activity and its impact on mood

Frenk Peeters [a,*], Jacco Ronner [b], Lonneke Bodar [a], Jim van Os [a,c], Richel Lousberg [a]

[a] Department of Psychiatry and Psychology, South Limburg Mental Health Research and Teaching Network, EURON, Maastricht University, Maastricht, The Netherlands
[b] Department of Instrumentation, Faculty of Psychology and Neuroscience, Maastricht University, The Netherlands
[c] King's College London, King's Health Partners, Department of Psychosis Studies, Institute of Psychiatry, London, UK

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ABSTRACT

It is claimed that neurofeedback (NF) is an effective treatment for a variety of psychiatric disorders. NF, within an operant conditioning framework, helps individuals to regulate cortical electroencephalographic (EEG) activity while receiving feedback from a visual or acoustic signal. For example, changing asymmetry between left and right frontal brain alpha activity by NF, is claimed to be an efficacious treatment for major depressive disorder. However, the specificity of this intervention in occasioning electrophysiological changes at target locations and target wave-frequencies, and its relation to changes in mood, has not been established. During a single session of NF, it was tested if the balance between left and right frontal alpha-activity could be changed, regardless of direction, in 40 healthy females. Furthermore, we investigated whether this intervention was electrophysiologically specific and if it was associated with changes in mood. Participants were able to decrease or increase frontal alpha-asymmetry during the intervention. However, no changes in mood were observed. Changes in EEG activity were specific in terms of location and wave-frequency.

1. Introduction

There is growing interest in neurofeedback (NF) as a treatment for a variety of mental disorders including ADHD, anxiety, and depression (Hammond, 2005; Lofthouse et al., 2011). It is postulated that this technique, within an operant conditioning framework, helps individuals to regulate cortical electroencephalographic (EEG) activity while receiving feedback from a visual or acoustic signal. The resulting change in EEG activity is presumed to be associated with a change in underlying cortical activation, and subsequently to result in a reduction of associated symptoms (Evans and Abarbanel, 1999). For each disorder, different electrophysiological abnormalities have been described, leading to more or less disorder-specific treatment protocols that each aim at specific electrophysiological changes. A NF treatment typically consists of 20 to 30 treatment sessions, lasting 30 min each, with an average frequency of 2 sessions a week.

However, some questions regarding the basic validity of the NF paradigm remain unanswered (Allen et al., 2004). One of the methodological questions is to what degree feedback within the operant conditioning framework really results specifically in the desired electrophysiological changes.

This is illustrated by the manipulation of resting (alpha, 8–13 Hz) activity in prefrontal regions by NF as a therapeutic intervention in major depressive disorder (MDD). To avoid confusion, it should be underlined that *increased* alpha activity in cortical structures is indicative of *decreased* cortical activation in those areas. Preliminary clinical work indicates that the increase of right relatively to left alpha activity at F3–F4 with the use of neurofeedback (alpha-asymmetry protocol) may be associated with a reduction in depressive symptomatology (Baehr et al., 1997, 2001; Choi et al., 2011; Hammond, 2005; Rosenfeld et al., 1996). However, the validity of the intervention is unknown because the researchers (i) did not examine whether it specifically changed the target frequency band (8–13 Hz) at the target cortical locations (F3–F4), (ii) did not employ sham-NF and blinded rating, and (iii) allowed participants to receive other treatments in addition to NF. Therefore, it cannot be ruled out that non-specific effects of the interventions resulted in the reported electrophysiological and clinical changes. As a proof of principle, experimental work is required showing double dissociation. In other words, the best proof that frontal cortical brain activity can be manipulated with NF, is to examine whether it is possible to train participants not only in the desired direction, but also in the opposite direction, and show differential associations with electrophysiological and mood parameters.

To our knowledge, only two studies examined whether manipulation of cortical activity with the use of NF targeting opposite cortical sites resulted in corresponding (opposite) electrophysiological changes. First, Hardman et al. (1997) showed that healthy subjects were able to regulate frontal (F3–F4) activity of slow cortical potentials towards

* Corresponding author at: Department of Psychiatry, University Hospital Maastricht, P.O. Box 5800, 6202 AZ Maastricht, The Netherlands. Tel.: +31 43 3874130; fax: +31 43 3875444.
E-mail address: f.peeters@maastrichtuniversity.nl (F. Peeters).

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right and left hemispheres with the use of NF. A second study reported that healthy subjects were able to gradually increase left relative to right frontal alpha activity (resulting in increased AA) during NF sessions over 5 consecutive days (Allen et al., 2001). The subjects were not able to increase right relative to left frontal alpha activity after the first day (which should result in a decrease of AA). Unprovoked mood ratings did not alter during the training. However, the subjects in whom left relative to right frontal alpha activity was stimulated, reported less interest, amusement, and happiness in response to a happy film in comparison to the subjects that were trained in the other direction. As both studies did not report on the specificity of the intended electrophysiological changes, it remains unclear if additional changes occurred in other frequencies and/or other cortical locations. Additionally, study samples were small (n's were 16 and 18 respectively) potentially hampering reliability.

The present study was designed for a larger sample to address the following questions. It should be noted again that an *increase* in cortical alpha-activity represents a *decrease* in corresponding cortical brain-activation. This means that *increasing* right relatively to left alpha activity at F3–F4, results in a *decrease* of brain activation in right relatively to left prefrontal cortical areas.

First, we set out to examine whether it is possible to change the balance between left and right frontal alpha-activity, in both directions, during a single session of NF. Second, we examined the specificity of the training by also measuring changes of electrophysiological activity at other frequencies (delta, theta, slow-beta and fast beta) and other cortical locations. Third, we investigated whether changes in resting frontal asymmetry were accompanied by changes in momentary emotions. It was hypothesized that an increase in left frontal activity relative to right frontal activity would be associated with an increase in positive affect and a decrease in negative affect, whereas an increase in right frontal activity relative to left frontal activity would be associated with emotional changes in the opposite direction. The experiment was carried out in healthy women given reports of sex-specific associations between AA and mood (Stewart et al., 2010).

2. Methods

2.1. Subjects

Forty right-handed female participants, aged between 18 and 34 years (M = 22.6. SD = 4.5) were recruited from available control research pools. Exclusion criteria were a current DSM-IV axis-I disorder and current use of psychoactive medication. Participants provided written informed consent and received € 20 as recompense for their time.

2.2. EEG recording and quantification

All EEG recordings took place in an electrically-shielded room. While subjects were seated in a comfortable chair, Ag/AgCl electrodes were placed on F3, F4, C3, C4, P3 and P4 using the international 10–20 system (Jasper, 1958). To control for possible vertical eye movements, an electro-oculogram (EOG) electrode was placed 1 cm under the midline of the left eye. EEG electrodes were referenced with averaged earlobes (A1 and A2). A ground electrode was placed at the forehead. In order to reduce skin resistance, Nuprep scrub gel was used. All electrodes were fixed using 10–20 conductive paste. Impedances were kept below 5 kΩ.

Data collection was channeled through an acquisition PC with a BrainAmp DC EEG amplifier (Brain Products) using a 1000 Hz sample frequency. Online calculations were done by a filter written for BrainVision RecView. The data was epoched online into 2.048-s epochs that overlapped by 75% and then transformed by a fast Fourier transform (FFT) to the frequency domain (frequency resolution 0.488 Hz). Vertical left and right EOG measures were used to reject invalid epochs. A criterion of +/− 50 μV was used. Every 0.512 s, the power within the alpha frequency band (7.8 Hz–13.1 Hz) of both F3 and F4 was calculated. F3–F4 alpha asymmetry was computed as the difference of the natural log-transformed F3 and F4-alpha power: Ln(F3-alpha) − Ln(F4-Alpha). Current asymmetry is subsequently compared to the personal mean baseline asymmetry. The result of the calculation was sent to a stimulus PC running Presentation stimulus delivery software (Neurobehavioral Systems) with an 8-bit parallel port (LPT-port) to control a paradigm showing a visual representation of the asymmetry. In the Presentation paradigm, the last 20 values of the asymmetry are used in a moving average to prevent 'jitter' in the feedback. Subjects received feedback with visual feedback; they were instructed to increase the level of a thermometer that was shown on a flatscreen. Additionally, a numerical score below the thermometer indicated their actual total performance. This score was adjusted (i.e. increased) continuously by a number ranging from 0 to 128, depending on the level of the thermometer. In this way a good actual performance (a shift in asymmetry in the desired direction) resulted in an increasing total score. A big shift in the desired direction resulted in a rapidly increasing total score, whereas a small shift in the desired direction resulted in a slow increasing total score. A shift in the undesired direction produced no change in total score. The purpose of this total performance score was to give subjects feedback on the differential effect of the session.

2.3. Measurement of momentary emotions

Participants rated their momentary emotions just before and immediately after the experiment by filling out the Dutch translation of the Positive and Negative Affect Schedule (Peeters et al., 1996; Watson et al., 1988).

2.4. Experimental design

Subjects were randomly assigned to one of two experimental groups. In the first group (DOWN), subjects attempted to reduce F3–F4 alpha asymmetry. This reduction should result in a decrease of alpha-activity at F3 in comparison to F4 that is accompanied by an increase of cortical activity at F3 relative to F4. In the second group (UP), conditioning took place in order to increase F3–F4 alpha asymmetry. This increase is supposed to lead to an increase of alpha-activity at F3 in comparison to F4 that is accompanied by a decrease of cortical activity at F3 relative to F4. Subjects were blind to group membership. Research staff was not blind to group allocation.

The total experiment consisted of seven 5-minute EEG recordings of which the middle five represented feedback (FB) blocks (hereafter: Pre, FB1, FB2, FB3, FB4, FB5, Post). The first 5-minute block was a baseline block after which the mean F3–F4 asymmetry was computed. This value was set as the reference point for all further feedback blocks. After the baseline block, 5 consecutive feedback blocks took place. Between the blocks, participants were granted a two-minute break. Immediately after the last feedback block, a second rest (i.e. without feedback) EEG was recorded.

2.5. Statistical analysis

The EEG asymmetry data of each session showed a normal distribution without outliers. An ANCOVA for repeated measures was carried out. The within-subject factor was time, the between-subjects factor was group and the covariate was the baseline EEG measure. *Post-hoc* t-tests were used to test for significant differences in asymmetry between the groups at the different points in time.

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3. Results

3.1. Effect of the feedback training on AA between F3 and F4

Data from 3 participants (2 in the DOWN, 1 in the UP group) were excluded as a result of technical problems with the EEG measurements. Baseline AA between the groups was not significantly different ($t = 1.18$, $p = .25$).

Fig. 1 provides a visual representation of the mean AAs at the 7 consecutive time points for both groups, showing a different course in AA over the duration of the training. Compared to the baseline, the UP group shows an increase until the fifth feedback block. An opposite effect appears to take place in the DOWN group. A marked rebound effect was apparent in both groups from feedback block 5 to the post-training time point.

A basic ANCOVA model for repeated measures was used with AA as dependent variable, time (the 7 block points) as the within-factor, group (UP and DOWN) as the between-factor, and number of artifact-free EOG segments from the baseline as a covariate. The a priori multivariate time × group interaction was significant ($F_{(Hot)} = 3.552$; $df = 6.28$; $p = 0.01$, $F_{(Huynh–Feldt)} = 2.24$; $df = 5.125$; $p = 0.05$), showing that the training succeeded in changing AA differentially in the desired directions in the two groups. Two hypothesis-generating *post-hoc* analyses regarding the data shown the in figure were performed. First, group means of FB1 in both groups show a graphically symmetrical unexpected effect in the opposite direction. A frequency analysis made clear that this was not caused by FB1 outlier values. However, these changes appeared to be non-significant ($F_{Huynh–Felt} = 1.41$, $p = 0.24$). Second, a rebound effect from FB5 to Post appeared to be significant ($F_{(Hot)} = 6.286$; $df = 1.34$; $p = 0.017$, $F_{(Huynh-Feldt)} = 6.286$; $df = 1$; $p = 0.017$).

3.2. Effect of the feedback training on AA at other cranial locations

An ANCOVA model was used to test potential AA effects on C3–C4 and P3–P4 locations. The time * group effect in both models did not reach statistical significance (C3–C4: $p = .0.30$; P3–P4: $p = .21$), indicating that the differential feedback effect on AA did not extend to central or parietal regions. Additionally, we included an EEG location (frontal–central–parietal) within-subjects factor in the ANCOVA model. The Greenhouse–Geisser corrected location * time * group effect did not reach significance ($p = 0.26$).

3.3. Effect of the feedback training on other frequency bands

An ANCOVA was used to test time * group effects on delta, theta, beta-1, beta-2, and gamma bands, at F3–F4, C3–C4 and P3–P4. As can be seen in Table 1, the training was associated with significant changes in symmetry in theta and beta-1 activity at F3–F4. Additionally, at C3–C4, delta activity changed significantly. No changes occurred at P3–P4.

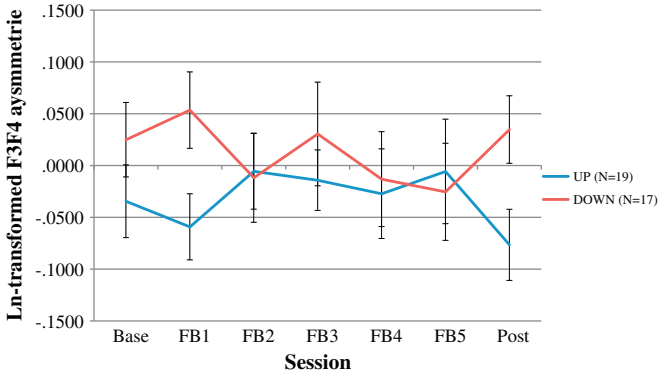

Fig. 1. Mean alpha-asymmetry between F3–F4 before, during (FB = feedback block), and after the training for both groups.

3.4. Effects of feedback training on momentary emotions

The mean scores of PA and NA in both groups before and immediately after the training are shown in Table 2. Pre–post differences were small. We did not find any significant pre–post difference in changes between both groups (positive versus negative; $F_{(Hot)} = 0.39$; $df = 1.34$; $P = 0.54$).

4. Discussion

This study is the first controlled investigation of the manipulation of frontal alpha-wave activity in a single neurofeedback session. Contingent on group membership, participants were able to decrease or increase AA. The groups were comparable with respect to AA because mean baseline AA was not different between the groups. The influence of between-subject differences in baseline AA was by-passed by defining changes in AA from the individual baseline for each participant. Our results are in line with an earlier study (Hardman et al., 1997), although this study targeted other cortical frequencies. A more direct comparison, given the same target frequency, can be made with the study by Allen et al. (2001), although their intervention consisted of NF sessions during 5 consecutive days as opposed to our single-session intervention. Participants in the study by Allen and coworkers were able to increase AA, but in contrast to our study, were not able to decrease AA. Moreover, significant changes in AA emerged only after 3 days of training, whereas in the current study it was apparent over a single session with repeated measures. Sampling variability, as well as methodological issues and sampling characteristics may explain these differences.

We have no explanation for the, albeit non-significant, changes of AA from baseline to FB1 in the opposite than hypothesized direction in both groups. There were no outliers that could explain this observation. One could speculate that it apparently takes some time before NF exerts its electrophysiological effects. In addition, a rebound to baseline AA was observed after the last NF episode indicating that the change in AA in a single NF-session is transitory. It is difficult to draw definite conclusions about this observation. The rebound seems to indicate that during NF meaningful electrophysiological changes indeed have occurred, but are only a temporary phenomenon after a single session. This observation may provide support for a common practice in NF; treatments typically last 20–30 sessions. Anecdotal evidence in a schizophrenic subject shows that the effect of NF-treatment on asymmetry may endure up to 3 months (Gruzelier et al., 1999). Thus, extensive repetitive feedback may be necessary for lasting changes in AA. Hardman et al. (1997) reported that there was no carry-over effect in their sample between the consecutive sessions separated by a few days. Although they did not report specifically the rebound that we observed, their data seems to point to a comparable phenomenon.

The manipulation of brain activity appeared to be specific in terms of intended electrophysiological changes. No changes in alpha-activity at other locations (C3–C4 and P3–P4) emerged during the intervention. Additionally, no changes in other frequency bands at these other locations were observed with the exception of delta-asymmetry at C3–C4.

Table 1
Effects of training on delta, theta, beta-1, beta-2, and gamma bands (F values) at different cortical locations.

	Delta	Theta	Beta-1	Beta-2	Gamma
F3–F4	1.62	3.57**	2.67*	1.51	1.62
C3–C4	3.05*	2.43	1.12	0.61	0.30
P3–P4	1.91	1.43	0.68	0.49	0.61

* $p < .05$.
** $p < .01$.

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Table 2
Mean scores (SD) on Positive Affect (PA) and Negative Affect (NA) before and after the training for both groups.

	PA		NA	
	Before	After	Before	After
DOWN	30.0 (5.7)	32.4 (7.5)	12.0 (2.8)	11.5 (2.8)
UP	29.2 (6.3)	30.2 (8.0)	11.5 (1.9)	11.1 (2.0)

In keeping with the changes in alpha-activity in prefrontal areas, significant changes (indicative of alterations in cortical activity) in theta and beta-1 bands occurred at the same locations in both groups. These cannot be explained by eye movements at frontal sites because EOG measures were used to reject invalid epochs. However, we cannot exclude the possibility that these changes in theta and beta-1 bands are associated with slow motor potentials at C3–C4.

No changes in mood were found in both groups. There are several explanations for this inconclusive finding. First, investigations into mood regulation in healthy subjects are vulnerable to floor and ceiling effects. Increasing already high levels of PA and decreasing low levels of NA in healthy subjects may be impossible or only be possible following repetitive interventions. This is probably not a sufficient explanation because participants in the UP-group, aiming at a decrease of PA and an increase of NA, similarly did not show mood alterations; in contrast with the DOWN group, floor and ceiling effects do not apply here. Second, it may well be that changes in emotion-regulation in healthy subjects cannot be found in basal mood *levels* ('resting state'), but only become apparent when mood *responses* to experimental stimuli are examined (Davidson, 2004). Earlier studies did not find evidence of mood changes, even after repetitive manipulation with NF, when no mood-induction was used (see; Coan and Allen, 2004; Coan et al., 2006), however exceptions exist when NF was applied for 9–10 sessions over several weeks (Gruzelier, in press; Raymond et al., 2005). These latter studies suggest the potential efficacy of NF as a treatment for affective disorders. Studies that did use a mood-induction procedure, reported associations between AA and emotional responses (Davidson and Fox, 1989; Harmon-Jones and Allen, 1997; Harmon-Jones and Sigelman, 2001; Wheeler et al., 1993). In keeping with these findings, alterations of emotion regulation during a NF intervention only became apparent when mood-inducing stimuli were used (Allen et al., 2001). Lastly, the AA in our participants returned quickly to their pre-intervention baseline levels. As mood was assessed only before and after the intervention, it may be that significant mood changes during the intervention remained undetected.

Our study has some limitations. First, although our results are commensurate with earlier studies (Allen et al., 2001; Hardman et al., 1997), it remains unclear whether the same findings apply to males and clinically depressed subjects. Because differences in cortical activity are known to be different between healthy and (previously) depressed subjects (Stewart et al., 2010), our study should be repeated in a clinical sample. Second, as outlined above, investigating mood changes without challenging emotional responses may have obscured significant effects of the NF manipulation. Third, nothing is known about the long-term effects of the training. We observed a rapid return to baseline AA scores after the last NF episode, indicating that any short-term change is highly transitory. A similar phenomenon was reported in a previous study even after training over 5 consecutive days (Allen et al., 2001). It can be hypothesized that lasting effects appear only after extensive training like in NF treatment sessions that typically last more than 15 sessions as opposed to the single session in the current study. Lastly, it is unknown if the observed electrophysiological changes occur as a direct result of the NF-intervention (Allen et al., 2001; Coan and Allen, 2004). It cannot be ruled out that an unmeasured third variable, for example one or more unknown cognitive or behavioral strategies used by the participants, mediates these changes. One way of addressing this limitation in future studies is to include sham-NF as one of the experimental conditions. However, such strategies may not be entirely conclusive. When, unexpectedly, similar electrophysiological changes are found in both real and sham-NF, different underlying causal processes may be responsible for these comparable outcomes. Lastly, participants were randomized into two groups without stratification, unlike Hardman et al. (1997) who examined changes in either direction within subjects. It is unknown if unmeasured personality and hemisphericity features have biased our results as we did not randomly assign subjects to the groups while stratifying for such features. Future studies should consider investigating changes in either direction within subjects while controlling for potential between-subjects differences instead of using two groups. A major strength of our study is the randomized single-blind design. Participants were unaware of the goals of the study, and the study conditions, expected for the opposite direction of the feedback, remained constant for all participants. This approach has also clear advantage over the inclusion of a sham procedure; using opposite feedback directions results in a potentially more robust contrast between conditions than using a sham procedure that may lead to more random results. Our design may be helpful for future studies that seek to investigate the validity of experimental manipulation of electrophysiological activity in the brain.

In this study, we have demonstrated that changing frontal AA in a single NF session in healthy participants is possible. Future randomized, double blind trials will have to show to what degree this approach is relevant for mood outcomes and efficacious for the treatment of MDD.

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