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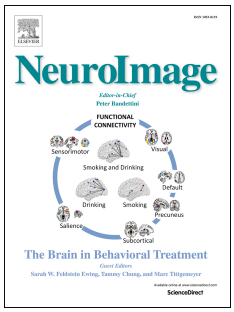

Success, but not failure feedback guides learning during neurofeedback: An ERP study

Ioanna Zioga, Rawan Hassan, Caroline Di Bernardi Luft

PII: S1053-8119(19)30486-0

DOI: https://doi.org/10.1016/j.neuroimage.2019.06.002

Reference: YNIMG 15924

To appear in: *NeuroImage*

Received Date: 1 July 2018

Revised Date: 30 March 2019

Accepted Date: 2 June 2019

Please cite this article as: Zioga, I., Hassan, R., Luft, C.D.B., Success, but not failure feedback guides learning during neurofeedback: An ERP study, *NeuroImage* (2019), doi: https://doi.org/10.1016/j.neuroimage.2019.06.002.

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Success, but not failure feedback guides learning during neurofeedback: An ERP study

Author names and affiliations

Ioanna Zioga[1], Rawan Hassan[1], Caroline Di Bernardi Luft[1]

[1] School of Biological and Chemical Sciences, Queen Mary, University of London

Corresponding author:

Dr. Caroline Di Bernardi Luft

E-mail address: c.luft@qmul.ac.uk

Permanent address:

School of Biological and Chemical Sciences

Queen Mary, University of London

Mile End Road

London E1 4NS

Office: +44 (0)20 7882 6604

Mobile: +44 (0)79 6486 4103

Success, but not failure feedback guides learning during neurofeedback: An ERP study

Abstract

Neurofeedback is a promising self-regulation technique used to modify specific targeted brain patterns. During neurofeedback, target brain activity is monitored in real time and fed back to the subject in a chosen format (e.g. visual stimulus). To date, we do not know how success and failure feedback are processed during neurofeedback learning. Here we analysed the event related potentials (ERPs) in response to success and failure feedback during a single neurofeedback session in two experiments. Participants in experiment 1 (n = 127) took part in one of the three neurofeedback conditions: RLA: trained to increase alpha power on the right frontal in relation to the left; LRA: the reverse of the RLA; FPA: trained to increase alpha power on the mid-frontal in relation to the mid-parietal region. In experiment 2 (n = 45), participants took part in a similar session but one group received random feedback whereas the other received valid feedback to increase right frontal alpha power. We analysed the feedback related negativity (FRN), correct positivity (CP), and P3a and P3b in response to success and failure feedback. We observed stronger FRN and CP in response to success compared to failure feedback. Additionally, the P3a in response to success feedback was higher in epochs preceded by subsequent good adjustments. Our findings indicate that people respond more strongly to success than failure feedback and that the P3a might mediate the encoding of the reinforced patterns in the brain.

Key-words: neurofeedback, FRN, ERP, learning, feedback.

1. Introduction

Neurofeedback is a technique which enables people to learn to regulate their own brain activity (Sitaram et al., 2017). During neurofeedback, target brain signals or patterns are recorded (e.g. EEG, fMRI) and presented to the participant in real time as feedback in any sensory modality (e.g. visual, auditory). The participant's task is to learn how to control the feedback by modifying her/his own brain activity, and several studies have shown that this learning can occur in as little as a single session (for a review, see: Enriquez-Geppert, Huster, & Herrmann, 2017). Neurofeedback is a promising technique which can be used to investigate brain function or to improve cognitive and affective function (Sitaram et al., 2017). For instance, it was observed that training to increase frontal alpha asymmetry (to the right) was associated with a reduction in stress (Quaedflieg et al., 2016) and mood disorders (Mennella, Patron, & Palomba, 2017). Neurofeedback has also been used to increase creativity in performing arts (Gruzelier, 2014).

The success of neurofeedback depends directly on how well people can learn to regulate the target brain signals. Therefore, it is crucial that we understand how people learn in this context. Most researchers in the field claim that learning during neurofeedback happens through operant conditioning. The first study reported with this technique monitored the activity of monkeys' single

neurons and delivered food pellets when these neurons increased firing (Fetz, 1969). It was found that the pairing of this target increase in firing with the reward was associated with an increase in neural firing, up to 500%. Neurofeedback was later used in humans: for example, feedback based on EEG has been used to increase control over alpha waves (for a historical perspective, see: Kamiya, 2011), and, more recently, neurofeedback has been used with fMRI to regulate activity in target brain regions with a high spatial resolution (for a review, see: Ruiz, Buyukturkoglu, Rana, Birbaumer, & Sitaram, 2014). Most studies in the field rely on operant conditioning as the main mechanism of learning, but do not evaluate the neural mechanisms associated with processing feedback during neurofeedback learning. As such, we know very little about how people process the feedback information for learning. This is interesting since the feedback is crucial for learning in this context.

To the best of our knowledge only one study to date has looked into how people process success and failure feedback in a neurofeedback session (Radua, Stoica, Scheinost, Pittenger, & Hampson, 2018). This study recorded fMRI signals during a neurofeedback session designed to increase activity in the orbitofrontal cortex. They observed that failure was associated with a deactivation of the cuneus and posterior cingulate cortex, whereas success was associated with the deactivation of the medial prefrontal cortex and anterior cingulate cortex. Interestingly, they observed that only the responses to feedback indicating success were associated with learning, suggesting different processes for learning from success vs. failure feedback information. Because this was an fMRI study, it is difficult to know the fast and dynamical responses to feedback, especially considering that in most used EEG-neurofeedback protocols, feedback is provided at every half second of brain activity, which is not well-captured by fMRI.

There is a great deal of research investigating very early and fast brain responses to performance feedback, rewards and punishments, and how they are associated with learning using EEG (some good reviews: Cohen, Wilmes, & van de Vijver, 2011; Ullsperger, Fischer, Nigbur, & Endrass, 2014; Walsh & Anderson, 2012). One of the most well-known event related potentials (ERPs) associated with feedback processing is the feedback-related negativity (FRN). The FRN is a negative deflection in the ERPs at the mid-frontal areas which starts as early as 140 ms following the feedback presentation (Miltner, Braun, & Coles, 1997). The FRN is sensitive to a number of parameters of the feedback, including its relevance, probability and learning (Walsh & Anderson, 2012). Other important ERP components associated with feedback processing are the correct positivity (CP) (Holroyd, Pakzad-Vaezi, & Krigolson, 2008) and the P300 or P3 (Polich, 2007). These signals are highly informative of the learning mechanisms involved in feedback guided learning (for a review: Luft, 2014), however no study has investigated whether they are similar in a neurofeedback task and how they enable learning of self-regulated brain activity.

In this study, we investigated the event-related potentials in response to success and failure feedback during a neurofeedback task. This is necessary since success and failure are not equally processed in the brain. Knowing how the brain learns from these two types of feedback is crucial for the development of more efficient neurofeedback protocols. It is of interest to understand how the brain responses to feedback affect how we learn to regulate brain signals. In most feedback learning situations, feedback information has to be constantly used to update our models of the environment. During neurofeedback, this brain response has to effectively update predictions about brain activity itself, which could possibly affect the process, creating a special type of feedback learning. In order to address this question, we conducted two experiments. In the first, we monitored the ERPs in response to success and failure feedback during a neurofeedback session using three different protocols to train a change in: right-left alpha brain asymmetry (RLA), left-right alpha brain asymmetry (LRA) and mid-fronto-parietal alpha difference (FPA). In this experiment, we focused on brain asymmetry neurofeedback since the RLA is often used to treat affective disorders (e.g. Mennella et al., 2017; Quaedflieg et al., 2016). In a subsequent experiment, we compared the ERP responses to success and failure feedback when the feedback was random (invalid) vs. when it was valid in a session which trained participants to up-regulate their alpha power in the right frontal region. We investigated: 1) the differences in the ERPs in response to success and failure neurofeedback, focusing on the main ERP components associated with feedback learning (FRN, CP and P3); 2) whether the ERP responses to success and failure feedback were associated with subsequent adjustments in brain activity; 3) whether these brain responses are dependent on the specific trained parameter; 4) whether the differences in the ERP responses to success and failure feedback are similar when the feedback is non-informative (random); 5) whether these differences remain significant in a protocol to up-regulate a single brain parameter.

2. Methods Experiment 1

2.1. Participants

One-hundred and thirty neurologically healthy adults (67 females) aged between 18 - 32 years (21.88 ± 2.63; Mean ± SD) with normal or corrected-to-normal vision (self-reported) took part in the experiment. Three participants were not included in the analysis due to noisy EEG data. Each participant was randomly assigned to one of three neurofeedback conditions: 1) Right alpha up (right/left) (RLA; N = 41): Participants were trained to increase alpha power on right frontal in relation to left frontal regions (electrodes F4/F3); 2) Left alpha up (left/right) (LRA; N = 43): Participants were trained to increase alpha power on left frontal in relation to right frontal regions (F3/F4); and 3) Mid-frontal alpha up (frontal/parietal) (FPA; N = 43): Participants were trained to

increase alpha power in the mid-frontal in relation to parietal regions (Fz/Pz). All groups were matched for age and gender.

Participants were recruited opportunistically through word of mouth and were reimbursed at a rate of £7.50 per hour. All participants gave written informed consent before the beginning of the experiment. The study protocol was approved by the QMUL college ethics board. Ethical considerations were met as all the data were kept anonymous and confidential, by using a unique identifier code for each participant. All participants were informed of their right to withdraw, and were debriefed at the end of the study.

2.2. Neurofeedback (NF)

Each participant completed three 5-minute bouts of neurofeedback (NF) while their EEG was recorded (see Figure 1). A fixation cross was presented in the centre of the screen for 1.5 minutes before and after each NF session while the resting state EEG was recorded with eyes open. At the start of each bout, a small white square was presented in the centre of the screen. Every 500 ms the power at the target electrodes (depending on the NF group) was calculated using *fast fourier transform*. The trained EEG patterns were different in each group. For the RLA, the natural log of alpha power (8-12 Hz) was calculated at the right and left frontal electrodes (F4 and F3) and then subtracted from each other ([F4 – F3]). For the LRA, the same procedure was adopted but the final value was the reverse subtraction ([F3 – F4]). Finally for the FPA, the index was the natural log of the alpha power on the mid-frontal minus mid-parietal ([Fz – Pz]). After each epoch was processed (600 epochs per neurofeedback bout), the participant received feedback (time-delay was tested and under 10ms). The feedback could be either success or failure. If the target EEG pattern increased, the size of the square increased and went green. If the target EEG pattern decreased, the square became smaller and red. The size of the increase/decrease was defined by its number of pixels, calculated as: increase: squaresize = current squaresize + (100 + squaresize*0.001); decrease: squaresize = current squaresize - (100 + squaresize*0.001). The square size changed at every epoch but only by the described proportional amount. Participants were required to try to increase the size of the square whilst learning to control and alter their own brain activity. As one bout was 5 mins and feedback was given every 500 ms, feedback was provided 600 times in each bout. The size of the square increased (or decreased) the same amount of pixels each time, independently of the amount of change of the EEG signal. Therefore, participants knew that they improved (or not), but were not aware of the amount of improvement (or decrement).

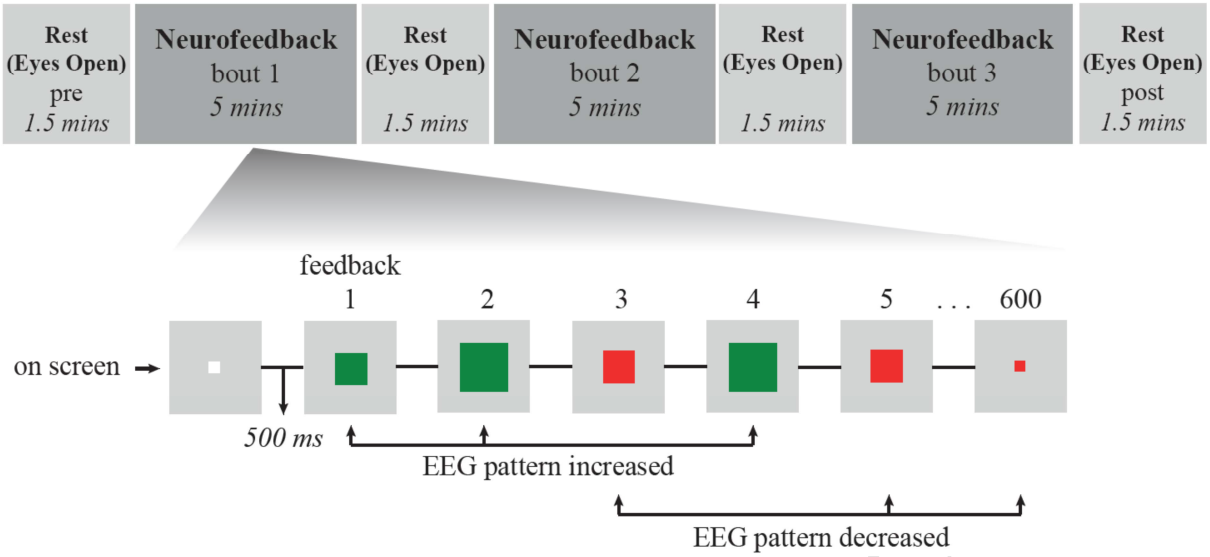

Figure 1. Illustration of experimental design. Each session consisted of three 5-minute neurofeedback (NF) bouts. Before, in between, and after the NF bouts, there were four 1.5-minute resting state blocks (top figure). EEG was recorded throughout the session. An example of a few NF trials is given (bottom figure). Every 500 ms the EEG is processed with Fast Fourier Transform (600 epochs per bout). If the EEG pattern increases (depending on the NF condition), the square becomes larger and goes green, whereas if it decreases it becomes smaller and red. It is important to notice that the change was only dependent on whether the trained pattern increased or decreased, which means that it was independent of the amount of the increase. This was done to ensure that the effects of the feedback valence were not confounded by the size of it and that the participants have some control over the feedback but not to the point of changing the proportion/probability of success and failure feedback.

2.3. Procedure

Participants were seated in front of a computer in a quiet room. Through written instructions on screen, they were informed that they would be trained to control their brain waves. They were instructed to try to increase the size of the square by manipulating their brain/thoughts. They were verbally informed that the size and colour of the square was related to their brain activity at that moment, as it would be analysed real-time. The overall duration was approximately 20 minutes. The neurofeedback task was programmed in Matlab. The communication between StarStim and Matlab was interfaced using *Matnic* (Neuroelectrics, Spain, http://www.neuroelectrics.com/products/software/matnic-remote-stimulation-client/) and the visual feedback was presented using the Psychtoolbox (Brainard & Vision, 1997).

2.4. EEG recording and pre-processing

The EEG signals were recorded with 18 PiStim electrodes placed according to the extended 10-20 electrode system (Jasper, 1958) using a battery-driven system (StarStim, Neuroelectrics, Spain). The EEG electrodes were: P8, F8, F4, C4, T8, P4, Fp2, Fp1, Fz, Cz, Pz, Oz, P3, F3, F7, C3, T7, and P7. Two ECG electrodes were attached to the right cheek bone to reduce noise, especially at 50 Hz.

The EEG data were re-referenced to the algebraic mean of the right and left earlobe electrodes (Essl & Rappelsberger, 1998). Continuous data were band-pass filtered from .5 - 47 Hz, and epoched from -.1 to 0.5 around the onset of the feedback. Data from electrodes with consistently poor signal quality, observed by visual inspection and by studying the topographical maps of their power spectra, were removed and reconstructed by interpolation from neighbouring electrodes. Subsequently, independent component analysis was run to correct for eye-blink related artefacts. Epochs with amplitude exceeding ± 70 uV were automatically removed. Further, the epoched data was low-pass filtered at 30 Hz, and baseline corrected to -100 ms before the feedback presentation. The data was averaged separately for each condition to analyse the ERPs.

2.5. Data analysis

Neurofeedback learning: In order to investigate whether any of the neurofeedback groups learned and improved during the neurofeedback session, we calculated the mean trained EEG patterns for each bout, separately for each group. Trained EEG patterns are defined differently for each group; for example, the trained EEG pattern of the RLA group is the value of the natural log of the right frontal alpha minus the natural log of left frontal alpha power. The higher the trained EEG pattern, the more successful the neurofeedback learning was. The statistical analyses adopted to address each question are described in the results section.

Feedback-related negativity (FRN) and correct positivity (CP) during neurofeedback: We analysed two ERP components elicited in response to failure and success feedback: first, a feedback-related negavity (FRN)-like component peaking around 120-200 ms after feedback, and, second, a correct positivity (CP)-like component peaking around 220-300 ms after feedback. Mean ERP amplitudes were calculated at the Fz electrode.

Neurofeedback adjustment following feedback: In order to investigate whether the responses to success and failure feedback actually resulted in adjusted brain activity during neurofeedback, we divided the data according to whether the feedback was followed by a good vs. bad/maladaptive change in the trained EEG index (increase vs. decrease, respectively). We analysed three ERPs: FRN (120 to 200 ms at Fz), P3a (220 to 300 ms at Pz) and P3b (320 to 450 ms at Pz)-like components. Mean ERP amplitudes were obtained for each of these component's time windows and locations. All

statistical analyses were conducted using the IBM Statistical Package for the Social Sciences (IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.).

3. Results Experiment 1

3.1. Neurofeedback learning

First, we examined whether participants successfully learned the neurofeedback (three different neurofeedback protocols) during the three 5-minute bouts. We entered the mean trained EEG indices for each group in each bout in a 3 (*neurofeedback condition*: RLA, LRA, FPA) X 3 (*training bout*: 1, 2, 3) mixed ANOVA. We observed a significant main effect of *training bout* ($F_{(2,248)} = 3.42$, $p = .034$, *partial* $\eta^2 = .027$) due to an increase in alpha power over the bouts, confirmed by a linear trend in the within-subject contrasts ($F_{(1,124)} = 4.97$, $p = .028$, *partial* $\eta^2 = .028$) (Fig. 2A). There was also a significant effect of *neurofeedback condition* ($F_{(2,126)} = 3.67$, $p = .028$, *partial* $\eta^2 = .056$), as the trained values were lower for the FPA group compared to the other groups (Fig. 2B). This is attributed to the fact that posterior alpha power is normally higher than frontal, making it hard for the FPA group to increase their frontal alpha compared to the posterior. Pairwise contrasts indicated significant differences between FPA and LFA (bout 1: $t_{(60.43)} = 2.27$, $p = .027$; bout 2: $t_{(61.3)} = 2.63$, $p = .011$; bout 3: $t_{(61.0)} = 2.31$, $p = .025$). There was no significant difference between the RLA and LRA groups in any of the bouts ($p > .3$). Importantly, there was no interaction between *training bout* and *neurofeedback condition* ($F_{(4,248)} = .516$, $p = .724$, *partial* $\eta^2 = .008$), suggesting that this effect was independent of the neurofeedback protocol. Pairwise contrasts showed that the trained EEG indices increased significantly from the first to the last bout ($t_{(126)} = 2.22$, $p = .028$), but only marginally from the first to the second ($t_{(126)} = 1.97$ $p = .051$) and not significantly from the second to the third bout ($t_{(126)} = .598$, $p = .551$). This suggests that participants improved incrementally from the first to the last bout of neurofeedback.

Considering that asymmetry is a relative measure, the participants could learn by either downregulating alpha in one site or up-regulating alpha on the other. For example, in the RLA condition the participants could learn by either increasing alpha power in the right frontal or by decreasing alpha power in the left frontal. Since there are different mechanisms associated with up and down-conditioning (Thompson, Chen, & Wolpaw, 2009), we investigated whether the observed changes in alpha asymmetry (right/left and frontal/posterior) were associated with increasing the activity in one site or decreasing in another. In order to evaluate that, we compared the alpha power values between neurofeedback bouts in each of the trained sites (F3, F4, Fz, Pz) and entered the values (separated by site) in a repeated measures ANOVA with training bout as a factor. We

conducted one ANOVA per neurofeedback condition (RLA, LRA, FPA). First, we observed a significant increase in alpha power over in the right frontal (F4) only in the RLA group ($F_{(1.36,57.1)}$ = 4.04, p = .037, *partial* η^2 = .088), but not in the LFA ($F_{(1.43,61.4)}$ = .295, p = .670, *partial* η^2 = .007), nor in the FPA ($F_{(1.64,68.7)}$ = 2.49, p = .100, *partial* η^2 = .056). Alpha power on the left frontal did not change significantly in the RLA group ($F_{(1.38,58.1)}$ =.572, p = .506, *partial* η^2 = .013) nor in the LRA group ($F_{(1.38,59.5)}$ =.647, p = .473, *partial* η^2 = .015), but increased in the FPA group ($F_{(1.53,64.3)}$ = 4.46, p = .023, *partial* η^2 = .096). We observed a significant increase in alpha power at the frontal midline electrode (FZ) over the FPA session ($F_{(2,1.51)}$ = 4.40, p = .025, *partial* η^2 = .095), but not for the groups which trained to change frontal asymmetry, including the RLA ($F_{(1.25,52.9)}$ = 1.82, p = .183, *partial* η^2 = .041) and the LRA ($F_{(1.45,62.4)}$ = .629, p = .487, *partial* η^2 = .014). Finally, we tested the differences in alpha power at the posterior midline (Pz) and observed no significant effect in any of the training conditions including RLA ($F_{(1.33,55.9)}$ = 1.84, p = .178, *partial* η^2 = .042), LRA ($F_{(1.73,74.3)}$ = .051, p = .931, *partial* η^2 = .001) and FPA ($F_{(1.64,68.8)}$ = 1.37, p = .259, *partial* η^2 = .032). Altogether, these findings seem to indicate that the participants might learn to change their brain asymmetry by up-regulating their alpha activity on one region rather than the opposite.

We also tested whether changes in the trained EEG indices would outlast the neurofeedback by comparing their values during rest before and after the session. We entered the trained values in a 2 (*resting state session*: before and after) X 3 (*neurofeedback condition:* RLA, LRA, FPA) mixed-design ANOVA (Fig. 2C). We observed no significant main effect of *resting state session* ($F_{(1,126)}$ = .149, p = .700, *partial* η^2= .001) or interaction between *resting state session* and *neurofeedback condition* ($F_{(1,126)}$ = .513, p = .600, *partial* η^2 = .008). There was a significant effect of *neurofeedback condition* ($F_{(2,126)}$ = 6.95, p = .004, *partial* η^2 = .099) due to the same reason explained above. The trained value of the FPA group was smaller than the asymmetry of the RLA and LRA in both pre- (FPA vs. RLA: $t_{(66.27)}$ = 2.28, p = .026; FPA vs. LRA: $t_{(56.39)}$ = 2.58, p = .013) and post-neurofeedback (FPA vs. RLA: $t_{(61.36)}$ = 2.99, p = .004; FPA vs. LRL: $t_{(58.1)}$ = 3.17, p = .002). There was no significant difference between the RLA and LRA groups in the pre- or post-neurofeedback session (p > .7).

Since this study is focused on the evoked responses to feedback, we looked into the percentage of success feedback in relation to the total feedback (total = success + failure). Even though the amplitude of the trained signal seemed to have increased over the session, we observed a similar percentage of success and failure feedback, around 50% (Fig. 2D). We entered the percentage of success feedback in a 3 (*neurofeedback condition*: RLA, LRA, FPA) X 3 (*training bout*: 1, 2, 3) mixed ANOVA. We observed no significant effect of *training bout* ($F_{(2,248)}$ = 1.68, p = .188, *partial* η^2 = .013) nor interaction with *neurofeedback condition* ($F_{(4,248)}$ = .960, p = .430, *partial* η^2 = .015). There was also no main effect of neurofeedback condition ($F_{(2,124)}$ = 2.41, p = .123, *partial* η^2 = .019).

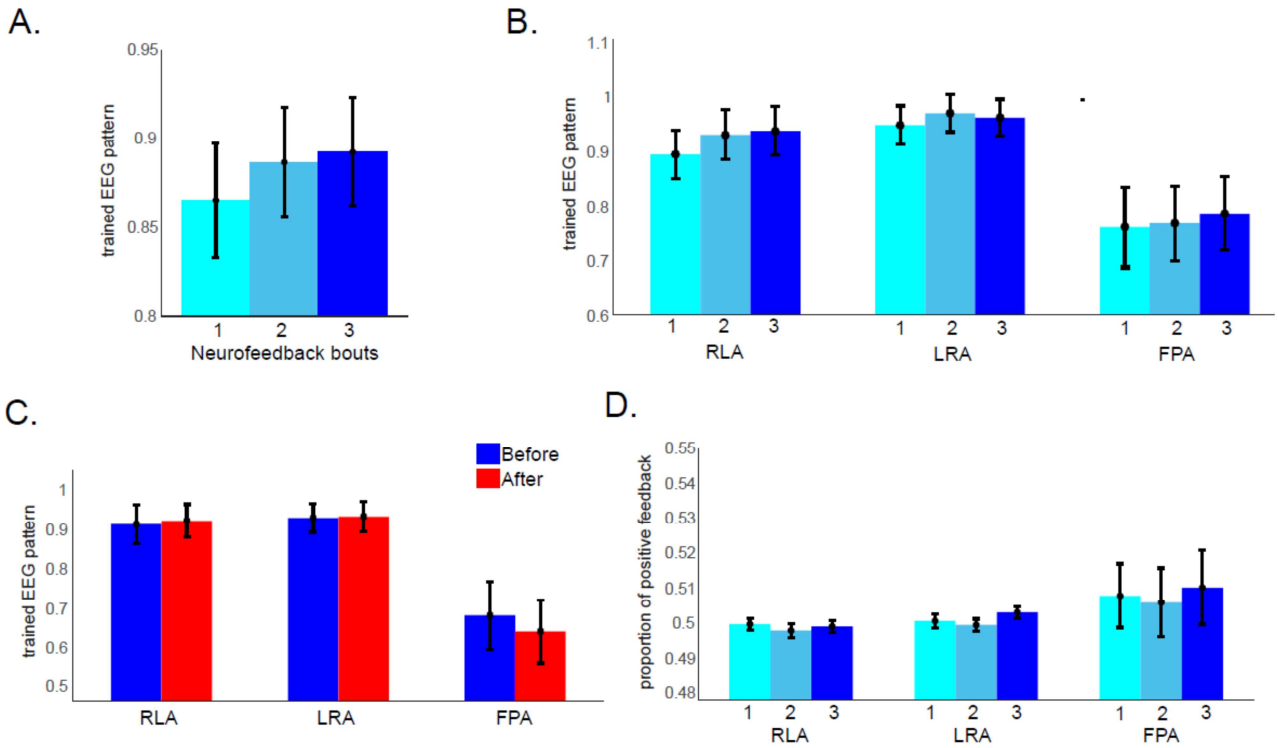

Figure 2. Performance before, during and after neurofeedback. A. Average trained EEG pattern (see in B) during the three 5-minute neurofeedback bouts. **B.** Average power ratio at the trained pattern in each neurofeedback condition and each bout. The trained pattern was different for each group – RLA: right alpha minus left alpha band power, LRA: left alpha minus right alpha, FPA: mid-frontal alpha minus mid-parietal alpha. **C.** Average trained EEG pattern during rest before (blue) and after (red) the neurofeedback session. **D.** Proportion of success feedback during each neurofeedback bout for each neurofeedback condition. Error bars represent +/- 1 S.E.M.

3.2. Feedback-related negativity (FRN) and correct positivity (CP) during neurofeedback

We investigated ERP responses to success and failure feedback during neurofeedback. An FRN-like component peaking at the fronto-central midline was observed around 120 to 200 ms after both types of feedback (Fig. 3AB). This component was followed by a large positivity resembling a correct positivity (CP) peaking between 220 to 300 ms after the feedback (Fig. 3AB). We analysed

these two components in two separate 2 (*feedback valence:* success vs. failure) X 3 (*neurofeedback condition*: RLA, LRA, FPA) mixed-design ANOVAs.

For the FRN (average amplitude at the Fz electrode from 120 to 200 ms after feedback), we observed a significant effect of *feedback type* ($F_{(1,125)}$ = 14.26, p < .001, *partial* η^2 = .102), since the FRN was larger following success compared to failure feedback (Fig. 2C). There was no significant effect of *neurofeedback condition* ($F_{(2,125)}$ = .394, p = .675, *partial* η^2 = .006), however, there was a significant interaction between *feedback type* and *neurofeedback condition* ($F_{(2,125)}$ = 3.75, p = .026, *partial* η^2 = .057). Follow up pairwise contrasts showed that only one of the groups (LRA) did not show a significant difference in the FRN between success and failure feedback ($t_{(42)}$ = .045, p = .964), whereas this difference was statistically significant for both the FPA ($t_{(42)}$ = 3.765, p < .001) and RLA groups ($t_{(41)}$ = 2.597, p = .013). For the CP-like component (average amplitude at the Fz electrode from 220 to 300 ms after feedback), we observed a significant effect of *feedback type* ($F_{(1,125)}$ = 35.43, p < .001, *partial* η^2 = .221), since the amplitude of CP was higher following success compared to failure feedback. There was no significant effect of *neurofeedback condition* ($F_{(2,125)}$ = .041, p = .959, *partial* η^2 = .001), and no significant interaction between the variables ($F_{(2,125)}$ = 2.84, p = .062, *partial* η^2 = .043) (Fig. 3D).

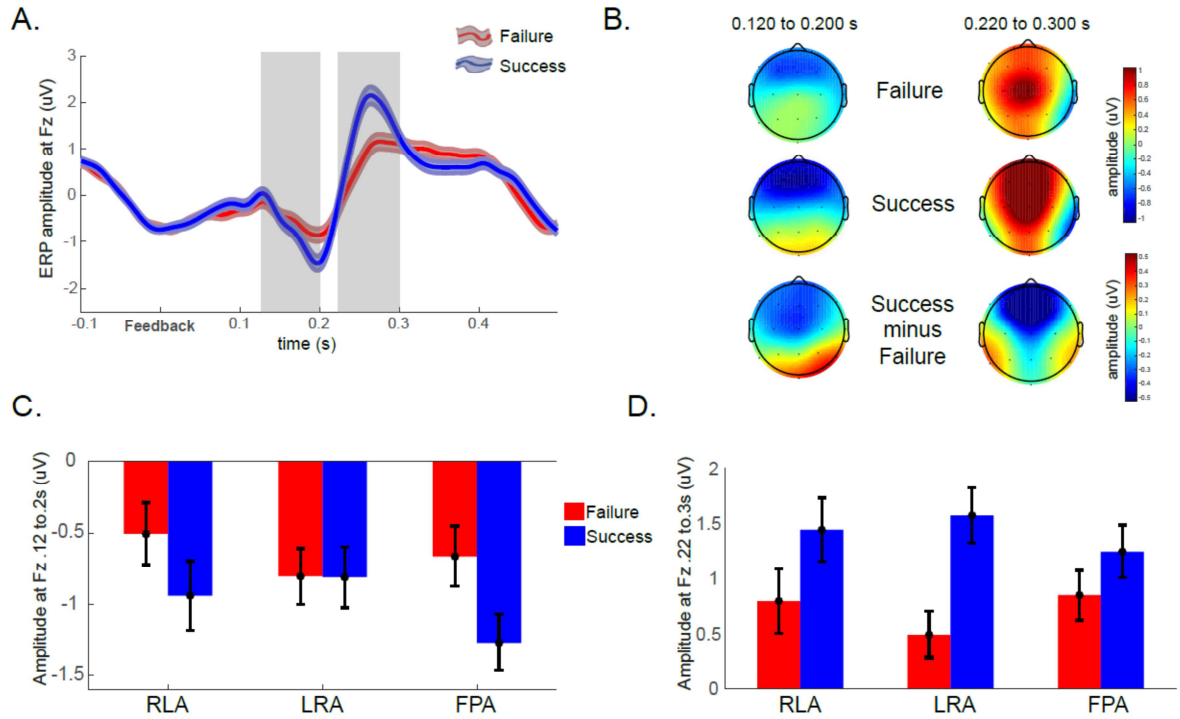

Figure 3. ERP responses to success and failure feedback. A. ERP waveforms in response to success (blue) and failure (red) feedback at the Fz electrode. The highlighted areas are associated with the FRN (feedback-related negativity) and CP (correct positivity) and their averages per condition are shown in C and D, respectively. **B.** Topographical distributions of

the ERP amplitudes following failure and success feedback, as well as the difference between them in the two time windows highlighted in A. **C.** Average amplitudes in response to feedback at Fz from 120 ms to 200 ms after feedback (first highlighted time window, FRN). **D.** Average amplitudes in response to feedback at Fz from 220 ms to 300 ms after feedback (second highlighted time window, CP). Error bars represent +/- 1 S.E.M.

3.3. Neurofeedback adjustment following feedback

In order to investigate whether the brain responses to feedback are relevant to adjusting brain activity during neurofeedback, we divided the data according to whether the success and failure feedback was followed by subsequent good vs. bad/maladaptive changes in the trained EEG patterns (increase vs. decrease, respectively). In Fig. 4A we present the ERP waveforms in response to success (left) and failure (right) feedback in trials followed by good (blue) and bad (red) adjustments. We analysed the FRN (120 to 200 ms at Fz), and P3 (P3a:220 to 300ms / P3b: 320 to 450 ms at Pz) -like ERP components. For each of these components, we entered the values as the dependent variable in a 2 (*feedback valence*: success vs. failure) X 2 (*adjustment:* good vs. bad*)* X 3 (*neurofeedback condition:* RLA, LRA, FPA) mixed-design ANOVA.

Regarding the FRN, there was no statistically significant difference between trials which were followed by a good vs. a bad performance adjustment (*adjustment:* $F_{(1,125)} = .007$, $p = .935$, *partial* $\eta^2 < 0.001$), and no significant interaction between *feedback valence* and *adjustment* ($F_{(1,125)} = .001$, $p = .979$, *partial* $\eta^2 = < 0.001$). There was a significant effect of *feedback valence* ($F_{(1,125)} = 4.745$, $p = .031$, *partial* $\eta^2 = .037$), since success feedback was associated with a higher FRN-like amplitude as previously shown.

With regards to the P3a-like component (Fig. 4A, second row), we observed a significant interaction between *feedback valence* and *adjustment* ($F_{(1,125)} = 5.07$, $p = .026$, *partial* $\eta^2 = .039$) since a stronger P3a was elicited in response to success feedback on trials immediately before a good adjustment or improvement. Pairwise contrasts indicated a significantly higher P3a in response to success feedback leading to a subsequent good adjustment compared to a bad adjustment ($t_{(127)} = 2.56$, $p = .012$), whereas there was no such a difference between P3a preceding good and bad adjustments following failure feedback ($t_{(127)} = -.637$, $p = .525$). No significant effects or interactions with the neurofeedback group were observed ($p > .1$), suggesting that this result was consistent across conditions (Fig. 4B).

The difference in the ERPs in response to feedback leading to good and bad performance adjustments carried on to a later time window corresponding to the P3b (320-450 ms, Fig. 4C). We

observed an interaction between *feedback type* and *adjustment* ($F_{(1,125)}$ = 8.09, p = .005, *partial* η^2 = .061), since the differences between good and bad adjustments were only significant in response to success feedback ($t_{(127)}$ = 2.98, p = .003) but not to failure feedback ($t_{(127)}$ = -.998, p = .326). We also found a three-way interaction between *feedback type*, *adjustment* and *neurofeedback condition* ($F_{(1,125)}$ = 3.93, p = .022, *partial* η^2 = .059): this reflected the fact that the FPA group also showed a difference between good and bad adjustments in response to failure feedback ($t_{(42)}$ = -3.76, p = .001), but this difference was not significant in the RLA ($t_{(41)}$ = .636, p = .529) or LRA feedback conditions ($t_{(42)}$ = 1.42, p = .164).

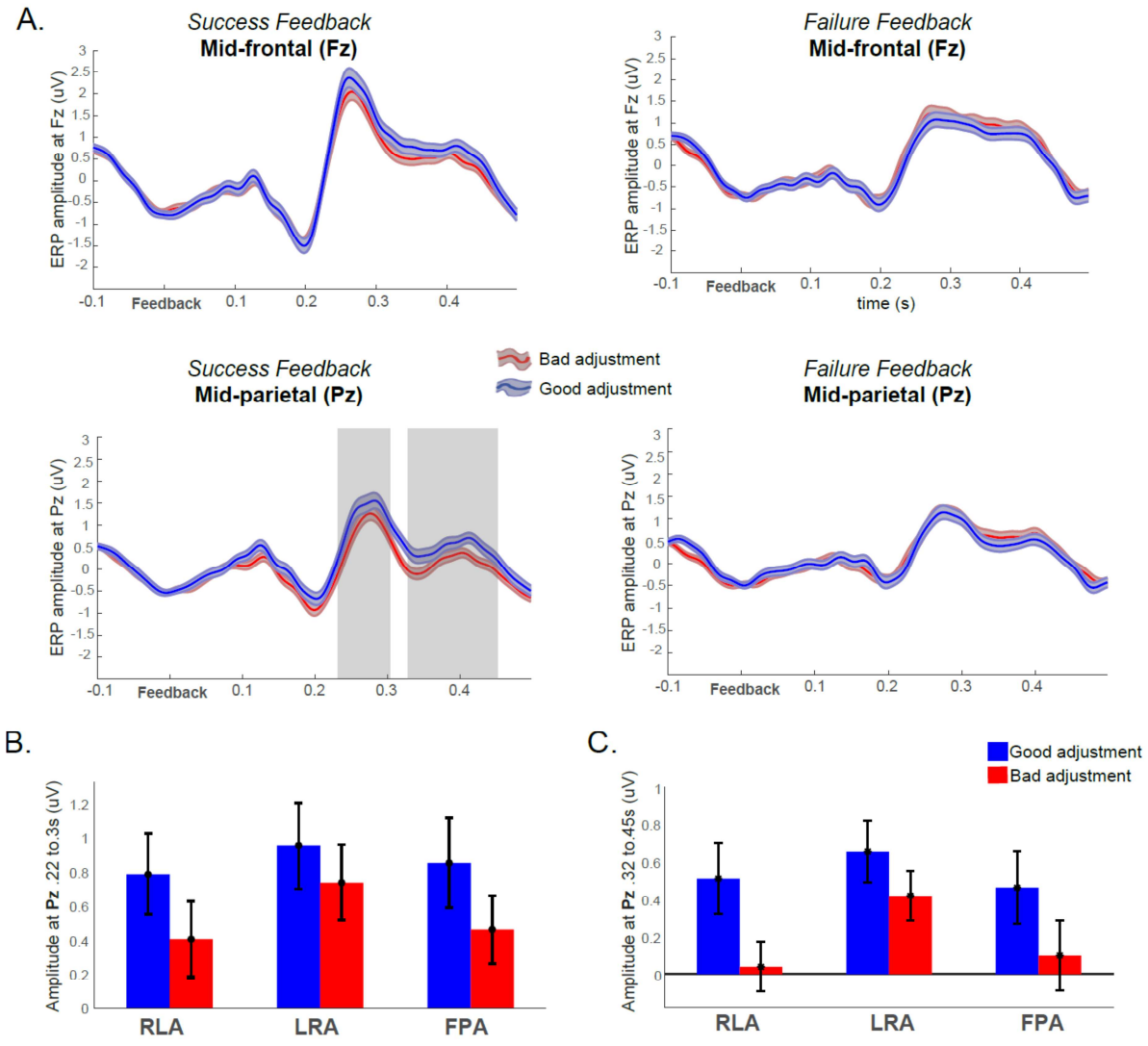

Figure 4. ERP responses to success and failure feedback in trials preceding good and bad adjustments in brain activity. A. ERP waveforms in response to success (left) and failure (right) feedback in trials followed by good (blue) and bad/maladaptive (red) brain activity adjustments at the mid-frontal electrode (Fz: top plots) and at the mid-parietal

electrode (Pz: second row). The highlighted areas show the time-windows associated with adequate adjustments in brain activity. **B.** Average amplitude at Pz during the P3a time-window (220-300 ms) in trials leading to good (blue) and bad (red) adjustments following success feedback. **C.** Average amplitude at Pz during the P3b time-window (320-450 ms) in trials leading to good (blue) and bad (red) adjustments following success feedback. *Note that the error bar figures for the failure feedback are not presented as we observed no effects in response to incorrect feedback. Error bars represent +/- 1 S.E.M.

4. Methods Experiment 2

We conducted another experiment to investigate the differences in the ERP responses to feedback when the feedback is invalid/random vs. when it is valid and the protocol targets an increase in alpha activity in a single region.

4.1. Participants

Fifty neurologically healthy adults (25 females) aged between 17 - 41 years (22.18 ± 3.81; Mean ± SD) with normal or corrected-to-normal vision (self-reported) took part in one experimental session. The participants were randomly assigned to one of the two conditions, to avoid potential carry-over effects of one condition to the other: 1) Random neurofeedback (N = 30); and 2) Right frontal alpha neurofeedback (valid feedback) (N = 20). Three participants had to be excluded due to technical problems and two participants were excluded due to poor data quality. The final sample was 26 participants in in the random feedback group and 19 in the valid feedback group. Participants gave written informed consent before the beginning of the experiment and were reimbursed at a rate of £7.50 per hour. The study protocol was approved by the QMUL ethics board. Ethical considerations were met as all the data were kept anonymous and confidential, by using a unique identifier code for each participant. All participants were informed of their right to withdraw, and were debriefed at the end of the study.

4.2. Neurofeedback (NF)

The neurofeedback session followed the same procedure of Experiment 1, i.e. there were three 5-minute bouts of NF, separated by 1.5 min of resting state EEG recording (Fig.1). However, for the random group the feedback was completely random: half of the times (300 epochs) the size of the square increased and it went green, whereas half of the times (300) the size of the square decreased and it went red (random order). The random feedback was defined by a random vector before the start of the experiment, and was different for each participant. The valid neurofeedback group received

success feedback everytime alpha power (calculated using the same methods described in experiment 1) increased in the F4 electrode (right frontal).

4.3. Procedure

Participants were seated in front of a computer in a soundproof room. They were given the same instructions as in Experiment 1, i.e. they were instructed to try to increase the size of the square by manipulating their brain/thoughts. The overall duration was approximately 20 minutes. The neurofeedback task was programmed in Matlab. The communication between StarStim and Matlab was interfaced using *Matnic* (Neuroelectrics, Spain, http://www.neuroelectrics.com/products/software/matnic-remote-stimulation-client/) and the visual feedback was presented using the Psychtoolbox (Brainard & Vision, 1997).

4.4. EEG recording and pre-processing

We used the same EEG set-up and followed the same pre-processing steps as in Experiment 1.

3.5. Data analysis

Neurofeedback learning: We calculated the mean relative alpha power in each neurofeedback bout for random and valid neurofeedback and also before and after the neurofeedback. The statistical analysis is described in the results section.

Feedback-related negativity (FRN) and correct positivity (CP) during neurofeedback: We analysed two ERP components elicited in response to failure and success feedback: first, a feedback-related negavity (FRN)-like component peaking around 120-200 ms after feedback, and, second, a correct positivity (CP)-like component peaking around 220-300 ms after feedback. Mean ERP amplitudes were calculated at the Fz electrode.

Neurofeedback adjustment following feedback: We compared the ERP responses to success and failure feedback which were followed by good vs. bad performance adjustments (i.e. increase in alpha power). We focused on the P3a (mean amplitude at Pz from 220 ms to 300 ms after feedback) and the P3b (mean amplitude at Pz from 320 ms to 450 ms after feedback) for this analysis since these were the significant components observed in experiment 1.

5. Results Experiment 2

5.1. Neurofeedback learning

First, we examined whether participants increased the trained pattern (right frontal alpha power) during the three 5-minute bouts in the real compared to the random feedback session (Fig.5A). We entered the mean trained alpha power for each group in each bout in a 2 (*feedback validity*: valid vs. random) X 3 (*training bout*: 1, 2, 3) mixed design ANOVA. The results showed a main effect of training bout ($F_{(2,86)}$ = 3.87, p = .025, *partial* η^2 = .082), reflecting an increase in alpha during the session. Since it increased in the same direction in both groups, there was no significant interaction between feedback validity and training bout ($F_{(2,86)}$ = 2.15, p = .127, *partial* η^2 = .047). Nonetheless, pairwise comparisons showed a significant increase in alpha power over the bouts only in the group which performed neurofeedback with valid feedback, from bout 1 to bout 3 ($t_{(18)}$ = 2.32, p = .032), but not significant from bout 2 to bout 3 ($t_{(18)}$ = 1.90, p = .073) or from bout 1 to 2 ($t_{(18)}$ = 1.14, p = .112). For the random feedback, alpha power was not significantly different in any contrast, between bout 1 and 3 ($t_{(25)}$ = 0.646, p = .524), bout 1 vs. 2 ($t_{(25)}$ = 0.81, p = .427) and between 2 and 3 ($t_{(25)}$ = -0.11, p = .911).

Second, we investigated whether these effects outlasted the feedback session. We compared alpha power over the right frontal electrode (F4) during rest before and after the neurofeedback session (Fig. 5B). We entered the values into a 2 (*feedback validity*: valid vs. random) x 2 (*resting state session:* before vs. after) repeated-measures ANOVA. We observed significant effects of *resting state session* ($F_{(1,43)}$ = 5.89, p = .019, *partial* η^2 = .121), a trend for the interaction between *resting state* and *feedback validity* ($F_{(1,43)}$ = 3.45, p = .070, *partial* η^2 = .074), and no main effect for *feedback validity* ($F_{(1,43)}$ = .672, p = .417, *partial* η^2 = .015). Pairwise contrasts were similar to the during session alpha contrasts: a significant increase in alpha in the post-test in the group that received valid feedback ($t_{(18)}$ = 2.25, p = .037), but not for the group which received random feedback ($t_{(25)}$ = 0.57, p = .574). Altogether these findings showed that there was a trend towards improvement for the valid feedback group, but the effects were not robust enough to show that solid learning occurred for the group.

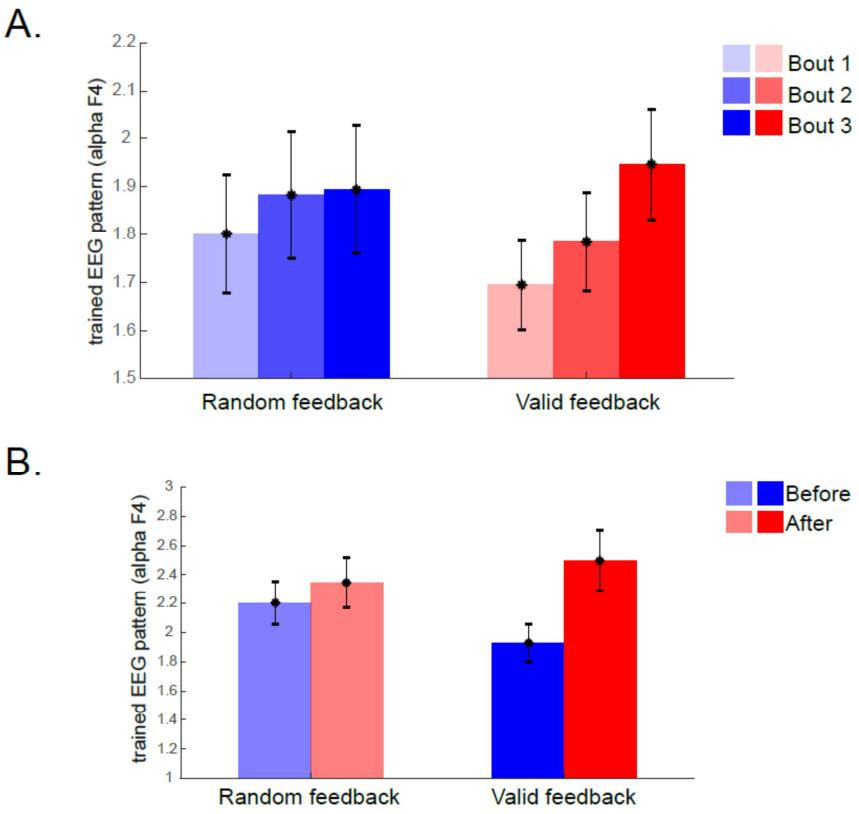

Figure 5. Performance before, during and after neurofeedback. A. Average trained EEG pattern (normalised alpha power at F4) during the three 5-minute neurofeedback bouts of the session with random (blue) vs. valid (red) feedback. **B.** Average trained EEG pattern during rest before (blue) and after (red) the neurofeedback sessions with random (transparent) vs. Valid (solid) feedback. Error bars represent +/- 1 S.E.M.

5.2. Feedback-related negativity (FRN) and correct positivity (CP) during neurofeedback

We investigated the differences in the FRN and CP in response to valid vs. random feedback. We entered the FRN values in a 2 (*feedback validity: valid vs. random)* x 2 (*feedback valence: success vs. failure)* mixed-design ANOVA. We observed a main effect for feedback valence ($F_{(1,43)}$ = 5.54, p = .023, *partial* η^2 = .114), replicating the key previous finding of stronger FRN in response to success feedback (Fig.6A). There was no interaction with feedback validity ($F_{(1,43)}$ = 0.009, p = .926, *partial* η^2 < .001), suggesting that this effect was similar when participants did the task with invalid/random feedback. Next, we conducted the same 2 x 2 mixed-design ANOVA using the CP as the dependent variable. Consistent with experiment 1, there was a strong effect of feedback valence ($F_{(1,43)}$ = 22.911, p < .001, *partial* η^2 = .348) since the CP was higher in response to success feedback. We observed a non-significant interaction trend ($F_{(1,43)}$ = 2.94, p = .094, *partial* η^2 = .064) between feedback validity and valence. This reflected the fact that the difference in the CP between success

and failure feedback was stronger in the participants who received valid ($t_{(18)}$ = 3.65, p = .002) vs. random ($t_{(25)}$ = 2.76, p = .011) feedback, even though the effect was statistically significant in both groups.

In order to track how the responses to feedback changed over the course of the feedback session, we compared the FRN and the CP across the three bouts of neurofeedback (Fig. 6B). First, we entered the FRN values in a 3 (*neurofeedback bout:* 1, 2, and 3) x 2 (*feedback valence:* success vs. failure) x 2 (*feedback validity: valid vs. random*) mixed-design ANOVA. Beyond the already observed main effects of *feedback valence*, we observed a significant interaction between feedback valence and neurofeedback bout ($F_{(2,86)}$ = 5.36, p = .006, *partial* η^2 = .111), showing that the differences between success and failure feedback were significantly higher at the beginning than the end of the session (see pairwise contrasts in Fig. 6C). There was a significant main effect of neurofeedback bout ($F_{(2,86)}$ = 3.86, p = .025, *partial* η^2 = .082) which suggests that the decrease in difference between success and failure was accompanied by a more general decrease in FRN. There were no significant effects of feedback validity nor other significant interactions (p > .1). We conducted the same statistical analysis using the CP as the dependent variable. Similarly to the analysis of the FRN, we observed a significant main effect of neurofeedback bout ($F_{(2,86)}$ = 6.02, p = .004, *partial* η^2 = .123), and this factor interacted with feedback validity ($F_{(2,86)}$ = 3.45, p = .036, *partial* η^2 = .074), as the group receiving valid feedback did not reduce their CP as much as the group receiving random feedback did. Pairwise contrasts indicate that the group receiving valid feedback showed a trend towards higher difference in CP between success and failure feedback over the course of the session, but the contrasts did not reach significance (p > .05, Fig.6D).

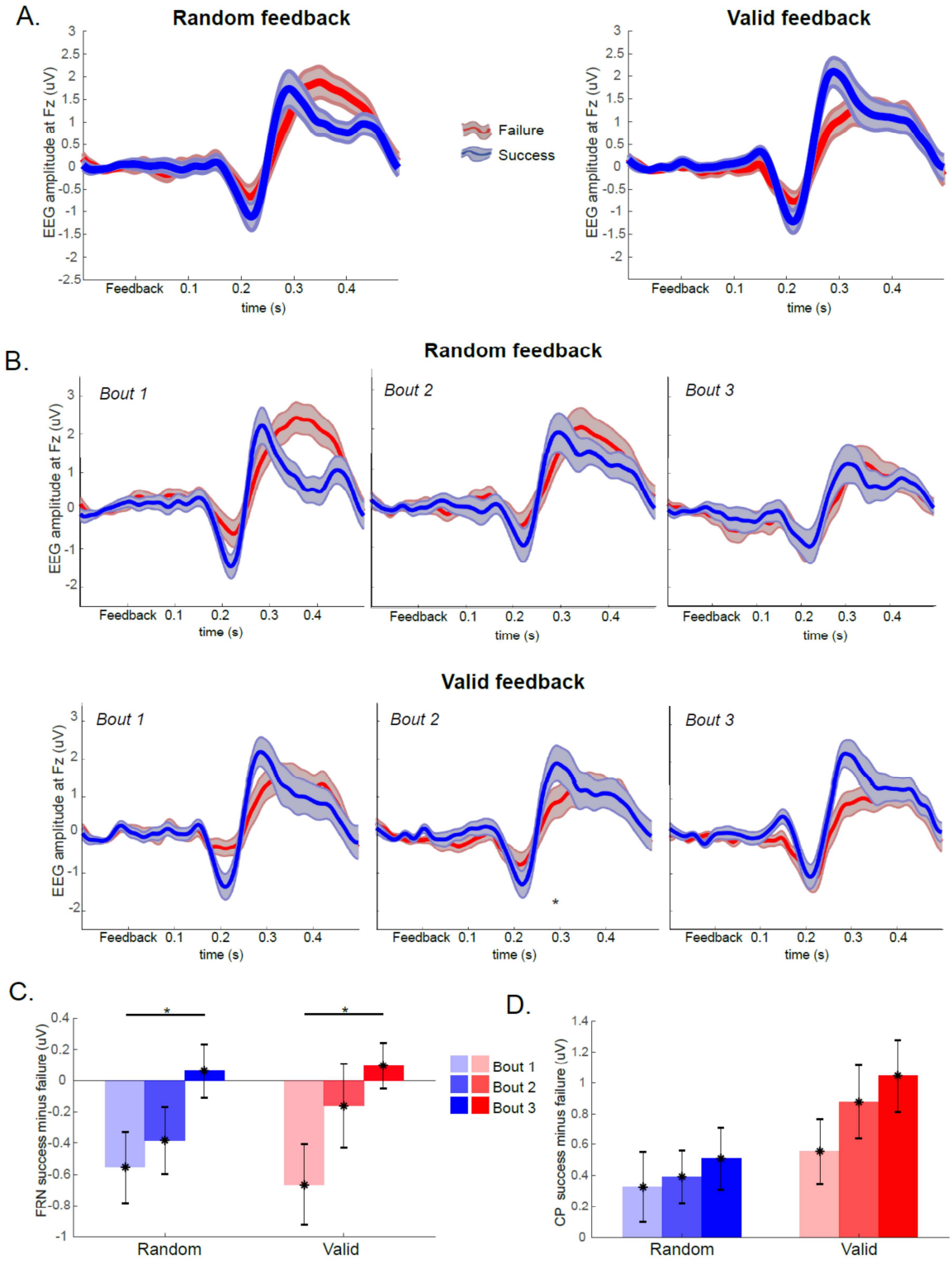

Figure 6. ERP responses to success and failure feedback in response to random vs. valid feedback. A. ERP waveforms in response to success (blue) and failure (red) feedback at the Fz electrode in response to random feedback (left) and valid feedback (right). **B.** First row: ERP responses to success (blue) and failure (red) feedback in the random condition in each bout (from left to right); Second row: the same ERP waveforms but in the valid feedback condition. **C.** FRN difference between success and failure feedback in each bout during

random and valid feedback neurofeedback conditions. **D.** CP difference in each neurofeedback bout during random and valid feedback. Error bars and shades represent +/- 1 S.E.M. *$p < .05$.

5.3. Neurofeedback adjustment following feedback

We tested whether stronger P3a and P3b components in response to success feedback were also predictive of a good adjustment, as was observed in experiment 1. We entered the P3a values in a 2 (*feedback valence:* success vs. failure) x 2 (*feedback adjustment:* good vs. bad) repeated-measures ANOVA. In the group receiving valid feedback, we observed a significant effect of feedback valence ($F_{(1,18)} = 6.27$, $p = .022$, *partial* $\eta^2 = .258$) and a significant interaction between feedback adjustment and valence ($F_{(1,18)} = 6.24$, $p = .022$, *partial* $\eta^2 = .258$), but no main effect of adjustment ($F_{(1,18)} = 2.12$, $p = .162$, *partial* $\eta^2 = .106$). This is because the differences in amplitude between good and bad adjustments were only significant following success feedback (see contrasts in Fig.7B). Importantly, there was no significant effect of these factors in the P3a in the group receiving random feedback: the main effects of feedback adjustment ($F_{(1,25)} = 0.81$, $p = .779$, *partial* $\eta^2 = .003$) and feedback valence ($F_{(1,25)} = 1.83$, $p = .188$, *partial* $\eta^2 = .068$), and the interaction ($F_{(1,25)} = .563$, $p = .460$, *partial* $\eta^2 = .022$) all failed to reach significance. We conducted the same analyses using the P3b as the dependent variable. As it is visible in Fig.7, none of the effects were significant ($p > .5$) except for the effect of feedback valence during valid feedback ($F_{(1,18)} = 4.90$, $p = .040$, *partial* $\eta^2 = .214$). This suggests that there is a possibility that the effects we observed in P3b in experiment 1 were a residual of P3a. The main findings (FRN, CP and P3a) of experiments 1 and 2 can be visualised in Figure S1 (Supplementary Material).

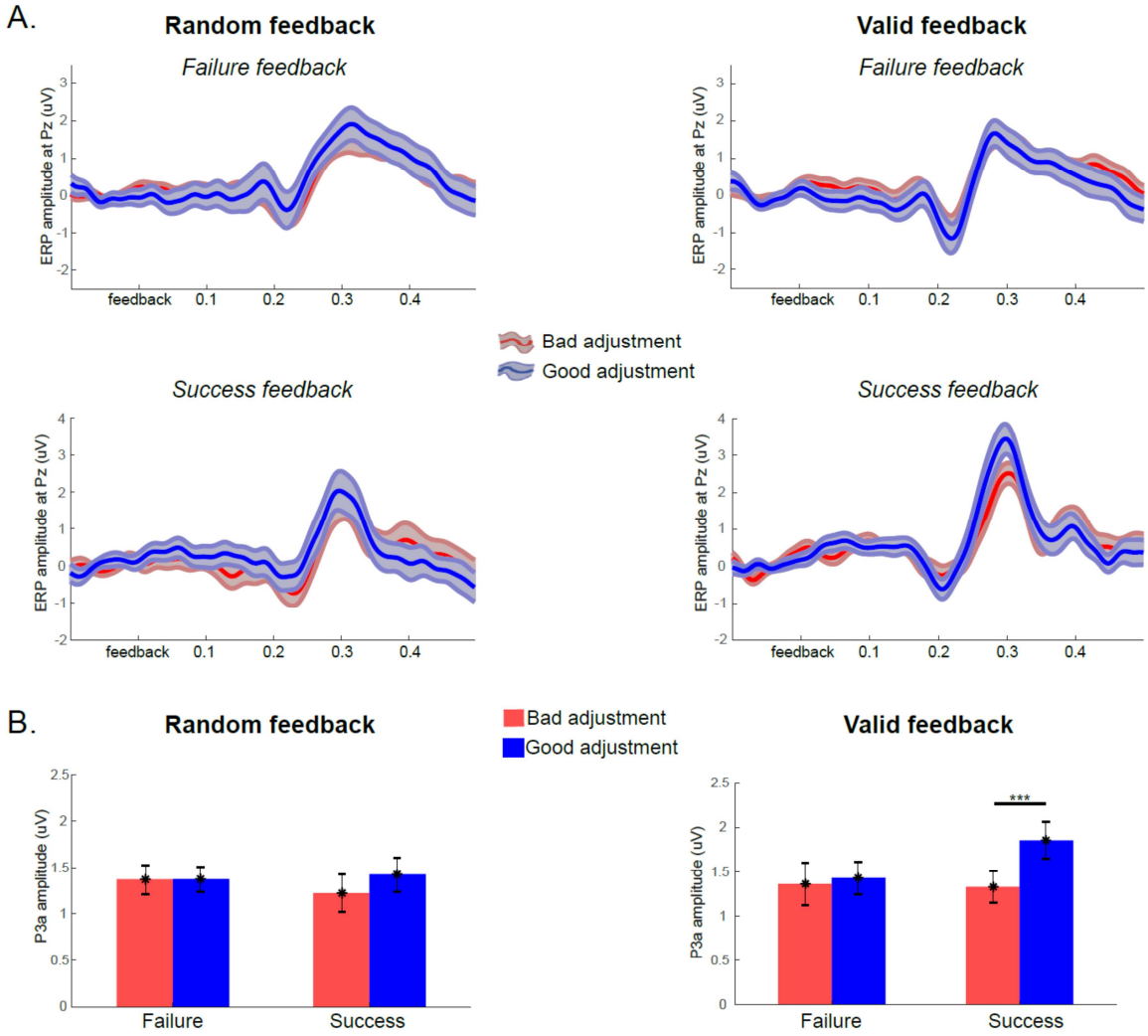

Figure 7. ERP responses to success and failure feedback in trials preceding good and bad adjustments in brain activity. A. ERP waveforms in response during random (left) and valid (right) feedback neurofeedback sessions in response to failure (top) and success (bottom) feedback followed by either good (blue) or bad/maladaptive (red) brain activity adjustments at the mid-parietal electrode (Pz). **B.** Average amplitude at Pz during the P3a time-window (220-300 ms) in trials leading to bad (red) and good (blue) adjustments following failure success feedback during sessions with random (left) and valid (right) feedback. Error bars represent +/- 1 S.E.M. *$p < .05$/ ** $p < .01$.

6. Discussion

This is the first study to investigate the event related potentials (ERPs) in response to feedback during neurofeedback. It is also the first to analyse how these responses are associated with

subsequent adjustments to the trained brain activity. We observed stronger ERP responses to success feedback, including higher feedback related negativity (FRN) and correct positivity (CP). The strength of the responses to success feedback on the later components (P3a and P3b) were associated with good adjustments on the subsequent epochs, whereas the responses to failure feedback were uninformative (unknown to the participants). All of these results, except for the P3b, were replicated in experiment 2, in which we contrasted random and valid feedback using a different neurofeedback protocol. Our findings contribute to the existing neurofeedback and feedback processing literature in four important ways. First, these results indicate that the ERPs in response to feedback during neurofeedback are similar to the ERPs in response to feedback in other learning contexts. Although this was hypothesised by Radua et al. (2018), our study is the first to examine this question directly. Second, we demonstrated that success feedback elicits stronger responses than failure feedback, suggesting that in a neurofeedback task, success feedback might be the most relevant for subsequent adjustments to brain activity. Third, we found that specific ERP responses to success feedback were higher preceding adaptive adjustment, whereas the responses to failure feedback were not. Finally, our results showed that the trained brain patterns improved during feedback in a single neurofeedback session, but not at rest once the feedback had ceased. In this section the discussion of the main ERP findings is followed by an explanation of how responses to success feedback can be quickly integrated to facilitate learning, elaborating on why success feedback might be more relevant to learning through neurofeedback.

Our findings confirm the prediction by Radua et al. (2018) that the processing of feedback during neurofeedback resembles the processing of success and failure in other feedback learning contexts. We found an FRN-like component in response to feedback, which is an important signature of feedback processing (Miltner et al., 1997) and the most investigated ERP component in the feedback learning literature (Walsh & Anderson, 2012). We observed a negative deflection starting around 120 ms after feedback and lasting until around 200 ms and peaking at the midfrontal region. This is similar to a typical FRN, although slightly earlier than originally described (Miltner et al., 1997). This negativity was observed following both failure and success feedback. This finding is surprising for three reasons. First, the FRN is hypothesised to be a signature of processing reward prediction errors (Holroyd & Coles, 2002) or unsigned prediction errors (Hauser et al., 2014), hence it is generally found to be higher in response to less likely outcomes (e.g. Cohen, Elger, & Ranganath, 2007; Hauser et al., 2014; Oliveira, McDonald, & Goodman, 2007; Walsh & Anderson, 2011; Walsh & Anderson, 2012). However, in the current study success and failure feedback were equally likely (both around 50%). Second, in situations where feedback types are equally likely (as in our study), we would expect the FRN in response to failure feedback to be higher due to the previously observed optimistic bias effect on the FRN (Oliveira et al., 2007). This study found the opposite: a higher FRN in response to success feedback. Third, the FRN seems to be more sensitive to failure than success

feedback (e.g. Hajcak, Moser, Holroyd, & Simons, 2006; Luft, Nolte, & Bhattacharya, 2013; Yeung & Sanfey, 2004). Since neurofeedback depends on implicit brain processes which cannot be easily monitored without external feedback, we suggest that the FRN codes the relevance of the provided information. This suggestion is supported by studies showing a stronger FRN in response to more reliable (e.g. Ernst & Steinhauser, 2018) and more informative (e.g. Schiffer, Siletti, Waszak, & Yeung, 2017) feedback.

We also observed a steep positive deflection on the ERPs, especially at the midline fronto-central area after the FRN. This component resembles the early positivity (early Pe) which is a positive deflection starting immediately after the FRN at the same mid-frontal region around 200ms after feedback (for a review see: Ullsperger et al., 2014). A similar positivity is the P2a, which was first observed in selective attention tasks (Kenemans, Kok, & Smulders, 1993) and it was later observed in response to rewards (Potts, Martin, Burton, & Montague, 2006). To avoid confusion, here we called this component correct positivity (CP). This component has been found to code the motivational relevance of the stimulus (Potts et al., 2006), which can be used to signal the need for enhanced control of the prefrontal cortex.

Here we suggest that the increased FRN and CP in response to success feedback indicate that this information is more relevant for learning how to regulate brain activity. Neither of these components were associated with adjustments in the trained brain activity, which suggests that they signal the importance of the event without necessarily assuring the integration of such information into the subsequent epoch. One important difference between a neurofeedback task and more traditional cognitive tasks is that learning is highly implicit during neurofeedback as it is not clear how one can control her/his own brain activity. For instance, a previous study observed that explicit instructions did not help neurofeedback learning (in fMRI) to control the activity in the supplementary motor area whereas monetary rewards did (Sepulveda et al., 2016). A previous review study from our group (Luft, 2014) observed that the FRN was only relevant for learning when the experimental task was explicit (e.g. probabilistic learning tasks). Therefore, we suggest that these components are associated with the initial processing of the feedback depending on its relevance for neurofeedback learning.

Regarding the incorporation of feedback information into the subsequent time, we observed that a later component around the P3 time-window was found to be associated with subsequent adaptive brain activity adjustment. The positive deflection started slightly earlier, around 200 ms, in the parietal region and lasted until almost the end of the epoch (around 450ms). The component resembles the well-known P3 (Sutton, Braren, Zubin, & John, 1965) which is a positive deflection in the ERPs in response to unexpected or relevant attentional stimuli of multiple sensory modalities. In the current study, the P3 was associated with adaptive adjustments to feedback. In line with previous

work in the feedback learning literature (for a review: Polich, 2007), we also identified two sub-components which we labelled the P3a and P3b. In an insightful review (Polich, 2007), it was suggested that the P3a increases in response to motivationally or sensory salient stimulus, leading to higher attentional mechanisms, whereas the P3b promotes memory operations in temporal-parietal areas for subsequent memory processing. In our case, both sub-components seem to have led to good adjustments on the following trial in experiment 1, which suggests that such operations are important for incorporating the feedback into the coming time-window for learning. However, in experiment 2 the P3b effect was not replicated, which seems to indicate that P3a is more relevant for successful short-term adjustments required in the neurofeedback task. In sum, then , our findings indicate that the P3a in response to success feedback is crucial for retaining the trained brain patterns during neurofeedback.

Importantly, our findings illuminate the discussion regarding the learning adjustment mechanisms of neurofeedback. Most researchers on neurofeedback theoretise that one can learn how to control her/his own brain activity through operant or instrumental conditioning (Enriquez-Geppert et al., 2017): in other words, promoting strong associations between a specific pattern of brain activity and a specific outcome can allow individuals to learn how to control this activity to obtain the desired outcomes. This was the key idea behind the first studies of neurofeedback with animals. For instance, Fetz (Fetz, 1969) recorded the activity of single neurons in the precentral cortex of unanesthetized monkeys and provided rewards when their firing rates increased (food pellet paired with auditory and visual feedback). He observed that monkeys increased the activity of newly isolated cells by 50 to 500%. It is important to note that this study relied on rewards (success feedback) as a teaching signal. Considering that the brain is a complex dynamical system which exhibits a large range of random and ordered activity (Chialvo, 2010), it has been hypothesised (Ros et al., 2016) that these fluctuating signals will eventually meet the threshold for reward, and after some repeated events/rewards they can be "tuned" to the feedback through synaptic plasticity. This will cause the brain to memorize a new "set-point" and tune to it for reaching the rewards. Notably, in such a scenario success feedback would be more informative. Failure feedback would be less informative since memorizing the brain activity leading to failure would require incredibly large memory resources given the infinite possibilities of variable brain states. Our current findings support this explanation since the FRN, CP and P3 were higher in response to sucess feedback and these signals are sensitive to the relevance of feedback information for learning (for a review: Luft, 2014). Since most neurofeedback studies in humans present provide both success and failure feedback (e.g. Mennella et al., 2017; Ros et al., 2016), we suggest that future studies test the efficacy of providing feedback only when the participant presents the desired pattern, as this might be more effective (as in Quaedflieg et al., 2016).

Interestingly, a recent study (Radua et al., 2018) analysing the brain responses to varying posititive and negative feedback using fMRI observed a progressive reduction of sensitivity to failure

and an increase in response to success feedback. They also observed that only the responses associated with processing success feedback (i.e. deactivation of the medial prefrontal cortex and anterior cingulate cortex) were correlated with neurofeedback learning. We suggest that it could be that the failure signals are processed more intensely at the beginning of learning, but as they do not provide enough information for effective learning, they start being inhibited in order to favour the most informative feedback for learning. In our study the differences between the ERPs in response to success and failure feedback reduced during the course of the session, and the ERPs were overall weaker, but this reduction was higher when the feedback was random. Altogether these findings might suggest that the responses to feedback reduce more when they are not informative or are redundant.

In this study, we can conclude that success feedback is processed more intensely than failure feedback during neurofeedback. We did not find consistent differences in feedback processing between different neurofeedback protocols, which might suggest that this is a general learning mechanism in EEG-neurofeedback protocols. As such, the feedback processing mechanisms we observed may be valid for a variety of neurofeedback protocols. However, our study was limited to a single and short session. Additionally, the learning we observed in this short session was weak and limited by the format of the feedback, which did not scale to the size of the change in the brain signal. Future studies need to investigate how these signals relate learning over multiple sessions, and how this could lead to long-term changes in resting state brain activity, which were not observed in this study.

5. Acknowledgments

We would like to thank *Leyla Al-Ashaab Mendiolea*, *Ellie Yorke*, *Naml Hussain*, *Ayesha Khan* for help with data collection. We would like to thank Dr Rachel Bennetts for proof reading and help editing the paper.

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