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MASTERARBEIT

Titel der Masterarbeit

„Here we go again - Test-Retest Reliability of the Feedback-Related Negativity in remitted patients and healthy controls“

verfasst von

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„The function of education is to reach one to think intensively and to think critically. Intelligence plus character - that is the goal of true education."

Martin Luther King, Jr.

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„It's the job that's never started as takes longest to finish.“

J.R.R. Tolkien, The Lord of the Rings

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Abstract

Feedback-related negativity (FRN) and P300 amplitude variations reflect sensitivity towards feedback outcome and have been linked to depression as they reflect the absence of the so-called reward-related positivity due to the dysfunction in the reward related system. The test-retest reliability of the FRN component is of great interest as it could validate the trait-like component to be a reliable biomarker for depression.

We examined the stability of FRN amplitudes at FCz and P300 amplitudes at Pz from time 1 to time 2 for 40 participants (20 remitted depressive patients, 20 healthy controls) with a monetary incentive delay task. Results showed in both groups medial-range reliability (ranging r =.41 to r=.64) in mean- and base-to-peak amplitudes, as well as interactions of feedback x group, cue x feedback, and main effects of run in repeated measurement ANOVAs. Most importantly, remitted depressive patients showed larger FRN amplitudes towards negative feedback than positive one. This effect could not be found for healthy controls. This negative focus of attention corroborates the notion that depressive patients are more sensitive towards negative information and cues and less responsive towards positive reinforcement.

1. Introduction

1.1 Electroencephalogram and feedback processing

Event-related potentials (ERPs), as measured by an electroencephalogram (EEG), can convey the processing of a given stimulus that is being perceived due to its high temporal resolution. This enables us to measure activity of the cerebral cortex seen in small voltage fluctuations which are being recorded and are due to currents that flow during synaptic excitation (Bear, Connors & Paradiso, 2006). These excitations can be inferred to cognitive processing and therefor to research fields like decision making and performance monitoring.

The research field of performance monitoring is of interest because it is concerned with error detection. Errors are a crucial part of human life and are inevitable on an everyday basis. The negative ERP deflection which occurs after an error is known as the error negativity (Ne) or the error-related negativity component (ERN; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, & Coles, 1993, Proudfit, 2014). As ERN and Ne are only performance error-related and do not require the need of a feedback concerning ones' performance further research was necessary. This particular ERP occurring after external feedback was a negative-going deflection which was first investigated by Miltner, Braun, and Coles (1997). Using a time estimation as a stimulus response task participants were in need of feedback to evaluate their performance. They discovered this negative feedback-related deflection, which occurred approximately 250-320ms after a negative feedback with a similar scalp distribution as the ERN. It was thought to reflect the error monitoring system and was then known as the feedback ERN (fERN, Miltner et al., 1997). After the findings of Holroyd and Coles (2002) regarding the connection of the fERN and reinforcement learn-

ing model, Gehring and Willoughby (2002) used a gambling paradigm to assess the ERN component after a loss irrespective of whether the choice was correct or incorrect. They found a negativity around 250 ms after feedback onset which did not appear after performance errors but only after losses and they defined it as the medial frontal negativity (MFN; Gehring, & Willoughy, 2002). As of then, the feedback-related negativity (FRN; Holroyd, & Coles, 2002; Nieuwenhuis, Holroyd, Mol, & Coles, 2004) or feedback negativity (FN; Hajcak, Moser, Holroyd, & Simons, 2006) was reported in papers using gambling paradigms (Proudfit, 2014). FRN amplitudes are more negative after salient outcomes in comparison to insignificant ones (Gehring, et al., 2002; Yeung, Holroyd, & Cohen, 2005) as well as after unexpected than expected events (Hajcak, Moser, Holroyd, & Simons, 2007; Pfabigan, Alexopoulos, Bauer, & Sailer, 2011). It is distinguishable between favorable and unfavorable outcomes with a higher amplitude for unfavorable outcomes (Gehring et al., 2002). Fluctuations in FRN amplitudes have been connected to classify feedback outcomes into an „better than expected“ or „worse than expected“ indicating that the FRN amplitude could serve as a reward prediction error which occurs if a person is surprised by an outcome (Hauser et al., 2014, Hayden, Heilbronner, Pearson, & Platt, 2011).

1.2 P300 amplitude

The P300 component is a positive ERP component which peaks at about 300-500 ms after stimulus onset (Sutton, Braren, Zubin, & John, 1965). Recent studies show a correlation of P300 amplitudes with neural activation in areas which are related to positive and negative anticipation (Pfabigan et al., 2014) as well as more pronounced amplitudes after positive than negative feedback (Hajcak, Moser, Holroyd, & Simons, 2007) which means P300 amplitudes are affected by feedback but opposed to the effect it has on FRN

amplitudes. In a study by Johnson and Donchin (1980) the P300 is similarly modulated like the FRN as it has a larger amplitude when a presented stimulus was unexpected than expected. It is therefor again dependent on feedback which can be expected or unexpected. Li and colleagues (2010) stated that the FRN and P300 amplitudes were increased if perceived responsibility of an outcome is high. A task which not only depends on feedback but also makes the participant responsible for a feedback will elicit higher amplitudes for P300 and FRN. The P300 has additionally been reported as a reward-related electrophysiological marker (Goldstein et al., 2006).

1.3 Feedback-related-negativity and depression

Just as the P300 component, amplitudes of the feedback-related negativity reflect a sensitivity towards reward and could be seen as a reflection of the absence of the so-called reward-related positivity in a monetary loss trial (Baker, & Holroyd, 2011; Bernat, Nelson, Steele, Gehring, & Patrick, 2011; Foti, Weinberg, Dien, & Hajcack, 2011; Bress, & Hajcak, 2013). Depression is characterized by a dysfunction in reward-seeking behavior (American Psychiatric Association, 2013) with its major symptoms concerning anhedonia and a lack of interest in any activities that are usually enjoyable as defined by the Diagnostic Statistical Manual (DSM-IV, Saß, Wittchen, & Zaudig, 1996) and the International Statistical Classification of Diseases (ICD-10, World Health Organization, 1992). As an increased depressive symptomatology accompanies a larger FRN amplitude, research relates FRN to depression and suggests greater FRN amplitudes as a possible biological marker for depression (Foti, Carlson, Sauder, & Proudfit, 2014; Bress, Mayer, & Hajcak, 2013; Bress, Mayer, & Proudfit, 2015).

Though anhedonia is for the most part a characteristic for the state of acute depression, research found that it can be a trait characteristic and also suggested it to be a trait-like potential endophenotypic marker of major depression (Hasler, Drevets, Manji, & Charney, 2004). Endophenotypic markers can be seen as internal phenotypes that can be discovered by any biochemical test or microscope and provide markers for identifying traits of clinical phenotypes (Gottesman, & Gould, 2003). Because the anhedonic symptoms remain constant to a certain point, even if a patient is in remission of an acute depression (Schrader, 1997), a patient with remitted major depression might be prone to have similar dysfunction in reward processing like acute depressive patients. Bress and colleagues (2015) stated that the feedback-related negativity component could be seen as a biological marker for depression. Their study showed a strong internal reliability of FRN amplitude variation at both time points (two years apart) and strong long-term stability. As their participants were neither acute nor remitted depressive patients but healthy children, a direct connection to a biological marker for depression is still questionable. Therefor, a comparison of patients with remitted depression and healthy controls over a certain time-span is necessary. To specifically address this question, a paradigm is required which allows a direct comparison of gains and losses, positive and negative outcomes, and also a comparison of expected and unexpected outcomes. The Monetary Incentive Delay Task (MID Task; Knutson et al., 2000) as a `stimulus response task' is considered as a suitable task. It allows an equal distribution of positive and negative as well as expected and unexpected outcomes. Additionally it enables a comparison of valenced feedback to a condition with neutral feedback.

The purpose of the current study is to assess the test-retest reliability of the feedback-related negativity amplitude variation regarding its trait-like stability as well as its possible purpose as a biological marker for major de-

pression. To this end, healthy participants and remitted depressive individuals will participate in the current study. High test-retest reliability for healthy controls and remitted depressive patients is expected. Since we assume the FRN component to have trait-like properties, we hypothesize that the FRN component does not differ in amplitude between the two sessions, separately in each group (Bress, 2015). In general, it is expected that the FRN amplitudes will be larger for remitted depressive patients than for healthy controls, due to the dysfunction in their reward-related neural system (Robinson, & Berridge, 1993). We further predict a larger FRN amplitude for unexpected in contrast to expected outcomes for both groups (Holroyd, & Coles, 2002; Hajcak, Moser, Holroyd, & Simons, 2006). In addition we hypothesize that the P300 component will be influenced by feedback and show a higher amplitude for unexpected in comparison to expected feedback in both groups.

2. Methods

The following experimental setup was part of a larger study, which included other measurements and paradigms out of scope of this master thesis. The chosen task had a duration of approximately 25 minutes depending on the breaks in between and the overall duration of the EEG experiment was about 1h per person

2.1 Participants

Twenty healthy controls (six men) and 20 matched remitted depressive patients (six men) from the age of 20 to 49 years (mean age 29.1, SD +/- 8.16) participated in the study (see fig. XX) with an average intelligence quotient (IQ) level of 104 (SD +/- 3.83) assessed by the Mehrfach-Wortschatz-Intelligenztest (MWT-B, Lehrl, 2005). Their handedness (18 right-handed in both groups) was determined by means of the Edinburgh Inventory (Oldfield, 1971). All participants were recruited through the ongoing research cluster MMI-CNS „Forschungscluster MAN-BIOPSY“ of the University of Vienna in cooperation with the Medical University of Vienna. Exclusion criteria were determined via a telephone screening and suitable participants invited to a diagnostic interview carried out by physicians (in training) of the Medical University of Vienna. In addition to the usual EEG exclusion criteria (neurological/high infectious diseases, diabetes or haemophilia, oversensitive skin, skull fractures as well as past or present substance abuse), fMRI exclusion criteria had to be met (no metal implants, pregnancy) due to the additional MR measurement which is not otherwise included in this thesis (for a full list of exclusion criteria see Table 1.). All participants had normal or corrected to normal vision and signed a consent-form preceding the measurement. The study was approved by the ethics

committee of the Medical University of Vienna (EC number 103/2011) and funded by the University of Vienna and the Medical University of Vienna, Austria.

The German version of the Structured Clinical Interview for disorders on axis I (SCID-I, First, Spitzer, Gibbon, & Williams, 2012) and II (SCID-II, Gibbon & Spitzer, 1997) of the diagnostic and statistical manual of mental disorders-IV-TR (DSM-IV) was conducted by all participants with a trained clinical interviewer. Participants who reported no history of neurological or psychiatric disorders were classified as healthy controls. All participants who reported prior history of depression but had no symptoms for the past year were classified as remitted with a HAMD-D (HAM-D, Hamilton, 1960) score of under ten points. The remitted and the control group had no significant difference in age, sex or IQ scores.

2.2 Procedure

After the diagnostic assessment of the SCID-I, SCID-II, and MWT-B (Lehrl, 2005) as described in section 3.1, participants had to additionally fill in questionnaires which will be described in detail in section 3.3.2. As part of the clinical study all participants underwent biological-physiological measurements which will not be included in the current thesis (electrocardiogram, hormones, blood analysis). If all criteria were met participants were invited to the fMRI experiment at the Centre for Medical Physics and Biomedical Engineering (Medical University of Vienna) which was implemented in the study. A few days later they were invited to the EEG experiment at the Social, Cognitive and Affective Neuroscience Unit (SCAN-Unit) at the University of Vienna. After three months both setups were repeated. Afterwards, all diagnostic and biological-physiological measurements were

repeated and all participants were debriefed and compensated with 120€ and the additional amount they won in the experiment.

The EEG experiment, which will be in focus of this thesis, consisted of three tasks. Following a resting state and a pain-paradigm (Seidel et al., 2015), the task which will be in scope was carried out.

2.3 Materials

2.3.1 Monetary Incentive Delay Task

The task to investigate the FRN component was a modified version of the Monetary Incentive Delay Task (MID Task; Knutson et al., 2000) which induces the anticipatory and executing phase of (monetary) reward processing. Participants were instructed to maximize gain and to minimize losses. The whole task was divided into four blocks where participants had to react to a square target-stimulus (presented on the screen in front of them) by pressing the button „1“ on a standard keyboard with their right index finger. Depending on the incentive cue (see Fig. 3.3.1) the participants were informed if the trial was an „attempt gain“ trial (plus symbol), an „avoid loss“ trial (minus symbol), or a „neutral“ trial (empty circle). In the gain condition, participants had the possibility to gain two euros if they pressed the button fast enough. In the loss condition, they could avoid the loss of two euros with a fast enough button press. The neutral condition did not result in any gain or loss, nevertheless participants were asked to respond to the target cue via button press as well. Following the incentive cue a question mark was presented for a varied time to ensure an unexpected target onset. Subsequently, the target square appeared followed by a black fixation point as a waiting sign. Depending on the incentive cue and whether the participant pressed the button within the given time-frame or not, feedback was given consisting of won or lost money and the total current money balance.

All participants started with a budget of 12€ and a short training session of six trials followed by the four blocks, consisting of 40 trials each (4x40=160), with a randomized presentation of the three incentive cues. An adaptive algorithm for the duration of the target stimulus resulting in adapted response-time windows for each incentive cue, which were shortened by 64ms after every correct and extended after every incorrect reaction, allowed an almost equal distribution of negative and positive feedback throughout the experiment resulting in approximately 32 trials of each condition: 64 gain cue trials (50% with a 2€ win [condition "gain"] and 50% without win [condition "no gain"]), 64 loss (50% with a loss of 2€ [condition „loss"] and 50% without loss [condition „no loss"]) and 32 neutral trials [condition "neutral"]. Task duration was about 25minutes depending on the chosen length of breaks. The overall earnings were on average 11.52€ (SD = 2.42), with 11.60€ (SD = 3.11) in the healthy control group and 11.45€ (SD = 1.50) in the remitted depressive group.

Figure 1. MID Task timeline overview

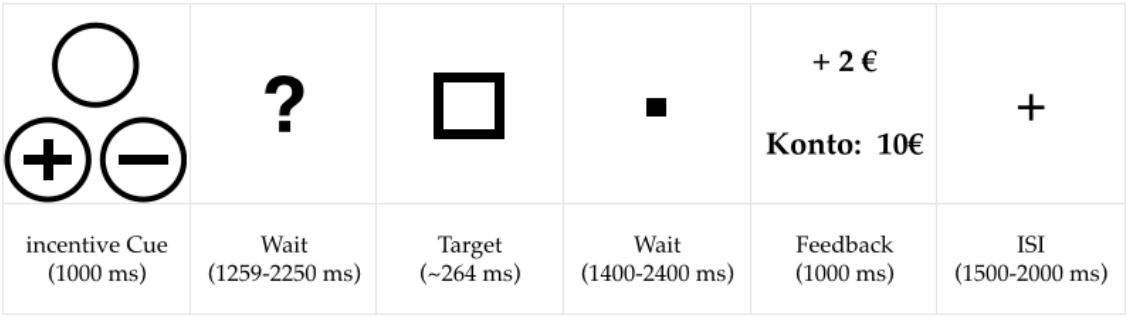

Figure 1: MID Task timeline summary displays the sequence of one trial. Incentive cue followed by the waiting cue and the target onset; afterwards another waiting cue is presented for a varied amount of ms, followed by performance feedback and the current balance. Between the feedback and next trial an inter-stimulus-interval (ISI) with a fixation cross is displayed.

2.3.2 Questionnaires

Furthermore a collection of general and disorder-specific questionnaires was assessed. To determine whether patients are currently suffering from major depression or anxiety scores on the Hamilton Depression Scale (HAM-D, Hamilton, 1960) and the Hamilton Anxiety Scale (HAM-A, Hamilton, 1959) each had to be under the total amount of ten. Additionally, the Beck Depression Inventory (BDI-II, Hautzinger et al., 1994), the State Trait Anxiety Inventory (STAI, Laux et al., 1981), and the Social Phobia Inventory (SPIN, Connor et al., 2000) were administered to ascertain negative emotionality and mental states of all participants.

2.4 Electroencephalographic recording

During each measurement stimulus presentation was controlled using E-Prime 2.0 (Psychology Software Tools, Inc., Sharpsburg, PA). With a distance of about 70cm in front of a 19“ CRT monitor with a 85Hz refresh rate (Sony GDM-F520), all participants were seated inside a sound-attenuated chamber. EEG was recorded using 61 Ag/AgCl electrodes arranged equidistantly, including FCz and Pz, on an EEG cap (model M10, EASYCAP, GmbH, Herrsching, Germany) including two electrodes placed over the left and right mastoids as off-line references. To further record electrooculogram (EOG) activity, two additional electrodes were placed above and below the left eye (vertical EOG) as well as two electrodes of the cap on the outside corner of each eye (horizontal EOG). The ground electrode (serving as online reference) was placed on the forehead. To ensure sufficient signal transmission during the experiment all impedances were kept below four kΩ. The EEG signal was amplified with a DC amplifier setup (NeuroPrax, Neuroconn GmbH, Ilmenau, Germany) with a sampling rate of 500Hz for digital storage.

3. Data analysis

The statistical analysis was performed using SPSS 22 (SPSS Inc., IBM Corporation, NY) as well as Statistical Data Analysis R (R Core Team (2013). R Foundation for Statistical Computing, Vienna, Austria) and Statistica 6.0 (StatSoft Inc., Tulsa OK).

3.1 EEG data

The off-line analysis of the EEG data was carried out with Matlab R2013a (The MathWorks, Inc. Natick, MA) using the EEGLAB 13.1.1b plugin (Delorme & Makeig, 2004). In the beginning, EEG data were filtered using a low-pass filter with a cut-off frequency of 30Hz and a high-pass filter with a cut-off frequency of 0.05Hz. All data were re-referenced in relation to the two mastoid electrodes average activity and epoched, starting 200ms before feedback onset, mean activity in this time window serving as a baseline, and lasting for 1200ms. These epoched data were initially visually scanned for striking movement artefacts and respective epochs were excluded from further analysis.

In the next step, an extended infomax independent component analysis (ICA, Bell & Sejnowski, 1995; Lee, Girolami, & Sejnowski, 1999) was conducted and all components which reflected eye movements artefacts were detected and discarded (Delorme, Sejnowski, & Makeig, 2007). Subsequently, all data were divided into the five conditions (gain, no gain, loss, no loss, and neutral) each consisting of approximately 30 trials each. In the following, a semi-automatic artefact correction was conducted in order to mark trials which voltage values exceeded +/- 60μV or which voltage drifts ranged > 50μV. Supported by visual inspection, assorted trials as well as tri-

als with obvious artefacts were eliminated from the following analysis. Resulting artefact-free trials were averaged participant- and condition-wise (gain, no gain, loss, no loss, neutral). Because there was no hypothesis concerning the neutral condition and feedback after the neutral incentive cue was not divisible in positive and negative outcomes, the neutral feedback was excluded from the main computation and analyzed separately. Each condition contained on average 26.85 (SD = 2.95) trials per condition.

A peak finding tool (BRL peak finder v.0.1b) was applied to determine peaks of the FRN component and the preceding (P200) and following positive peaks (P300) at FCz as well of the P300 component at Pz. Furthermore, mean FRN amplitudes were extracted within a timeframe of 200-300 ms (Gehring & Willoughby, 2002) at FCz, and mean P300 amplitudes at Pz within a timeframe of 300-400 ms (Picton, 1992) and P200 amplitudes in a timeframe within 150-200 ms (Potts, Dien, Harry-Speiser, McDougal, & Tucker, 1998) preceding after feedback onset.

Because the P200 and P300 amplitude had a strong impact on FRN amplitude variation, base-to-peak measures and mean FRN amplitudes were used for analyses. In a first analysis step, Pearson correlations of the first and second measurement were calculated for both groups (HC & REM) and for each group separately for FRN mean amplitudes, FRN base-to-peak amplitudes at FCz, and for P300 amplitudes at Pz.

Table 3.1.1 Correlation table

Pearson correlations		
HC base-to-peak FRN t1	—	HC base-to-peak FRN t2
REM base-to-peak FRN t1	—	REM base-to-peak FRN t2
HC mean amplitude FRN t1	—	HC mean amplitude FRN t2
REM mean amplitude FRN t1	—	REM mean amplitude FRN t2
HC base-to-peak P300 t1	—	HC base-to-peak P300 t2
REM base-to-peak P300 t1	—	REM base-to-peak P300 t2

Table 3.1.1.: Display of the planned Pearson correlations between the first and second measurement (time 1 and time 2).

The ERP components were analyzed separately in a 2x2x2x2 repeated-measure of analysis of variances (ANOVA). The factor *group* was used as the between-subject factor (healthy control, remitted depressive patient). For within-subject factors *incentive cue* (gain, loss), *feedback outcome* (positive, negative) and *run* (first and second measurement). The ANOVA was calculated using both base-to-peak and mean amplitudes to compare the result of each. For both the significance level was set to $\alpha = .05$ for data analysis. A further 2x2 ANOVA for *time* (first and second measurement) and *group* (HC & REM) to include the neutral condition in the analysis which was not possible in the 2x2x2x2 ANOVA due to the unbalanced design. Tukey post-hoc tests were computed for all significant interactions that were found in the repeated measurement ANOVA. Additionally, split-half reliability between even and odd trials was calculated using trial-wise mean amplitude values.

3.2 Behavioral data

To analyze behavioral data, the reaction time during target presentation following each incentive cue was calculated. Both groups were compared using a 3x2x2 ANOVA with *group* as the between-subject factor as well as *incentive cue* (gain, loss, neutral), and *run* as the within-subject factors.

3.3 Questionnaire data

All questionnaires were analyzed via an independent *t*-test to examine group differences concerning HAM-D (HAM-D, Hamilton, 1960), HAM-A (HAM-A, Hamilton, 1959), BDI (BDI-II, Hautzinger et al., 1994) and STAI (STAI, Laux et al., 1981) as well as to ensure no significant difference in IQ (via MWT-B (Lehrl, 2005)) between healthy controls and remitted depressive patients. All significance levels were set to $\boldsymbol{\alpha} = 0.01$ to avoid cumulative alpha errors due to multiple comparisons. As all remitted patients had to fill in all questionnaires after the second measurement to ensure they were still in remission, a paired *t*-Test was computed to ensure no significant difference in HAM-D, HAM-A, BDI and STAI between the two measurement time points. The SPIN (SPIN, Connor et al., 2000) questionnaire was additionally analyzed for all remitted depressive patients. To analyze associations between statements collected by questionnaires and the ERP amplitudes, Pearson correlations were calculated.

4. Results

4.1 FRN amplitude data

For the Pearson correlation with base-to-peak values calculated for both groups separately did show different levels of significance and correlations for each group. The highest correlation for healthy controls between the two session can be found in the gain condition (r = .556, p = .013) followed by the neutral (r = .627, p = .003), the loss (r = .483, p = .031), and the no gain condition (r = .419, p= .028). Only the no loss condition (p = .071) showed no statistical significance. The group of remitted patients had the highest significant correlation in unexpected conditions with no loss (r = .540, p = .014) and no gain (r = .652, p =.003), followed by the expected condition loss (r = .501, p = .024) and gain (r = .457, p = .043). The correlations in the neutral conditions (p = .132) showed no significance (see table 4.1.1) between the two sessions for remitted patients.

Table 4.1.1. FRN (base-to-peak) - main results of the Correlation between measurements (separated by groups): significant results are marked by ** (with * for $p \leq .05$ and ** for $p \leq .01$)

Group	FRN (base-to-peak) correlations	r	*p*
HC	gain-gain t1 * gain-gain t2	.617	**.004****
	gain No gain t1 * gain No gain t2	.419	**.028***
	loss-loss t1 * loss-loss t2	.483	**.031***
	loss No loss t1 * loss No loss t2	.413	.071
	neutral t1 * neutral t2	.556	**.013***
REM	gain-gain t1 * gain-gain t2	.457	**.043***
	gain No gain t1 * gain No gain t2	.521	**.018***
	loss-loss t1 * loss-loss t2	.501	**.024***
	loss No loss t1 * loss No loss t2	.540	**.014***
	neutral t1 * neutral t2	.579	.132

To compare the two groups with respect to their correlation coefficients, a Fisher z-transformation (see Leonhart, 2008) was applied. No significant difference (see table 4.1.2.) was found between the two groups as all p-values were above .05.

Table 4.1.2. FRN (base-to-peak) z-transformation

FRN (base-to-peak) correlations group difference	*z*	*p*
gain-gain HC * gain-gain REM	.661	.254
gain No gain HC * gain No gain REM	-.383	.351
loss-loss HC * loss-loss REM	-.069	.472
loss No loss HC * loss No loss REM	-.481	.315
neutral HC * neutral REM	-.099	.461

Table 4.1.2.: Display of the comparison of the correlation coefficient between the two groups via the Fisher z-transformation

The Pearson correlation for mean amplitudes which was calculated for each group separately did show significant correlations between the conditions. In healthy controls, the highest correlation between the two session was found in the neutral condition (r = .637, p = .003) followed by the expected gain (r = .627, p = .003) and loss conditions (r = .582, p = .007). No significance could be found (see table 4.1.3.) for the unexpected conditions no gain and no loss (both p-values > 0.05). In the group of remitted patients the highest significant correlation for no gain (r = .652, p = .003) was followed by the loss (r = .599, p = .005) and the gain condition (r = .498, p = .026). The correlations of the neutral conditions (p = .968) and the no loss conditions (p = .089) were not significant.

Table 4.1.3. FRN (mean amplitude) - main results of the Correlation between measurements (separated by groups): significant results are marked by ** (with * for $p \leq .05$ and ** for $p \leq .01$)

Group	FRN (mean amplitude) correlations	r	*p*
HC	gain-gain t1 * gain-gain t2	.627	**.003****
	gain No gain t1 * gain No gain t2	.439	.053
	loss-loss t1 * loss-loss t2	.582	**.007***
	loss No loss t1 * loss No loss t2	.352	.128
	neutral t1 * neutral t2	.637	**.003****
REM	gain-gain t1 * gain-gain t2	.498	**.026****
	gain No gain t1 * gain No gain t2	.652	**.003****
	loss-loss t1 * loss-loss t2	.599	**.005****
	loss No loss t1 * loss No loss t2	.391	.089
	neutral t1 * neutral t2	.010	.967

As above, a Fisher z-transformation and comparison of the two correlation coefficients of each condition for both group was performed to control for significant differences in correlation coefficients. For all p-values which were above .05 the correlation coefficient did not significantly differ (see table 4.1.4.) between the two groups. Consistent with the base-to-peak amplitudes only the neutral condition ($z = 2.167$, $p = .015$) showed a significant difference for the correlation coefficient between remitted patients and healthy controls.

Table 4.1.4. FRN (mean amplitude) z-transformation

FRN (mean amplitude) correlations group difference	***Fisher z***	***p***
gain-gain HC * gain-gain REM	.553	.290
gain No gain HC * gain No gain REM	-.897	.185
loss-loss HC * loss-loss REM	-.076	.470
loss No loss HC * loss No loss REM	-.132	.448
neutral HC * neutral REM	2.167	**.015***

Table 4.1.4: Display of the comparison of the correlation coefficient between the two groups via the Fisher z-transformation

The 2x2x2x2 repeated measurement ANOVA with base-to-peak FRN amplitudes shows a main effect for the factor feedback (positive/negative) ($F(1, 38) = 8.579$, $p = .006$, $\eta_p^2 = .184$) and a main effect for run ($F(1, 38) = 7.447$, $p = .034$, $\eta_p^2 = .112$). The FRN had significantly more negative amplitudes in the second measurement compared to the first measurement (see table 4.1.5. and figure 1). The main effects for cue ($F(1, 38) = 63.572$, $p = .066$, $\eta_p^2 = .086$) and group ($F(1, 38) = 0.139$, $p = .711$, $\eta_p^2 = .004$) were not significant. Interactions showed a significance for incentive cue and feedback ($F(1, 38) = 86.834$, $p < .001$, $\eta_p^2 = .696$)(see figure 3).

To investigate the interaction of incentive cue x feedback, a Tukey post-hoc test was calculated. The post-hoc comparison of base-to-peak FRN amplitudes showed that expected feedback (gain, loss) did not differ significantly (p-value > .144) from each other. The unexpected negative feedback (no gain) did not show a significant difference (p-value > .998) from unexpected positive feedback (no loss) in FRN base-to-peak amplitudes. Base-to-peak FRN amplitudes for both unexpected feedback conditions (no gain, no loss) were significantly more negative ($p < .001$) in respect to expected feedback (gain, loss). A more negative FRN deflection is found in case the incentive cue does not predict the feedback.

A further significant interaction (see figure 4 & 8) was found for feedback and group ($F(1, 38) = 6.369$, $p = .016$, $\eta_p^2 = .144$). The results of the Tukey post-hoc test showed that no significant difference ($p > .992$) could be found for the positive and negative feedback in the healthy control group. Only the group of remitted depressive patients showed a difference in FRN amplitude in comparison of positive to negative feedback. The FRN amplitude for negative feedback (no gain, loss) was significantly ($p = .002$) more negative than the amplitude for positive feedback (gain, no loss). No further comparisons showed significance (all p-values > .801).

No further significant interactions could be determined (all p-values > .291) in the base-to-peak repeated measurement ANOVA.

Table 4.1.5. FRN (base-to-peak) - main results of the repeated measurement ANOVA: significant results are marked by ** (with * for $p \leq .05$ and ** for $p \leq .01$)

FRN main effects and interactions	***F* (df1,df2)**	***p***	***ηp*²**
incentive cue(positive/negative)	3.572 (1,38)	.066	.086
feedback (positive/negative)	8.579 (1,38)	**.006****	.184
run (1/2)	7.447 (1,38)	**.034****	.112
group (HC/REM)	0.139 (1,38)	.711	.004
incentive cue x group	0.039 (1,38)	.845	.001
feedback x group	6.369 (1,38)	**.016****	.144
run x group	0.482 (1,38)	.492	.013
incentive cue x feedback	86.834 (1,38)	<**.001****	.696
incentive cue x feedback x group	1.148 (1,38)	.291	.029
incentive cue x run	0.001 (1,38)	.975	.000
incentive cue x run x group	0.418 (1,38)	.522	.011
feedback x run	0.380 (1,38)	.541	.010
feedback x run x group	0.552 (1,38)	.462	.014
incentive cue x feedback x run x group	0.018 (1,38)	.895	.001

The repeated measurement ANOVA with mean FRN amplitudes showed a significant main effect for incentive cue (positive/negative) ($F(1, 38) = 9.770$, $p = .003$, $\eta p^2 = .205$) and feedback (positive/negative) ($F(1, 38) = 14.035$, $p = .001$, $\eta p^2 = .270$), but no main effect for group (see table 4.1.6.). Though the main effect of run ($F(1, 38) = 3.878$, $p = .056$, $\eta p^2 = .093$) with an alpha-level over .05 is not significant, a trend is visible for the FRN amplitude to be more negative in the second measurement compared to the first measurement (see figure 2), as seen also in the base-to-peak repeated measurement ANOVA. A significant interaction for incentive cue and feedback ($F(1, 38) = 128.569$, $p < .001$, $\eta p^2 = .772$) was further analyzed using a Tukey post-hoc test. Similar to the findings for the base-to-peak amplitudes, the two unexpected conditions (no gain, no loss) did not differ significantly ($p > .985$) from each another. Unlike the post-hoc analysis of the base-to-peak amplitudes, a significant difference ($p = .015$) of gain and loss conditions was found. The condition loss had more negative amplitudes than the gain condition. All further comparisons showed a p-value < .001. No further significant interactions could be found (all p-values > .162).

Table 4.1.6. FRN (mean amplitudes) - main results of the repeated measurement ANOVA: significant results are marked by ** (with * for $p \leq .05$ and ** for $p \leq .01$)

FRN main effects and interactions	***F* (df1,df2)**	***p***	***ηp²***
incentive cue (positive/negative)	9.770 (1,38)	**.003****	.205
feedback (positive/negative)	14.035 (1,38)	**.001****	.270
run (1/2)	3.878 (1,38)	.056	.093
group (HC/REM)	0.387 (1,38)	.538	.010
incentive cue x group	0.058 (1,38)	.889	.001
feedback x group	6.447 (1,38)	.162	.051
run x group	0.102 (1,38)	.751	.003
incentive cue x feedback	128.569 (1,38)	<**.001****	.772
incentive cue x feedback x group	0.572 (1,38)	.454	.015

FRN main effects and interactions	*F* (df1,df2)	*p*	ηp^2
incentive cue x run	0.187 (1,38)	.668	.005
incentive cue x run x group	1.966 (1,38)	.169	.049
feedback x run	0.188 (1,38)	.667	.005
feedback x run x group	0.491 (1,38)	.488	.013
incentive cue x feedback x run x group	0.347 (1,38)	.559	.009

Because of the unbalanced experimental design, an additional 2x2 repeated measurement ANOVA (see table 4.1.7.) was calculated with the within-subject factor run and the between-subject factor group for the FRN amplitudes of the neutral condition, using base-to-peak amplitudes. As seen in the base-to-peak FRN ANOVA for the other four conditions, this ANOVA also showed a main effect for run ($F(1, 37) = 6.183$, $p = .018$, $\eta p^2 = .143$). The second measurement had more negative FRN amplitudes than the first measurement. Though the correlations showed that the neutral condition was only significant for the HC no main effect for group was found.

Table 4.1.7. FRN (base-to-peak) - main results of the repeated measurement 2x2 ANOVA for run and group: significant results are marked by ** (with * for $p \leq .05$ and ** for $p \leq .01$)

FRN main effects and interactions neutral (base-to-peak)	*F* (df1,df2)	*p*	ηp^2
run (1/2)	6.183 (1,37)	**.018***	.143
group (HC/REM)	1.540 (1, 37)	222	.040
run x group	0.020 (1,37)	888	.001

The calculated split-half reliability (see table 4.1.8.) for trial-wise mean FRN amplitudes of odd and even trials across the two groups for both runs showed a significant correlation for the expected condition loss ($r = .072$, $p = .020$). All other conditions showed no significance with a p-value $> .141$.

Table 4.1.8. FRN (trial-wise mean amplitudes) split-half reliability of odd and even trials for both groups: significant results are marked by ** (with * for $p \leq .05$ and ** for $p \leq .01$)

FRN Split-Half reliability	trials	r	*p*
gain-gain	odd*even	.005	.875
gain No gain	odd*even	-.005	.871
loss-loss	odd*even	.072	**.020***
loss No loss	odd*even	-.004	.910
neutral	odd*even	.045	.141

To check if any main effects or interactions occurred for the FRN latency in the two groups for incentive cue, run, feedback, and group, a repeated measurement 2x2x2x2 ANOVA (see table 4.1.9) was calculated. The main effect for incentive cue ($F(1, 38) = 4.614$, $p = .038$, $\eta p^2 = .108$) showed that the FRN differs in latency significantly in response to the incentive cue. The FRN component peaked significantly earlier after the positive incentive cue ($M = 253.475$ ms, $SE = 3.935$) compared to the negative incentive cue ($M = 258.863$, $SE = 3.414$).

Table 4.1.9. FRN (base-to-peak) latency repeated measurement ANOVA: significant results are marked by ** (with * for $p \leq .05$ and ** for $p \leq .01$)

FRN main effects and interactions (latency)	*F* (df1,df2)	*p*	ηp^2
incentive cue (positive/negative)	4.614 (1,38)	**.038***	.108
feedback (positive/negative)	1.173 (1,38)	.286	.030
run (1/2)	3.176 (1,38)	.083	.077
group (HC/REM)	0.212 (1,38)	.648	.006
incentive cue x group	0.364 (1,38)	.550	.009
feedback x group	0.027 (1,38)	.871	.001
run x group	0.183 (1,38)	.671	.005
incentive cue x feedback	1.504 (1,38)	.228	.038

FRN main effects and interactions (latency)	***F* (df1,df2)**	***p***	**ηp^2**
incentive cue x feedback x group	0.000 (1,38)	.986	.000
incentive cue x run	0.005 (1,38)	.945	.000
incentive cue x run x group	0.051 (1,38)	.822	.001
feedback x run	0.284 (1,38)	.597	.007
feedback x run x group	0.107 (1,38)	.746	.003
incentive cue x feedback x run x group	0.128 (1,38)	.723	.003

4.2 P300 amplitude data

The correlation of the P300 amplitude between the first and second measurement were significant for all conditions and both groups (see table 4.3.1.) with an alpha level at .05. The healthy control group had the highest correlation for the neutral condition (r = .776, p < .001) followed by the gain condition (r = .715, p = < .001). The conditions no gain (r = .668, p = .001), loss (r = .656, p = .002), and no loss (r = .634, p = .003) were also significantly correlated.

The remitted depressive group did not show as high correlations as the healthy control group. The highest correlation was found in the no loss condition (r=.663, p = .001), followed by the no gain (r = .651, p = .002), and loss condition (r = .613, p = .004). The lowest correlations were found in the gain (r = .559, p = .010) and neutral condition (r = .558, p = .011).

Table 4.2.1 P300 (base-to-peak) correlation between first and second measurement for both groups separately: significant results are marked by ** (with * for $p \leq .05$ and ** for $p \leq .01$)

group	P300 (base-to-peak) correlations	r	*p*
HC	gain-gain t1 * gain-gain t2	.715	**<.001** **
	gain No gain t1 * gain No gain t2	.668	**.001** **
	loss-loss t1 * loss-loss t2	.656	**.002** **
	loss No loss t1 * loss No loss t2	.634	**.003** **
	neutral t1 * neutral t2	.776	**<.001** **
REM	gain-gain t1 * gain-gain t2	.559	**.010** *
	gain No gain t1 * gain No gain t2	.651	**.002** **
	loss-loss t1 * loss-loss t2	.613	**.004** **
	loss No loss t1 * loss No loss t2	.663	**.001** **
	neutral t1 * neutral t2	.558	**.011** *

A repeated measurement 2x2x2x2 ANOVA with base-to-peak P300 amplitudes was calculated (see table 4.3.2.). The factor group served as the between-subject factor and incentive cue, feedback, and run as the within-subject factors. A main effect was found for incentive cue ($F(1, 38) = 5.193$, $p = .028$, $\eta p^2 = .120$) and feedback ($F(1, 38) = 10.453$, $p = .003$, $\eta p^2 = .216$). Additionally the analysis shows an interaction effect between the factor incentive cue and feedback ($F(1, 38) = 62.469$, $p < .001$, $\eta p^2 = .622$) (see figure 5). To further analyze the interaction a Tukey post-hoc test was assessed. Unlike to the FRN findings for the incentive cue and feedback interactions, the P300 had significantly higher amplitudes for the conditions with expected feedback ($p < .001$) than for unexpected feedback. Both expected conditions showed no significant difference from each another as well as both unexpected conditions (all p-values > .090).

Table 4.2.2. P300 (base-to-peak) repeated measurement ANOVA: significant results are marked by ** (with * for $p \leq .05$ and ** for $p \leq .01$)

P300 main effects and interactions	***F* (df1,df2)**	***p***	***ηp²***
incentive cue (positive/negative)	5.193 (1,38)	**.028** *	.120
feedback (positive/negative)	10.453 (1,38)	**.003** **	.216
run (1/2)	1.709 (1,38)	.199	.043
group (HC/REM)	.293 (1,38)	.591	.008
incentive cue x group	.341 (1,38)	.563	.009
feedback x group	.000 (1,38)	.984	.000
run x group	.203 (1,38)	.655	.005
incentive cue x feedback	62.469 (1,38)	<**.001** **	.622
incentive cue x feedback x group	1.390 (1,38)	.246	.035
incentive cue x run	1.027 (1,38)	.317	.026
incentive cue x run x group	.392 (1,38)	.535	.010
feedback x run	.020 (1,38)	.887	.001
feedback x run x group	.167 (1,38)	.685	.004

4.3 Behavioral data

For the repeated measurement 3x2x2 ANOVA a significant main effect for incentive cue ($F(1, 36) = 48.067$, $p < .001$, $\eta_p^2 = .572$) was found. Participants independent of run or whether they were HC or REM had longest reaction times for the neutral incentive cue (M = 242.11ms, SD = 30.90ms) followed by the incentive loss cue (M = 225.12ms, SD =28.76ms). The fastest reaction time was found for the incentive gain cue (M = 223.79ms, SD = 30.95ms). The main effect of run ($F(1, 36) = 0.141$, $p = .709$, $\eta_p^2 = .004$) and group ($F(1, 36) = 2.505$, $p = .122$, $\eta_p^2 = .065$) did not show significance. No further interactions (see table 4.3.1) showed any significance with an alpha level exceeding .490 (see figure 9).

Table 4.3.1. 3x2x2 repeated measurement ANOVA with **r**eaction time from target onset to button press: significant results are marked by ** (with * for p ≤ .05 and ** for p ≤ .01)

FRN main effects and interactions	***F* (df1,df2)**	***p***	**ηp^2**
incentive cue (gain, loss, neutral)	48.067 (1,36)	**<.001****	.572
run (1/2)	0.141 (1,36)	.709	.004
group (HC/REM)	2.505 (1,36)	.122	.065
incentive cue x group	0.785 (1,36)	.460	.021
run x group	0.005 (1,36)	.945	< .001
incentive cue x run	0.096 (1,36)	.908	.003
incentive cue x run x group	1.764 (1,36)	.179	.047

4.4 Questionnaire data

Following the analysis of FRN amplitudes the collected questionnaires were evaluated (see table 4.4.1.). A significant difference between HC and REM was found in the HAM-D ($t(20.323) = -3.800$, $p = .001$) as well as in HAM-A scores ($t(19.330) = -3.584$, $p = .002$) for the first measurement. Remitted depressive patients had significantly higher scores in HAM-D and HAM-A compared to healthy controls (see figure 4.4.2).

As in the HAM-D, a significant difference between HC and REM was found for the first measurement for BDI scores ($t(20.146) = -3.252$, $p = .004$), another questionnaire to determine the current degree of depression. Again, remitted patients show a significant higher BDI score in relation to healthy controls. The STAI showed a non-significant difference between the groups concerning state and trait anxiety ($t(26) = 2.540$, $p = . 017$). Additionally to the questionnaires for both groups, the REM group completed the SPIN ($M = 13.18$, $SD = 9.882$). With a score less than 20 social phobia can be ruled out. To additionally verify that the two groups do not differ concerning their IQ

level, the MWT-B (Lehrl, 2005) was analyzed and showed no significant difference between HC and REM ($t(32.574) = 0.375$, $p = .710$).

Table 4.4.1 Descriptive statistics of questionnaire scores with mean and standard deviation for both groups

Questionnaire	Group	M	SD
HAM-D t1	HC	.20	.410
HAM-D t1	REM	2.11	2.258
HAM-A t1	HC	.20	.523
HAM-A t1	REM	2.42	2.652
BDI t1	HC	.75	1.164
BDI t1	REM	4.32	4.643
STAI t1	HC	91.22	4.944
STAI t1	REM	85.05	6.416
SPIN	REM	13.18	9.882

Table 4.4.2 Analysis of questionnaires via independent *t*-Test comparing HC and REM; significant results are marked by ** (with * for $p \leq .05$ and ** for $p \leq .01$)

Questionnaire	*t*	df	p	SE	lower CI	upper CI
HAM-D t1	-3.800	20.323	**.001****	.500	- 2.942	- .858
HAM-A t1	-3.584	19.330	**.002****	.620	- 3.516	- .926
MWT-B	0.375	32.574	.710	1.309	-2.174	3.154
BDI t1	-3.252	20.146	**.004****	1.097	-5.852	-1.279
STAI	2.540	26	**.017***	2.210	1.563	10.777

To examine whether the remitted depressive patients had a change in their scores concerning their reported depression (HAM-D, BDI) or anxiety (HAM-A), a second measurement was conducted only for REM (see table 4.4.2.). A *t*-test did not show significant difference in HAM-D ($t(18) = 280$, p = .782), HAM-A ($t(16) = 1.059$, p = .305) or BDI scores ($t(16) = 1.083$, p = .295) between the first and second measurement in the REM group.

Table 4.4.2 Analysis of questionnaires via independent *t*-tests comparing the scores between the two measurements of REM

Questionnaire	***t***	**df**	**p**	**SE**	**lower CI**	**upper CI**
HAM-D	0.280	18	.782	.563	-1.026	1.341
HAM-A	1.059	16	.305	.778	- .825	2.472
BDI	1.083	16	.295	1.412	- 1.464	4.523

5. Discussion

The current study investigated whether or not the FRN amplitude in remitted depressive patients and healthy controls is stable over time using Pearson correlations of the two measurements. It was further investigated whether or not the reported difference in FRN amplitudes due to depression (Bress et al., 2015) and the dysfunction of the reward-related system of depressive patients (Robinson, & Berridge, 1993; Martin-Soelch, 2009) could be validated. Further, processing differences in unexpected and expected feedback processing could be validated with the FRN data. A difference for the processing of negative feedback was found in the remitted depressive group in comparison to healthy controls as they did show more enhanced FRN amplitudes, independent of the incentive cue.

5.1 Test-retest reliability

Using analysis of reliability to estimate whether or not a measure is sufficient to assess clinical change in cognitive states of participants is a common method. The test-retest reliability has been shown to be an adequate measure to assess reliability for ERP components (McEvoy, Smith, & Gevins, 2000). Researchers such as Segalowitz and colleagues (2010) were one of the first to assess test-retest reliability for the ERP component feedback-related negativity over a short and a long time period. Recent research suggests the averaging across multiple trials for an improved test-retest reliability instead of using single trials and to include at least 30 trials for each condition (Huffmeijer, Bakermans-Kranenburg, Alink, & van IJzendoorn, 2014). In the current study all these suggestions were successfully implemented. Similar to the study of Segalowitz et al. (2010) we can report high test-retest reliability of the FRN component at the electrode site FCz with correlations ranging

up to .64 in mean amplitudes in healthy controls. For base-to-peak measures, we also can observe the stability for gain and loss trials even though correlations could not be found for both groups equally. Because of the of the differing correlation patterns of FRN base-to-peak and mean amplitudes, split-half reliability of even and odd mean amplitude trials was additionally calculated as suggested by Gruendler, Ullsperger, and Huster (2011). The results showed weaker reliability than the test-retest reliability analysis which can be due to the combination of healthy controls and remitted depressive patients. Concerning our scoring method, we chose besides the mean amplitudes method also the base-to-peak method. It has been reported that peak-to-peak and base-to-peak methods are both equally contaminated by P300 component on which the FRN is „riding“ on top due to stimulus evaluation (Segalowitz et al., 2010). It was also reported that base-to-peak and peak-to-peak methods perform similarly at FCz, our side of interest (Segalowitz et al., 2010). Though Segalowitz and colleagues (2010) did not compare base-to-peak and mean amplitudes, we found that they do show similar results concerning the investigated test-retest reliability. Because mean amplitudes deal better with component variability than base-to-peak amplitudes, they are recommended for a comparison of a control group with a patient group (Luck, 2014). Additionally, peaks are very sensitive to variability in trial-to-trial latency which can result in averaged waveforms that are not comparable (Luck, 2014). This could be an explanation of the difference in correlations found in the two scoring methods. As our data showed similar trends in correlations and ANOVA results, we conclude both scoring methods as dependable for measuring test-retest reliability, but recommend a comparison of different scoring methods in this regard for future research.

5.2. The influences on FRN amplitudes

A finding which was consistently observed with both the base-to-peak and the mean amplitudes was the interaction of the incentive cue and feedback outcome. These results replicate the data from Pfabigan and colleagues (2015) who discussed the possibility of the FRN component as an absolute reward prediction error signal (aRPE). The aRPE in comparison to the negative reward prediction error serves as the modulation of the FRN which is due to motivational salience rather that a distinction in „worse than expected“ or „better than expected“. The impact of the incentive cue on feedback processing can be seen in the following. As our study showed equally to recent findings (Pfabigan et al., 2015), the incentive cue does impact the FRN amplitude as it determines whether a feedback was perceived as an expected (i.e., matching the valence of the incentive cue) or unexpected outcome (i.e., not matching the valence of the incentive cue). The resultant amplitude reveals that a feedback is dependent on the anticipation of the participants. Healthy controls and remitted depressive patients both showed that expected outcomes did elicit smaller FRN amplitudes than unexpected ones, leading to the conclusion that feedback processing is not only dependent on whether feedback is positive or negative but also on whether if it is expected or not. An effect which could be related to this finding is the priming effect of stimuli and the processing of the incentive cue. The reason why the feedback is unexpected could be due to the incentive cue that serves as a visual prime. As observed by Bentin, McCarthy and Wood (1985), primed and unprimed words differ significantly in their neuronal processing at around 200-250 ms following the stimulus onset which can be transferred to our feedback onset. One could consider the incentive cue a prime and the unexpected feedback the unprimed stimulus which shows greater negativity than primed (expected) feedback.

Though the interaction of feedback and incentive cue was expected, the further interaction of feedback x group found in base-to-peak amplitudes was not anticipated. Only remitted depressive patients showed a greater negative deflection in case the feedback outcome was negative. While our hypothesis predicted a more negative deflection for remitted depressive patients for negative feedback, we did not consider feedback to be independent of the incentive cue, most of all because of the expected interaction of cue and feedback. This finding underlines the difference in feedback processing also for remitted patients who were suffering from major depression and also the possibility of the FRN component being a biomarker.

These results could further validate data by Murphy and colleagues (2003) who found that misleading negative feedback for patients suffering from major depression disrupted their cognitive performance which was not found in healthy matched controls. As our study used an adaptive algorithm so ensure an equal distribution for negative (loss, no gain) and positive (no loss, gain) feedback conditions, the perceived feedback could have been experienced as misleading by participants. Some of our participants reported that they think that the task is being manipulated and that they were sure they were fast enough even though the feedback was negative. Our group of remitted depressive patients might have found the feedback in some of the trials misleading resulting in a disruption of their cognitive performance. Furthermore, it has been reported that depressed patients show a particular form of deficit or impairment concerning the response to negative feedback (Elliot, Sahakian, Herrod, Robbins, & Paykel, 1997). Research suggests that patients with major depression are more sensitive towards negative information and negative contextual cues and show less response towards positive reinforcement which validates the negative focus of attention (Chiu, & Deldin, 2007). Though all of the studies that have been executed so far agree on the fact that negative feedback is processed differently in de-

pressive patients and that the impairment seems to be a trait rather than a state of the illness, it has never been shown before that negative feedback is independent of contextual factors such as the incentive cues in the current paradigm (Elliot et al., 1997). The reason could be seen in the performance monitoring system in the anterior cingulate cortex (ACC) which is also activated due to violations in expectancy (Oliveira, McDonald, & Goodman, 2007).

Our study could not verify the influence of the so-called reward positivity which Proudfit (2014) states in his research. We could not find a smaller reward positivity for depressed patients in both conditions but only a larger FRN in the negative feedback condition. If the dysfunction in the reward system caused by depression would lead to reduced reward sensitivity and therefore a blunted reward positivity, a larger FRN amplitudes would be seen in both positive and negative feedback outcome conditions (Kujawa, Proudfit, & Klein, 2014). In the current study we only found larger FRN amplitudes in the negative feedback outcome condition which is in contradiction to the research by Proudfit (2014).

A reason why the test-retest reliability did not reach levels as high as expected could be explained by the observed effect of run in our data. As the effect was independent of the group variable, both groups had significantly higher FRN amplitudes for the second measurement. Similar results can be seen in the study by Segalowitz et al. (2010). Though their study did not include an ANOVA, their figures showed more negative peaks at the FCz electrode for the FRN component in the second measurement compared to the first measurement. We could validate the results by Huffmeijer et al. (2014) who stated that the FRN has a stable and strong stability within a task and shows distinguishable deflections for positive and negative feedback, but has a rather weak long-term stability and poor test-retest reliability. A possible reason for a larger FRN amplitude in the second measurement could be

the familiarity with the task. As reported before, participants had the assumption that to some degree the task was being manipulated. After they had told the investigator about the assumption they were told that it was not manipulated and that the outcome did indeed depend on their performance. This was done to ensure that participants' motivation stayed high and to prevent participants from cheating. It could be possible that participants therefore felt more responsible in the second measurement and thus they might have responded stronger towards the feedback (Li, 2010). Even though the two measurements did not show comparable FRN amplitudes in both time settings, a group effect was found which was stable over time. This suggests that the FRN component might have a trait-like stability and to be a feasible biomarker for depression.

Interestingly, FRN latency measures did not differ for healthy controls and remitted depressive patients, but they differed in regard whether the incentive cue was positive or negative. With a positive incentive cue, and thus the possibility of a gain, the FRN component peaked earlier than after the presentation of the negative or neutral incentive cues. In the study by Pfabigan, Alexopoulos, Bauer, and Sailer (2011) expected positive outcomes also had a shorter latency than expected negative outcomes. These similar results suggest a tendency for early processing of positive primes or cues. As previous studies showed (Schirmer, Kotz, & Friederici, 2005), positive incentive cues or primes lead to faster reaction times than negative and neutral ones. Possible faster processing for positive primes, which is seen in shorter reaction time and latency, might be due to the valence of positive stimuli (Leppänen, Tenhunen, & Hietanen, 2003).

5.3 P300 and depression

Compared to the correlations for base-to-peak and mean FRN amplitudes, the P300 showed high correlations between .56 and .77 over all conditions. This could be due to its distinctiveness and that it can be determined even in a task with a low trial count (Luck, 2014). Just as for the FRN component, an interaction of incentive cue on feedback outcome was found. Similar to the FRN this was expected as it replicates the results of the study by Pfabigan et al. (2015). While the incentive cue and feedback outcome interaction was found, we could not validate a group effect that was postulated for the comparison of depressed patients and healthy controls (Zhang, Hauser, Conty, Emrich, & Dietrich, 2006). Research by Zhang et al. (2006) proposed the P300 as a possible neurocognitive vulnerability marker for the development of a depressive disorder. We could not find this reported difference between the two groups in our data compared to the group difference which was found in FRN amplitudes. Given that the FRN is in need of more trials to show high test-retest reliability as the P300 (Huffmeijer et al., 2014), the found group effect in a minimal trial-number of 30 further validates its role as a reliable biomarker for depression.

The limitations concerning our study are the missing comparison of the current data with those of acute depressive patients. This is necessary to validate the current findings and to ensure that FRN amplitudes actually serve as a reliable biological marker or endophenotypic marker for depression. The correlation of the ERPs between two measurements is influenced by the number of trials as well as the number of electrodes used to collect the ERP data (Huffmeijer et al., 2014). Though we used two different scoring methods for defining our amplitudes, only one electrode (FCz) was used during the data

analysis. In future research, a mean over more electrodes could help to get better results in correlations for test-retest reliability.

Our study aimed to show that FRN amplitude variation could serve as a biomarker for depression. Though we could not fully validate its stability over time in depressive patients and healthy controls, we did find a group effect for negative feedback which distinguished remitted depressive patients from healthy controls. These findings might give advantage to upcoming research in the field of cognitive neuroscience as it shows a distinct difference in feedback processing, and therefor the difference in performance monitoring for patients with remitted depression in contrast to healthy controls.

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Appendix

Table 1: EEG and fMRI exclusion criteria

No EEG can be conducted if one of the following criteria are violated:

1.) Skull fracture, Implants on the head

2.) Eczemas, birth marks on the scalp, or if your scalp is lightly irritable

3.) contagious blood disorder (e.g. HIV)

4.) fear of needles

5.) if you have diabetes or homeopathy

6.) neurological disorder e.g. lightheadedness, epilepsy

7.) regular use of medication (psychotropic drugs)

No fMRI can be conducted if one of the following criteria are violated:

1.) Any metal parts on the body

   a. implants or prostheses
   b. dentures, retainer
   c. cardiac pacemaker
   d. vessel clip/wires on blood vessels
   e. infusion pump
   f. shell splinter / remains of gunshot injury
   g. piercings
   h. if woman: contraceptive coil made of copper

2.) permanent makeup, larger tattoo

3.) dependence of alcohol, drugs, medication

4.) Hypertension, disease in kidney, lung, thyroid, or heart, operations on the head, heart of throat

5.) epilepsy, stroke, other neurological disease

6.) if woman: pregnancy

7.) claustrophobia

Figure 1: Main effect of run in base-to-peak amplitudes over both groups.

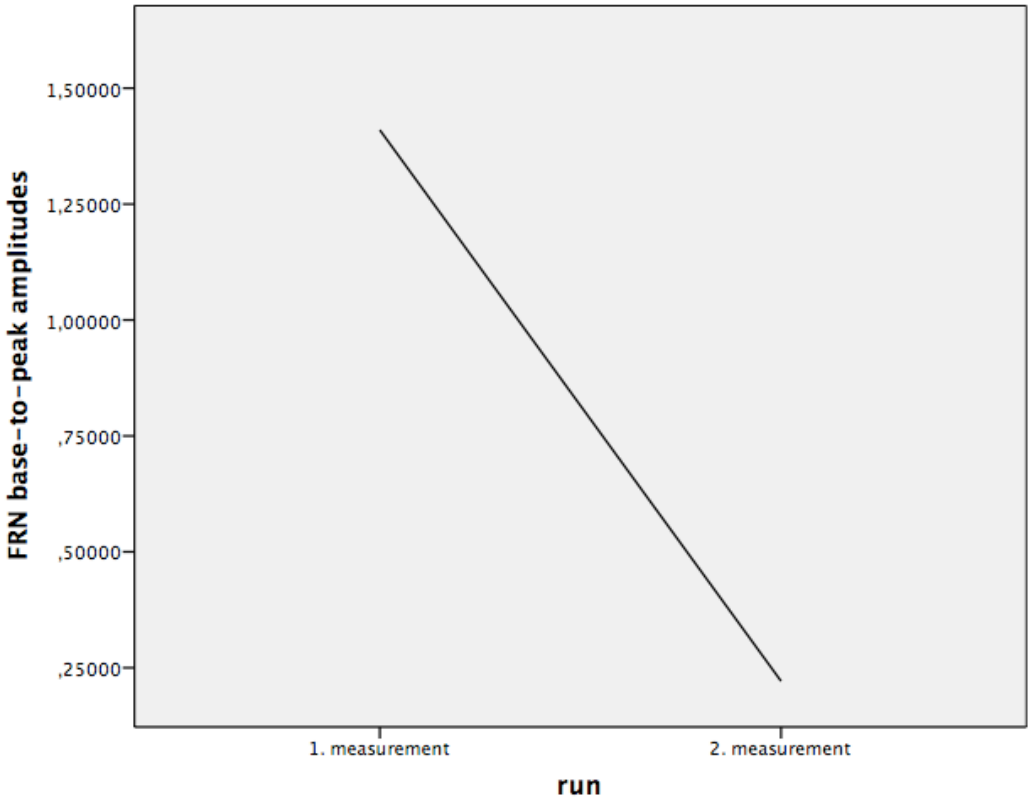


Figure 2: Main effect of run in mean amplitudes over both groups.

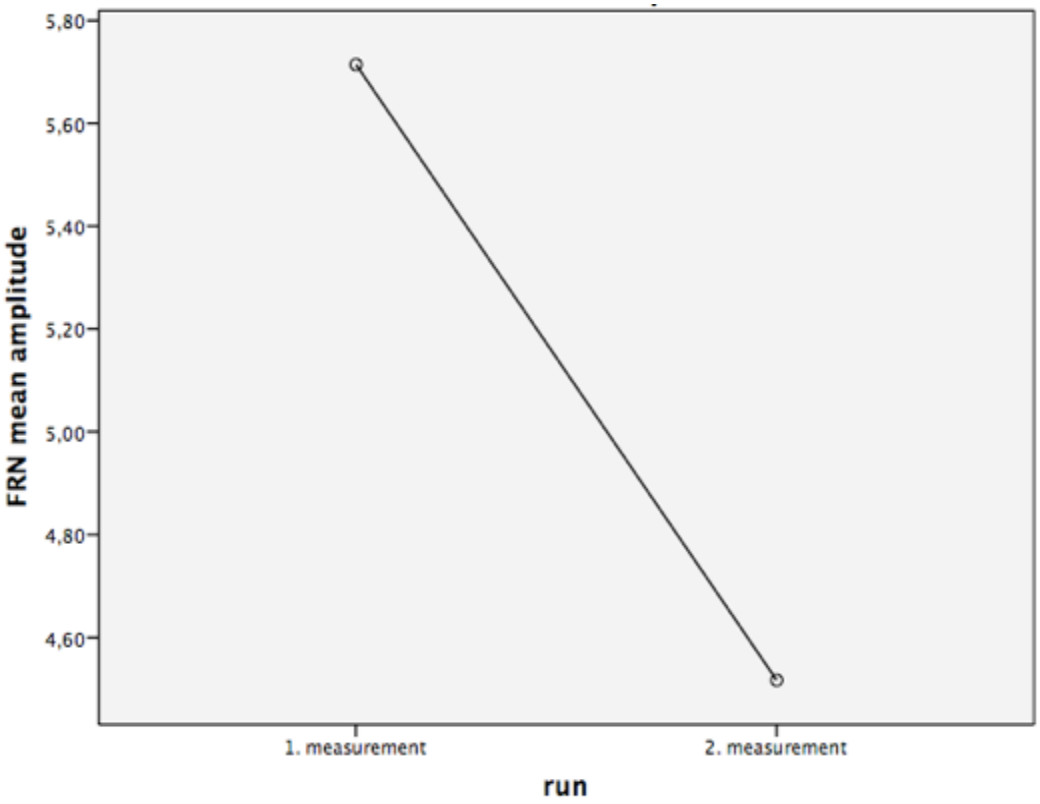


Figure 3: FRN base-to-peak interaction of incentive cue and feedback over both groups (healthy control and remitted depressive patients)

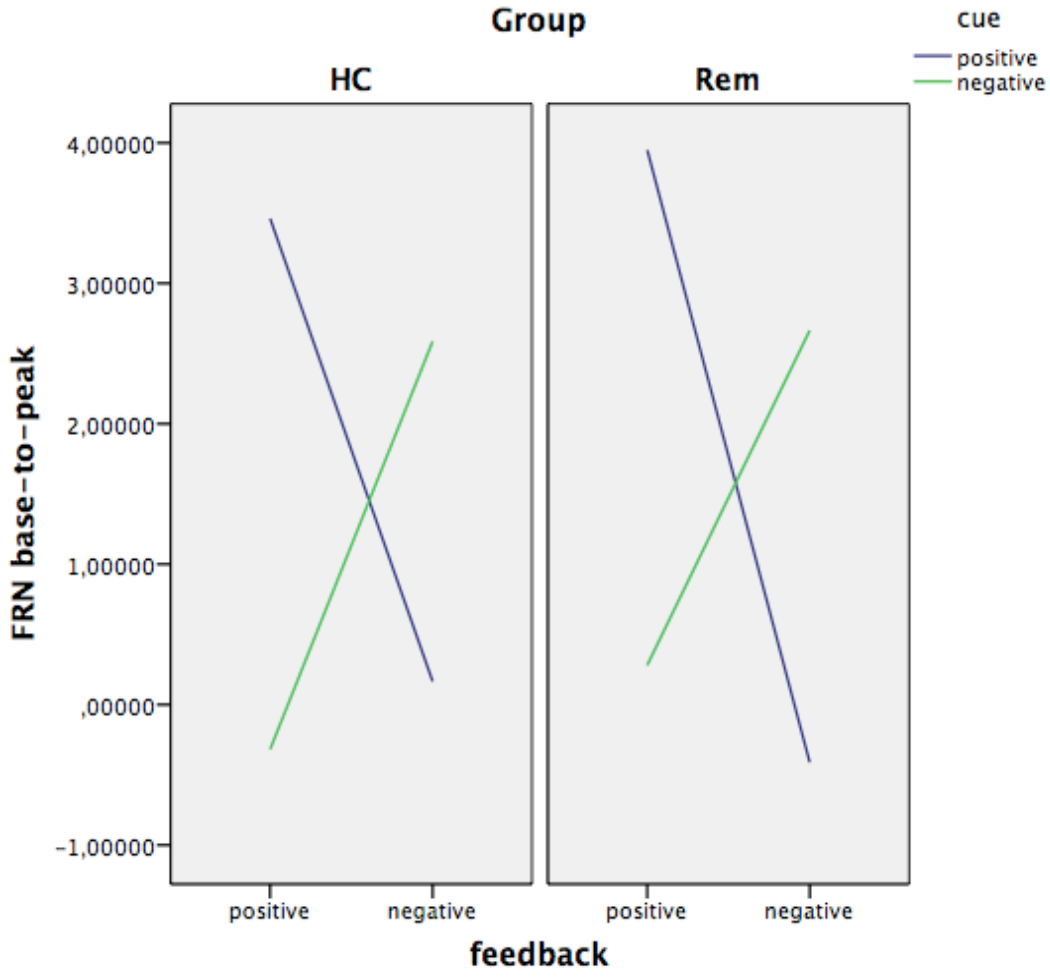


Figure 4: FRN base-to-peak amplitude interaction of group and feedback over both measurements.

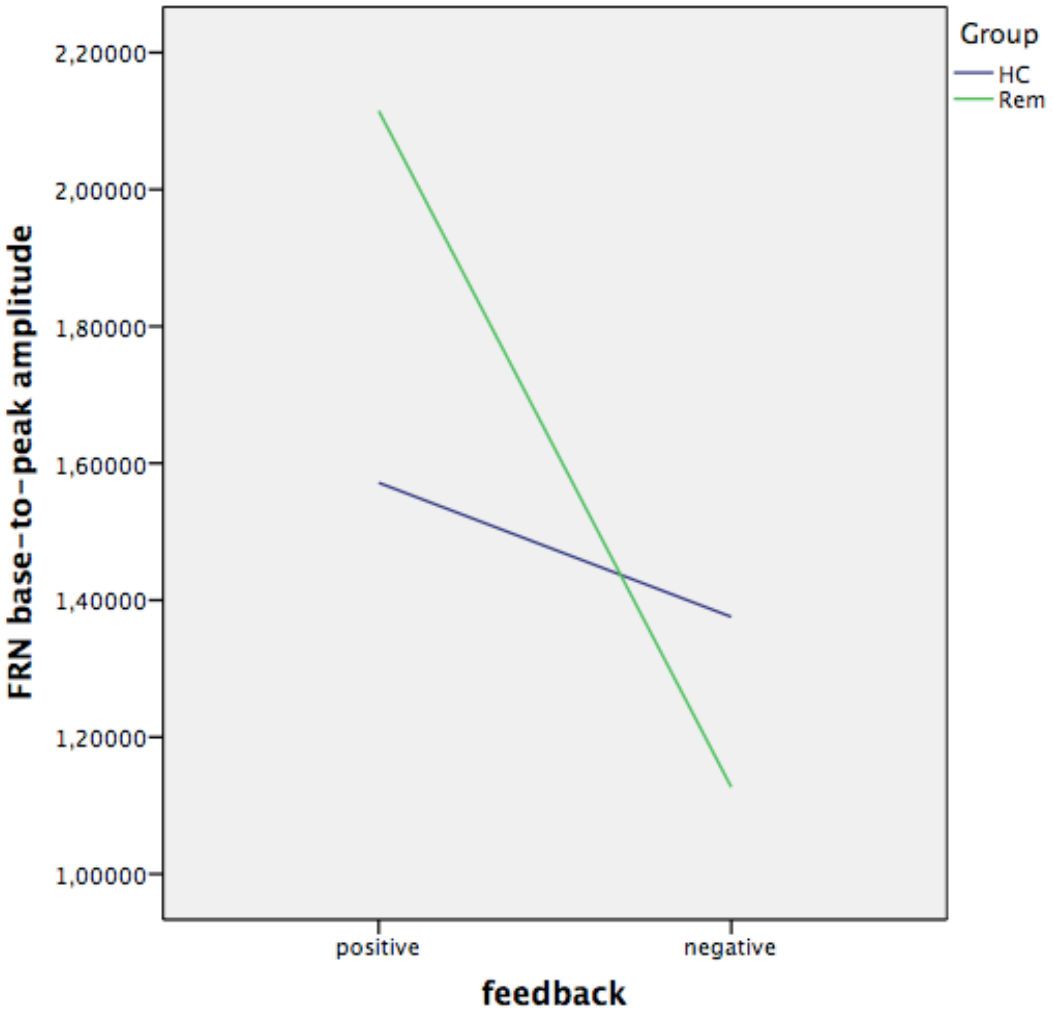


Figure 5: P300 amplitude interaction of cue and feedback over both measurements.

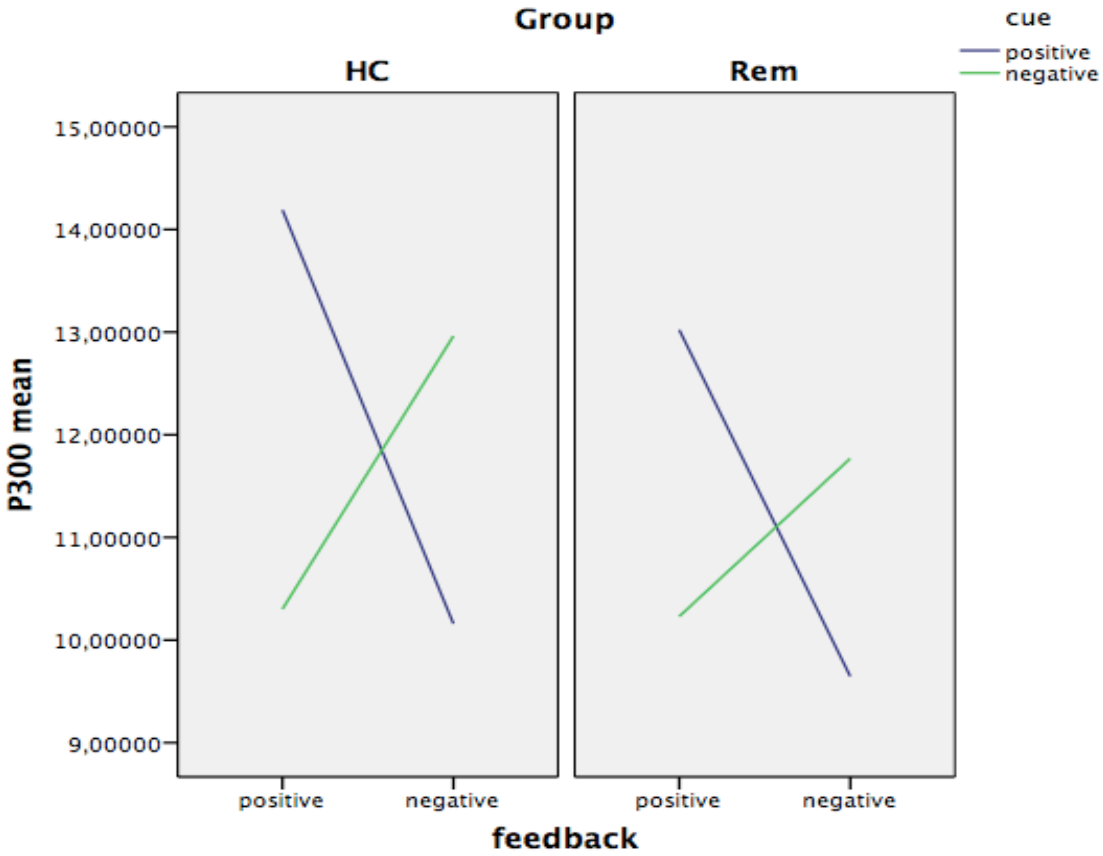


Figure 6: Amplitudes of the four conditions (excluding the neutral condition) for healthy controls over both measurements

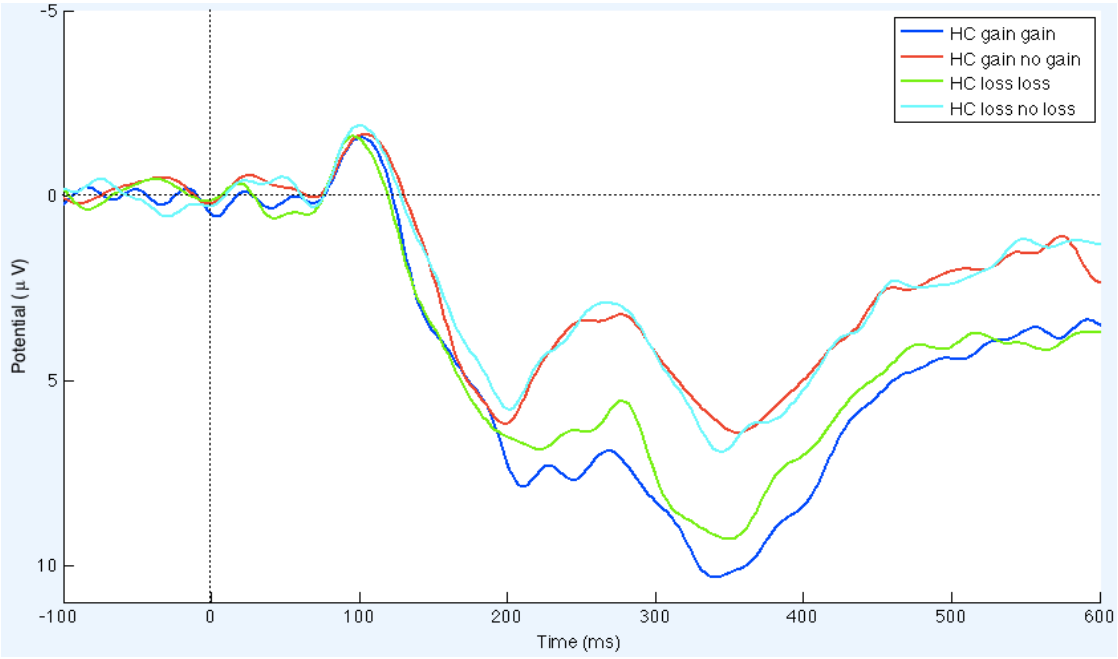


Figure 7: Amplitudes of the four conditions (excluding the neutral condition) for remitted depressive patients over both measurements

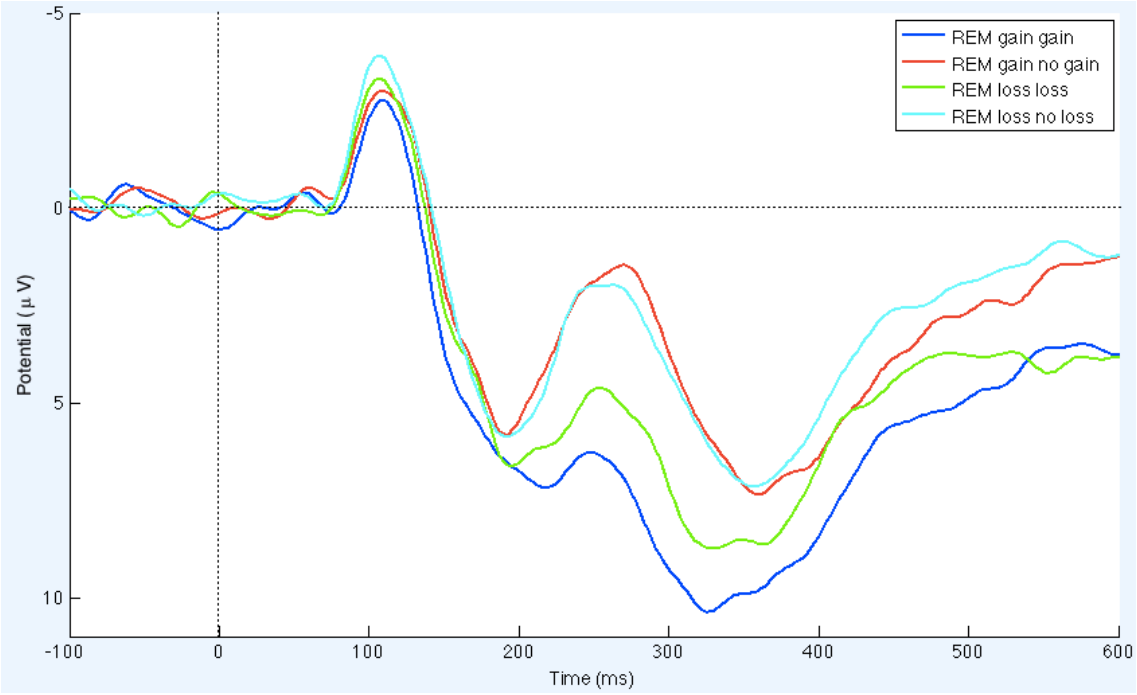


Figure 8: Amplitudes of positive (gain, no loss) and negative (no gain, loss) at FCz for remitted depressive patients

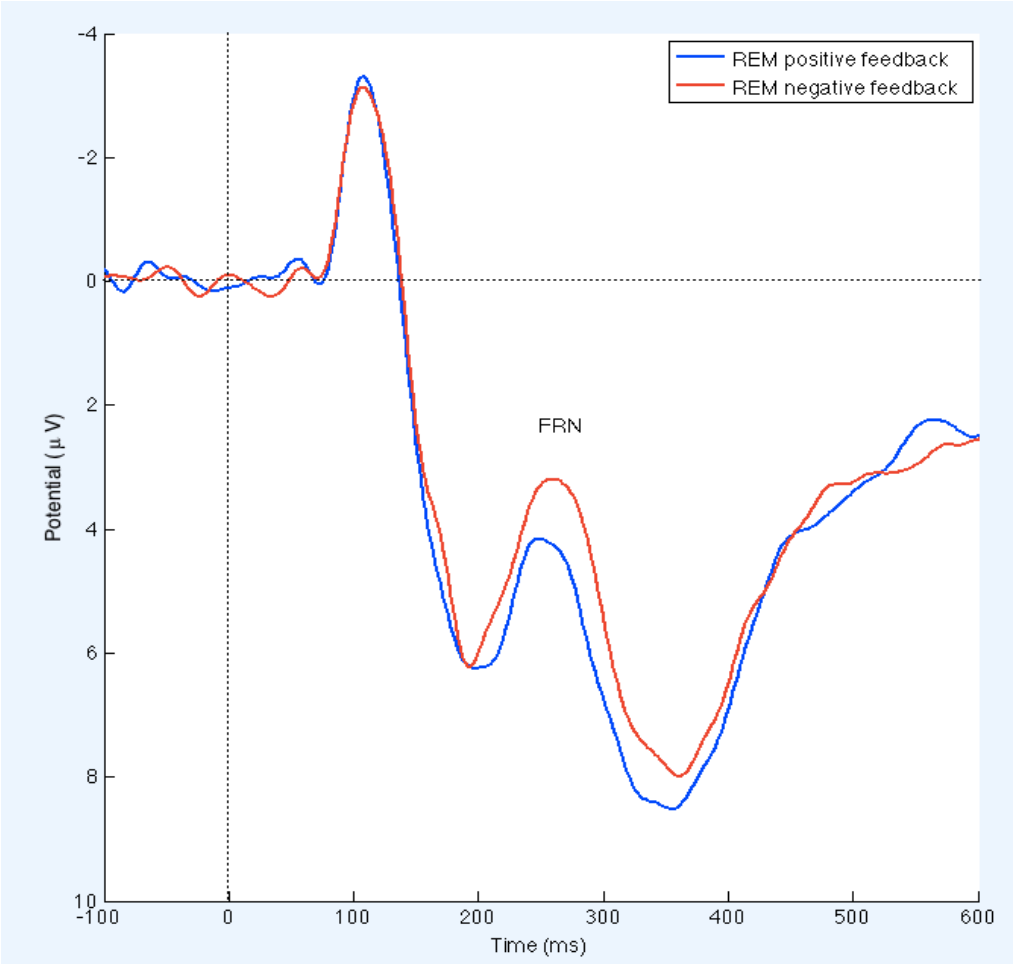
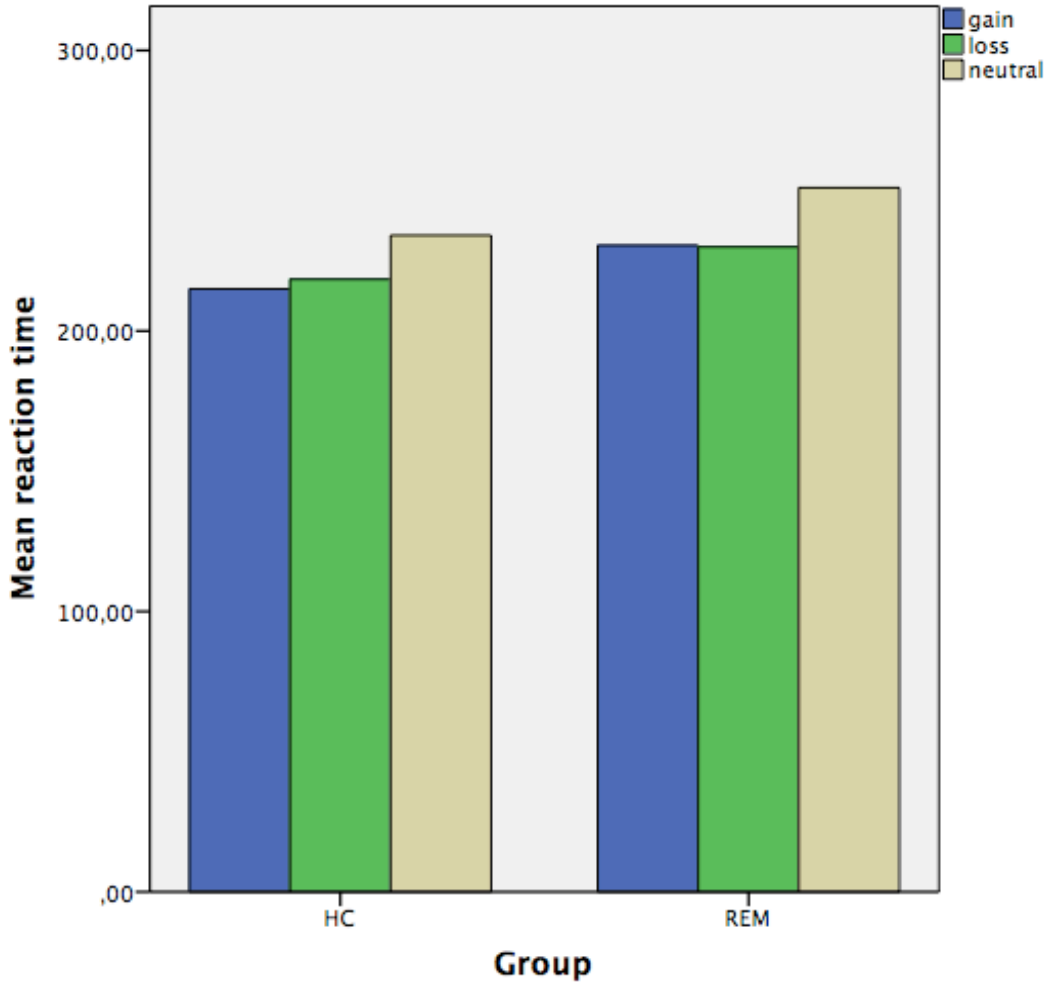


Figure 9: Mean reaction time for remitted depressive patients and healthy controls for the three incentive cues (gain, loss, neutral) over both runs.



German Abstract - Zusammenfassung

Die beiden Komponenten Feedback-related Negativity (FRN) und P300 werden in der Forschung eng mit Depression in Verbindung gebracht. Die Fehlfunktion des Belohnungszentrums bei Patienten mit Depression führt zu einer Abschwächung der Positivierung, die bei gesunden Kontrollprobanden durch Belohnung eintritt. Diese Fehlfunktion wird in einer größeren FRN Amplitude deutlich und stellt damit die Möglichkeit dar, FRN Amplituden als biologische Marker für Depression anzuwenden. Die Stabilität und Zuverlässigkeit dieser Komponente soll mittels der Test-Retest Analyse ermittelt werden.

In der vorliegenden Studie wurde die Test-Retest Reliabilität, der FRN Komponente am FCz und der P300 Komponente am Pz, zwischen zwei Testzeitpunkten mittels einer Reaktionsaufgabe (*Monetary Incentive Delay Task*) an 40 Versuchspersonen (20 Patienten mit remittierter Depression, 20 vergleichbare gesunde Kontrollprobanden) untersucht. Es zeigte sich eine mittlere Reliabiliät (zwischen r=.41 bis r=.64) in beiden Gruppen in sogenannten *mean-* und *base-to-peak* Amplituden. Die Varianzanalyse mit Messwiederholung zeigte Haupteffekte des Messzeitpunktes, welcher die Reliabilität verringerte. Interaktionen zwischen Cue x Feedback sowie zwischen Gruppe x Feedback wurden gefunden. Von besonderem Interesse war, dass Patienten mit remittierter Depression größere FRN Amplituden bei negativem Feedback als bei positivem Feedback zeigten. Diese Ergebnisse bestärken die Vermutung des negativen Fokus, welchen Patienten mit Depression auf negative Informationen und Cues legen.

Curriculum Vitae

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Certificates	Certificate of Advanced English (C1- ESOL) OCR Certificate of Adult Literacy
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Poster Listings OHBM 2015

Lucia Navarro de Lara, Martin Tik, Michael Woletz, Henryk Bukowski, Inga-Lisa Stuerkat, Claus Lamm, Ewald Moser, Christian Windischberger. *Boosting the sensitivity of concurrent TMS/fMRI in motor cortex using a dedicated MR coil array*. Poster session will be presented at the meeting of the OHBM 2015, Honolulu, Hawaii.

Ronald Sladky, Andreas Hahn, Inga-Lisa Stürkat, André Hoffmann, Georg S. Kranz, Martin Tik, Rene Seiger, Daniela Pfabigan, Claus Lamm, Rupert Lanzenberger, Christian Windischberger. *Effective connectivity modeling of large-scale emotion processing networks using 7T fMRI.* Poster session will be presented at the meeting of the OHBM 2015, Honolulu, Hawaii.

28th ECNP Congress 2015

Kraus, C., Stürkat, I.-L., Sladky, R., Hahn, A., Pfabigan, D., Tik, M., Köck, P., Windischberger, C., Lamm, C., Lanzenberger, R. (21015). Altered structural plasticity in acute and remitted depressive patients investigated with ultra-high field magnetic resonance imaging. Abstract will be presented at the ECNP Congress 2015, Amsterdam, Netherlands.