FRUSTRATING FEEDBACK EFFECTS ON ERROR-RELATED NEGATIVITY (ERN) AS A DIMENSIONAL MARKER OF NEGATIVE AFFECT

Ana Maria Frota Lisbôa Pereira de Souza, Roberto Guedes de Nonohay, Gustavo Gauer

Abstract

Objective: The Error-Related Negativity (ERN) is an event-related potential component characterised by a negative-going deflection in predominantly midline frontal and central scalp sites, appearing between 50-100 ms after the commission of an error. Heightened ERN is a potential physiological marker of an endophenotype encompassing several anxiety- and affect-related mental symptoms. In the Research Domain Criteria (RDoC) dimensional matrix, such marker crosses three domains: sustained threat, performance monitoring and reward learning. In this exploratory study, we aimed to evaluate if ERN would interrelate with a manipulation of negative affect (frustration through negative feedback).

Method: Ten adults with heterogeneous symptoms from a community sample responded to a modified Flanker task. The task comprised 7 blocks of 60 trials, and subjects received increasingly aversive negative feedback at the end of blocks 4, 5, and 6.

Results: Our findings suggest that the number of errors increased after the introduction of negative feedback, and reaction times and ERN amplitudes diminished, possibly mediated by scores on the negative affect scale.

Conclusions: This might suggest that underlying mechanisms, such as worry and rumination, may play a role in different negative affect disorders.

Key words: error-related negativity (ERN), Flanker task, negative affect, Research Domain Criteria (RDoC)

Declaration of interest: none

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Since Kraepelin's (1896/1987) attempts to categorise mental disorders, psychiatry has found difficulties in associating the traditional model of diagnoses based on clinical observation and clustering of symptoms, with the discoveries of clinical research and clinical neuroscience (Insel et al. 2010, Sanislow et al. 2010). Categorical diagnoses based on signs and symptoms have often failed to cohere with the neural mechanisms that underlie mental disorders and might be slowing the development of new, more efficient treatments. To address this issue among others, the National Institute of Mental Health (NIMH) has launched, in 2008, the Research Domain Criteria (RDoC) project. It aims to "incorporate data on pathophysiology in ways that will eventually help to identify targets for treatment development, detect subgroups for treatment selection, and provide a better match between research finding and clinical decision making" (Insel et al. 2010, p. 748).

The RDoC premise is that mental disorders are disorders of brain circuits and that their neural underpinnings can be identifiable through neuroscience tools, such as brain imaging, electroencephalography and in vivo connections (Insel et al. 2010). The RDoC framework is composed of a matrix, divided in five domains (i.e. negative valence systems, positive valence systems, cognitive systems, social processes and arousal and regulatory systems) and eight units of analysis (i.e. genes, molecules, cells, circuits, physiology, behaviour, self-report and paradigms). Thus, research according to RDoC approach is encouraged to study mechanisms in a transdiagnostic way, in order to elucidate the process underlying the disorders (Sanislow et al. 2010). From this perspective, research focusing on brain mechanisms and neural responses fits well within the RDoC matrix (Weinberg et al. 2015).

Error-Related Negativity and the RDoC approach

The ability to detect errors and adapt behaviour is essential in a changing environment (Meyer et al. 2012, Riesel et al. 2013, Weinberg et al. 2015). The Error-

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Related Negativity (ERN) is a fronto-centrally maximal negative deflection in Event-Related Potentials (ERP) that reaches maximum amplitude between 50ms and 100ms after the commission of an error. It occurs in simple reaction time tasks, even in the absence of conscious awareness (Meyer et al. 2012, Hanna and Gehring 2016, Weinberg et al. 2015, 2016). The ERN was first discovered in the 1990s by two independent teams. In Germany, Falkenstein (1991) called it Error Negativity (Ne) and in the United States it was called by Gehring (1993) the Error-Related Negativity (Weinberg et al. 2015). From its discovery until this day, the ERN remains the most widely investigated electrophysiological cortical index of cognitive error processing (Wessel 2012).

The Anterior Cingulate Cortex (ACC) appears to be the main generator of ERN, as evidenced by multiple lines of research (Meyer et al. 2012, Hanna and Gehring 2016, Olvet and Hajcak 2008, Weinberg et al. 2015, 2016). That is particularly important in the RDoC framework, since abnormalities in performance monitoring have been implicated in multiple forms of psychopathology (Riesel et al. 2017).

The ERN has been shown to be a valid and reliable tool for measuring traits and a promising biomarker candidate of psychiatric disorders (Kappenman and Luck 2015, Olvet and Hajcak 2008). It has been documented across different levels of task difficulty and modalities, across the life-span, in children as young as 5 years old and in adults as old as 80 years old, and in most psychiatric disorders (Hanna and Gehring 2016; Weinberg et al. 2015). Empirical findings consistently suggest that enhanced ERN amplitudes are associated with anxiety and some inconclusive research point to depression as a diagnosis marked by enhanced error-monitoring, indicating that high amplitudes on ERN might reflect an endophenotype of negative affect disorders (Weinberg et al. 2015). In the RDoC framework, though, the ERN appears as a physiological unit of analysis in three domains (Cognitive Control – Performance Monitoring; Negative Valence Systems – Sustained Threat; and Positive Valence Systems – Reward Learning) indicating that the ERN reflects variance in each of these domains and functionally integrates both cognitive and motivational factors (Hanna and Gehring 2016).

The definition provided by the working group convened to establish the Cognitive Control construct is: "Cognitive control involves multiple subcomponent processes, including the ability to select, maintain, and update goal representations and performance monitoring and other forms of adaptive regulation". In this sense, the ERN, as a measure of error monitoring, is deeply integrated in this construct, since in order to regulate behaviour to achieve goals it is necessary firstly to recognise what did not go right (Weinberg et al. 2015). However, the inclusion of ERN in this construct is still a matter of debate, as different theories contradict it (i.e. the mismatch theory, the conflict theory, the reinforcement learning theory, among others). In addition, one could argue that cognitive control cannot be separated from affect, which makes it difficult to solely attribute the magnitudes of ERN to cognition (Weinberg et al. 2015). Another construct related to the ERN is the Reward Learning, more thoroughly discussed below. Of particular interest to this article, though, is the construct "sustained threat" in the negative valence systems domain.

Negative Valence Systems – Sustained Threat

The definition provided by the RDoC working group establishes Negative Valence Systems as "An aversive emotional state caused by prolonged (i.e., weeks to months) exposure to internal and/or external condition(s), state(s), or stimuli that are adaptive to escape or avoid. The exposure may be actual or anticipated; the changes in affect, cognition, physiology, and behaviour caused by sustained threat persist in the absence of the threat, and can be differentiated from those changes evoked by acute threat".

Negative Affect and ERN

Negative affect can be described as a tendency to experience distress (Luu et al. 2000) in multiple forms, such as anxiety, fear, sadness and anger. Thus, psychiatric disorders as anxiety, depression and irritability are incorporated in this affective cluster.

Nevertheless, the relationship between negative affect disorders and ERN remains unclear. Hajcak, McDonald and Simons (2004) argue that abnormal ERN amplitude may not be caused by specific pathological disorders as anxiety and depression, for instance, but rather reflect an underlying mechanism that could be understood as negative affect. Contradicting their hypothesis, Weinberg et al (2015) present several studies emphasizing the complicated performance monitoring measures in individuals with depression, often marked by blunted ERN amplitudes, reflecting a prevalence of blunted dopaminergic system. Therefore, ERN would be modified by the evaluation the individual makes of mistakes and the sensitivity they experience to punishment and rewards (Weinberg et al. 2015). Another disorder characterised by negative affect is irritability, as expressed in some depressive conditions, bipolar disorder, intermittent explosive disorder and conduct disorders, among others. A study conducted by Lievaart et al (2016) has evaluated ERN amplitudes in individuals with high and low trait anger, finding no significant differences in ERN amplitudes between groups, concluding that individuals high in trait anger are not sensitive to mistakes.

The relationship between those disorders and ERN, therefore, seems to be less related to a common cluster of "negative affect", but more associated to underlying mechanisms as worry/apprehension, in the case of anxiety (Moser et al. 2013), blunted dopaminergic system in the case of depression (Weinberg et al. 2015) and poor inhibitory control in the case of irritability (Lievaart et al. 2016). Thus, instead of being characterized as part of a construct named negative affect, ERN fits well within the RDoC matrix as a transdiagnostic biomarker of abnormal underlying mechanisms.

Negative Feedback and Error-Related Negativity

Committing errors can be dangerous and, in almost every case, mistakes require attention and corrective action (Weinberg et al. 2015). Errors can be associated to exogenous threats (i.e. physical danger) or endogenous threats (i.e. not related to an environment threat, such as academic performance or feedback). In their article, Weinberg et al (2016) discuss the inclusion of the ERN as part of the sustained threat domain and propose that enhanced ERN amplitudes are only caused by endogenous threats. The authors argue that rather than reflecting the degree of instantiated cognitive control,

the magnitude of the ERN varies according to within- and between-subject variables that impact the evaluation of errors. The ERN would reflect, then, the degree to which errors are considered threatening (Weinberg et al. 2016). According to their hypothesis, that could explain why anxiety disorders are more susceptible to enhanced ERN.

Individuals require frequent feedback from the environment in order to adapt behaviour to succeed in their actions. Relevant feedback, in those cases, must comprise both valence (i.e. a good or bad outcome) and magnitude (how good and how bad was the performance/outcome) (Hajcak et al. 2006). Feedback can also state whether a goal was reached or not and is highly associated with expectancy. Unexpected negative events produce more enhanced brain waves than expected ones (Holroyd et al. 2006).

Studies have been conducted manipulating the magnitude of gain and loss of participants on tasks and results suggest that the brain treats neutral and negative feedback stimuli in much the same way (i.e. the brain considers two possible outcomes: gaining or not gaining, which indicates that neutral feedback is negative) (Hajcak et al. 2006, Holroyd et al. 2006).

That evaluative system of the brain reflects a component of a neural mechanism that underlies how humans learn to pursue reward and avoid punishment (Holroyd et al. 2006) and is intimately related to reward learning. Reward learning is defined by RDoC's cognitive control research working group as: "A process by which organisms acquire information about stimuli, actions, and contexts that predict positive outcomes, and by which behaviour is modified when a novel reward occurs, or outcomes are better than expected. Reward learning is a type of reinforcement learning, and similar processes may be involved in learning related to negative reinforcement".

Ford and colleagues (2010, 2012) demonstrate that individuals with higher irritability traits are attentionally biased towards prizes and rewards, and Wilkowski and Robinson (2008) suggest that angry individuals are more intolerant to frustration. Moser et al (2013) propose that anxious individuals are more sensitive to punishment and Weinberg et al (2015) demonstrate that subjects with depression are characterised by insensitivity to rewards. With this evidence, it can be discussed if reward learning would be different from reduced sensitivity to threat, again returning to the point of the influence of affect in the ERN (Weinberg et al. 2015).

Regarding those differences, it is possible to infer that the ERN is related to expectancy and frustration. Individuals, regardless of the diagnoses of anxiety, irritability or depression, perform actions in expectancy of positive outcomes. When those outcomes are not achieved, frustration is experienced in the form of anger (irritability disorders), insensitivity to reward (depression) or sensitivity to punishment (anxiety). Several studies have suggested that inhibitory control tasks may lead to frustration and activate the same portion of the ACC that shows sensibility to errors (Saunders et al. 2015), indicating that neural monitoring would be experienced in much the same way as frustration (Spunt et al. 2012). One study has tried to address the effects of frustration and the evaluation of errors in the cortex during a Go/No-Go task. It has found that, similarly to situations of disengagement of tasks due to mind wandering, frustration may lead to a decrease in cognitive and affective sensibility to tasks (Randles et al. 2016). Another study, however, has found an increase in ERN amplitudes following negative feedback, but this result was independent of self-report measures of frustration during the performance of a Go/No-Go task. Frustration, on the other hand, has predicted decreased response caution and, therefore, a higher number of errors (Saunders et al. 2015). Due to these aspects, tasks that introduce neutral and negative feedback, unrelated to the participant's actual performance, may mislead the reward learning system, consisting of an interesting measure of the effect of negative feedback to study negative affect.

Goals

The main goal of this exploratory study was to investigate the relationship between error-related negativity, negative task feedback, and negative affect in adults from a community sample. Specific goals were to compare ERN amplitudes before and after the introduction of negative performance feedback, relating it to behavioural (accuracy and reaction time) measures and to scores on self-report questionnaires measuring mood, humour and affect.

Hypothesis

Due to the exploratory characteristic of the study and the contradictory findings of research on Error-Related Negativity and negative affect, it was expected that the ERN would either enhance or diminish after negative feedback was provided. This is justified by the different variables that play a role in negative affect, such as worry, rumination, frustration to non-reward, punishment and individual characteristics. If frustration leads to anxiety, it was expected that ERN would enhance. However, if individuals experienced sadness caused by frustration, a decrease on the amplitudes of ERN was more plausible. In this scenario, the goal was to observe which of these variables would greatly influence ERN and determine the direction of it.

Method

Participants

Ten adults (6 female), from a community dataset from Hospital de Clínicas de Porto Alegre (HCPA) in Porto Alegre, Brazil, participated in this study. This sample size is justified by a convenience factor, given that was the number of participants available at the time of data collection. Participants were the parents of children previously evaluated in another research project and had no relationship to the Hospital. Subjects were contacted by telephone and invited to come to the hospital to be part of the experiment. The study was previously approved by the ethics committee of the Hospital. All participants received verbal and written information about the aims and procedures of the study and written consent was obtained. All participants had normal or corrected-to-normal vision and reported no history of head trauma or neurological disease. No participant had used alcohol, any kind of drug or ingested caffeine 4 hours before the experiment, as requested by the researcher when scheduling the interview and as measured by a questionnaire. All participants were right-handed. The mean age was 37.80 years (SD =12.14). 70.0 % of the sample was Caucasian, 10% was Black, 10% was Asian and 10% was mixed race white and black African. A Socio-Demographic and General Health Questionnaire was filled by all participants. Only one participant was medicated with psychotropics (fluoxetine).

Instruments and Materials

Socio-Demographic and General Health Questionnaire. This instrument was developed for this study and evaluates data such as age, gender, educational levels and general health conditions that might affect the participant's performance.

Positive and Negative Affective Scale - PANAS (Watson et al. 1988). Scale translated and adapted to Brazil (Zanon et al. Hutz, 2013). It is a paper self-report scale, composed by 20 items, that measures state positive and negative affect (i.e., in the past days). It presents a likert scale of 5 points, being 1= not at all and 5= extremely. The scale presents $\alpha = 0.83$ for positive affects and $\alpha = 0.77$ for negative affects.

Anxious Thoughts Inventory (Wells 1994). Scale translated and adapted to Brazil (Moreno et al. 2015). The scale is composed by 22 items that are evaluated through a likert scale of 4 points, being 1= almost never and 4= almost always. This instrument evaluates frequency of anxious thoughts, such as rumination and worry, in the present time. Internal consistency of this instrument is 0.86.

Anger Rumination Scale (Sukhodolsky et al. 2001). Scale translated and adapted to Brazil (Sperotto et al., submitted for publication). This instrument consists of 19 items that measure anger rumination over the past six months. It is composed by a 4 points likert scale, being 1= almost never and 4= almost always.

Task and Procedure

The experiment consisted of a modified Flanker Task, which was originally developed by Eriksen and Eriksen (1974), to evaluate the effect of noise in an experimental task of inhibitory control. The task was programmed in xml and run in the Psytask software. On each trial of the Flanker task, five horizontally aligned black arrowheads were presented and participants were instructed to respond with the left or right mouse button in accordance with the direction of the central arrowhead. The responses had to be performed with the right hand. The task consisted of 420 trials, divided in seven blocks of 60 trials, with a one-minute interval between blocks. Participants sat at a viewing distance of approximately 70cm from a 19-inch computer monitor. Set up included one personal computer and a split screen for presenting stimuli. All participants received written instructions on the computer and were instructed to respond as fast and as accurate as possible. If no answer was given during the intertrial interval (ITI), the next trial started and the previous one was computed as missing and, therefore, an error. No feedback was given between trials, only at the end of the blocks. The trials were randomised and comprised the four congruent X incongruent possibilities (all arrowheads to the right; all arrowheads to the left; distracting arrowheads to the right and central one to the left; and distracting arrowheads to the left and central one to the right). Each set of arrowheads was presented for 200ms, followed by a randomised ITI varying from 600ms to 1000ms, presented as a blank screen. The total duration of a trial was approximately 1 second, and the whole task had 14 min duration, approximately. Accuracy and reaction-time for all participants were computed. To evaluate the effect of performance-worrying and error-monitoring, a procedure to induce worry was introduced. This procedure consisted of providing feedback about the participant's performance by the end of each block. Feedback was programmed in advance on the computer and was not related to the individual's actual performance but was designed in order to increase worry and frustration. Feedback was displayed on the screen during the one-minute interval. On the first, second and third blocks, feedback was neutral and uninformative of the subject's performance (i.e. "okay until this point"). On the fourth, fifth and sixth blocks, feedback was negative and increasingly aversive (i.e. "your performance is very bad, try to be more accurate"). This procedure has been constantly replicated in the literature (Hajcak et al. 2006, 2012, Holroyd et al. 2006) and presents effectiveness in eliciting higher amplitudes of ERN, calling the individual's attention to the error and being related to RDoC dimensions of performance monitoring and sustained threat.

Data were collected at the hospital, in a room especially designed for EEG experiments. Participants that agreed to participate in the study by phone calls scheduled an appointment with the researcher and were independently evaluated. Exclusion criteria, such as the presence of head trauma, neurological disease, use of antipsychotic drugs and left-hand dominance were questioned on the phone calls. Participants that met criteria for the study and accepted to participate were then seen by the researcher. Firstly, all participants received and signed the ethical consent. Secondly, participants were instructed about the EEG procedures and an EEG preparation phase was initiated. Once prepared, participants were instructed about the task and began the experiment.

At the end of the experiment, participants were asked to complete the self-report questionnaires. Total duration of the procedures was approximately one hour and a half.

Psychophysiological recording, data reduction and analysis

EEG was recorded from 32 Ag/AgCl scalp electrodes embedded in a Mitsar EEG 202 system and referenced to linked mastoids. Midline electrode locations were Fz, Fcz, Cz, Cpz, Pz, Fpz and Oz, and left and right hemisphere sites were Fp1, Fp2, F3, F4, F7, F8, T3, T4, T5, T6, C3, C4, P3, P4, O1, O2, Ft7, Ft8, Fc3, Fc4, Tp7, Tp8, Cp3 and Cp4. Reference electrodes A1 and A2 were located on left and right mastoids, respectively. Signals were amplified with a bandwidth of 0.1 to 30 Hz using a 32-channel Mitsar 202 EEG system running EEGSTUDIO 1.14 (Mitsar, Saint Petersburg, Russia), acquiring EEG data at a 500 Hz sampling rate. Electrode scalp impedances were kept below 5 kΩ. Acquired data were analysed using EEGLAB (Delorme and Makeig, 2004) and ERPLAB (Lopez-Calderon and Luck 2014), two open source toolboxes for EEG and ERP analysis in MATLAB (MathWorks, Inc, Natick, MA). The continuous EEG data were filtered digitally with a high-pass of 0.1 Hz and a low-pass of 35 Hz. Artifacts were removed using ERPLAB's Moving Window Peak-to-Peak algorithm. ERPs were quantified at electrode FCz where error-related brain activity was maximal. Continuous data were separated into epochs ranging from -200 to 1000ms locked to response onset. ERPs were analysed using Mean Amplitude Peaks in the selected time frame given visual inspection. Trials were eliminated when response times were below 100ms and higher than 700ms after stimulus onset. Responses were separated into correct and incorrect trials. Only incorrect answers were analysed for ERN components. ERNs were calculated as the mean amplitude in the negativities found in FCz. Delta ERNs were calculated as the difference of the area under the curve for correct

and incorrect answers. ERN and delta ERN for group comparison were calculated in a similar fashion.

Results

Results of self-report questionnaires and electrophysiological data are presented below. Analyses were conducted with SPSS version 22 and JASP version 0.8.0.1 statistical software.

Self-Report Questionnaires

The means of the scores of the three questionnaires are presented in **table 1**.

Table 1. *Means of the scores of the self-report questionnaires*

	Anger Rumination Scale TOTAL	Anxious Thoughts Inventory TOTAL	PANAS Negative Items TOTAL
Mean	35.9000	41.4000	19.9000
N	10	10	10
SD	14.37938	12.01111	9.13418

Note: ARI= Anger Rumination Scale; ATI= Anxious Thoughts Inventory; PANAS Neg= PANAS Negative Items.

Due to the overlap in symptoms among participants that scored high in more than one questionnaire, a Bayesian correlation analysis was conducted to measure correlation between scales and is presented in **table 2**. A Bayesian analysis was considered more appropriate considering the small sample, since it provides a clearer estimate of the amount of evidence present in the data (Jarosz and Wiley 2014). The hypothesis tested was that the correlation was positive.

Table 2. *Bayesian Pearson Correlation for self-report questionnaires*

			r	BF_{+0}
ARSTOTAL	-	ATITOTAL	0.826	32.92
ARSTOTAL	-	PANASNEG	0.787	18.40
ATITOTAL	-	PANASNEG	0.848	49.19

Note. For all tests, the alternative hypothesis specifies that the correlation is positive.

In all pairs, Pearson's r is higher than 0.78, indicating a strong correlation between questionnaires. Bayes Factor, on the other hand, is also supporting a positive correlation, as it indicates the number of times the alternative hypothesis predicts data better than the null hypothesis.

Behavioural results

Table 3 depicts a Wilcoxon analysis comparing number of errors and reaction time values in the task for phases 1 and 2. Phase 1 refers to the blocks 2, 3 and 4, and Phase 2 concerns the blocks 5, 6 and 7 (post introduction of negative feedback). Block 1 was excluded from analysis and considered a practice phase.

Table 3. *Wilcoxon Test for Reaction Time (RT) and Errors between phases 1 and 2*

Mean RT phase2 – Mean RT phase1	Errorsphase2 - Errorsphase1
-1.836[a]	-1.897[b]
.066	.058

a. Based on positive ranks.

b. Based on negative ranks.

c. Wilcoxon Signed Ranks Test

As shown in **table 3**, the difference between reaction time in phases 1 and 2 and number of errors was close to reaching statistical significance, indicating a possible difference between both phases concerning those variables, especially the commission of errors ($p = .058$). One participant was excluded from EEG analyses due to the commission of fewer than six errors (Olvet and Hajcak 2009).

ERP results

Figure 1 presents the response-locked ERP waveforms for correct and error responses on FCz. Consistent with previous studies, the ERN was observed as a sharp frontocentral negative deflection that peaked shortly after the commission of an error. Across conditions the difference between negativity following an error and following a correct answer was statistically significant around 16ms until 96ms, as measured by an ANOVA ($F(1.16)=6.6$, $p<0.05$). Comparing phases 1 and 2, visual inspection of the ERN shows it was less

Figure 1. *ERN waveforms from correct vs. incorrect trials*

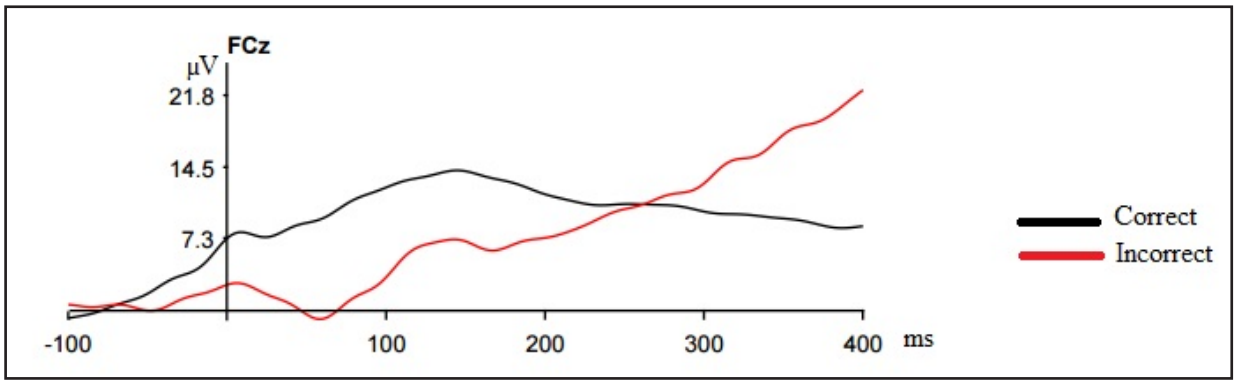
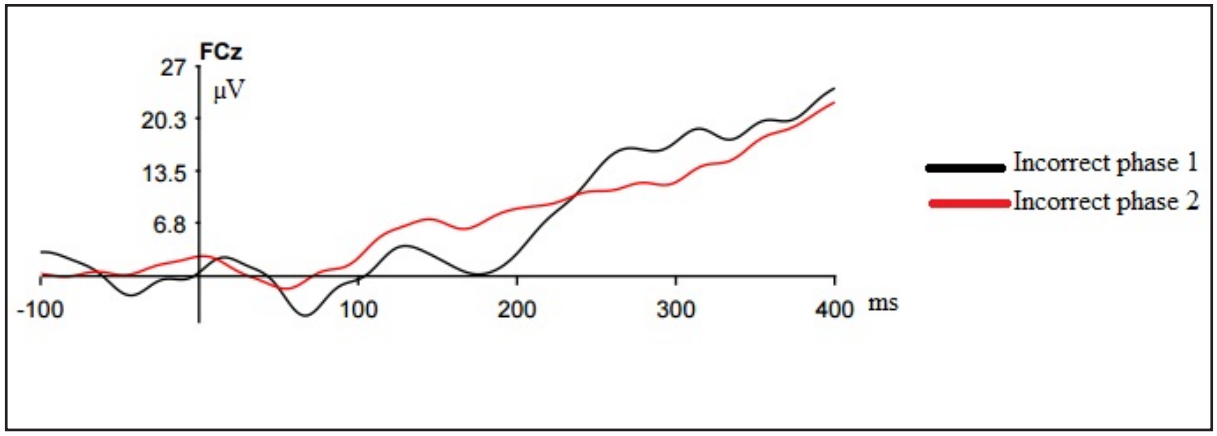


Figure 2. *ERN waveforms from incorrect trials phase 1 vs. incorrect trials phase 2*



enhanced in phase 2, compared to phase 1 around 20ms to 118ms. The difference, however, was not significant ($F(1.16)=0.236$, $p=0.633$), as shown in **figure 2**. ΔERN around 20ms until 124ms was not statistically significant between phases 1 and 2 ($F(1.16)=2.678$, $p=0.121$).

Finally, we performed Bayesian linear regression analyses with the clinical measures as predictors of ERP components and as dependent variables, as seen in **tables 4** and **5**.

The results indicate that scores on the Anxious Thoughts Inventory (ATI) (BF10=5.25) explain moderate variance in ΔERN1. A BF10 between three (3) and ten (10) is considered moderate evidence in favour of the alternative hypothesis, as discussed by Schönbrodt and Wagenmakers (2017). Anecdotal evidence in favour of the alternative hypothesis and presented by scores on PANAS Negative Items + ΔERN1 explained ΔERN2.

Discussion

In this paper we have examined performance monitoring of individuals from a community sample, with angry rumination, negative affect and anxious thoughts traits. We aimed to explore the effects of the presence of one of those symptoms/traits, combined to negative feedback in a Flanker task on amplitudes of ERN, number of errors and reaction times post-feedback, since the literature of anxiety, depression, anger and negative affect is contradictory (Meyer et al. 2012, Moser et al. 2013, Riesel et al. 2017).

Interestingly, the comparison between phases 1 (blocks 2,3,4) and 2 (blocks 5,6,7, after the introduction of negative feedback) suggested less enhanced amplitudes of ERN (i.e. less negativity), faster reaction-times and increase of number of errors in phase 2, contradicting the findings on anxiety (Moser et al. 2013). Negative affect, especially anxiety, have been exhaustively studied for performance monitoring, and shows a pattern of enhanced ERN in basal state and post-feedback (Moser et al. 2013). Inducing negative affect and negative state has also been studied, but its relationship with enhanced ERN remains unclear.

To answer this question, Clayson, Clawson and Larson (2012) divided subjects in two groups that performed a Flanker task. One group received positive and the other received derogatory feedback. Their findings showed that there was no difference between groups for reaction-time or ERN amplitudes, suggesting that a state of negative affect is not related to enhanced

Table 4. *Bayesian Linear Regression for ΔERN1*

Models	P(M)	P(M|data)	BF_M	BF_{10}	% error
Null model	0.063	0.024	0.368	1.000	
ARS	0.063	0.075	1.214	3.125	1.677e-4
ATI	0.063	0.126	2.158	5.251	0.003
ARS + ATI	0.063	0.055	0.881	2.315	0.001
panasneg	0.063	0.030	0.470	1.269	0.004
ARS + panasneg	0.063	0.038	0.594	1.589	0.004
ATI + panasneg	0.063	0.056	0.895	2.351	0.001
ARS + ATI + panasneg	0.063	0.035	0.540	1.450	0.005
ΔERN2	0.063	0.040	0.621	1.659	0.003
ARS + ΔERN2	0.063	0.118	2.012	4.938	0.004
ATI + ΔERN2	0.063	0.116	1.966	4.837	0.004
ARS + ATI + ΔERN2	0.063	0.062	0.989	2.582	9.694e-5
panasneg + ΔERN2	0.063	0.084	1.383	3.523	0.001
ARS + panasneg + ΔERN2	0.063	0.054	0.852	2.245	0.001
ATI + panasneg + ΔERN2	0.063	0.054	0.864	2.273	9.364e-4
ARS + ATI + panasneg + ΔERN2	0.063	0.032	0.497	1.339	0.006

Note: ARS= Angry Rumination Scale; ATI= Anxious Thoughts Inventory; panasneg = PANAS Negative Items.

Table 5. *Bayesian Linear Regression for ΔERN2*

Models	P(M)	P(M|data)	BF_M	BF_{10}	% error
Null model	0.063	0.070	1.121	1.000	
ARS	0.063	0.044	0.694	0.636	0.002
ATI	0.063	0.055	0.873	0.791	0.003
ARS + ATI	0.063	0.036	0.559	0.517	0.002
panasneg	0.063	0.037	0.574	0.530	0.001
ARS + panasneg	0.063	0.038	0.588	0.543	0.001
ATI + panasneg	0.063	0.057	0.907	0.820	0.002
ARS + ATI + panasneg	0.063	0.038	0.593	0.547	0.002
ΔERN1	0.063	0.115	1.956	1.659	0.003
ARS + ΔERN1	0.063	0.091	1.495	1.304	0.004
ATI + ΔERN1	0.063	0.072	1.161	1.033	0.003
ARS + ATI + dΔERN1	0.063	0.054	0.855	0.776	0.001
panasneg + ΔERN1	0.063	0.116	1.970	1.670	0.004
ARS + panasneg + ΔERN1	0.063	0.066	1.058	0.948	0.003
ATI + panasneg + ΔERN1	0.063	0.068	1.099	0.982	0.003
ARS + ATI + panasneg + ΔERN1	0.063	0.044	0.686	0.629	0.001

Note: ARS= Angry Rumination Scale; ATI= Anxious Thoughts Inventory; panasneg = PANAS Negative Items.

ERN. On the contrary, ERN seems to be more related to traits than states, indicating its suitability as an endophenotype (Kappenman and Luck 2016, Olvet and Hajcak 2008).

The significant ERN amplitudes on phase 1 support the literature and can be explained by scores on the Anxious Thoughts Inventory (ATI), as observed in the Bayesian Linear Regression Model. Moser et al. (2013) propose that, rather than being associated to anxiety, increased ERN amplitudes relate to the constructs of worry and apprehension, the same constructs evaluated by ATI. Though worry and apprehension are often seen as sub-components of anxiety, the RDoC framework makes it possible to differentiate those constructs. Moser et al (2013) propose that enhanced ERN is related to anxiety, that would be composed by worry and verbal rumination. However, Tanovic, Hajcak and Sanislow (2017) suggest a different interpretation, in which worry would likely increase ERN amplitudes due to the compensatory error monitoring framework (Moser et al. 2013), since individuals would require more cognitive effort to avoid future mistakes. Rumination, on the other hand, concerns past events and requires no compensations as the individuals disengage from the task to engage in the negative thoughts prompted by the error (Tanovic et al. 2017). We hypothesize, then, that frustration would likely play the same role as rumination on ERN due to the fact that it is characterised by attention to a past event, and not a future one, such as worry. Frustrated individuals might as well disengage from the task after the negative feedback to engage in negative thoughts. This could also elucidate why the scores on the negative items of state PANAS explained the variance in the diminished ERN, the increase in the number of errors and the faster reaction times in phase 2 of our study. Literature shows different ERN amplitudes depending on diverse mechanisms, such as worry (Moser et al. 2013), checking behaviours (Endrass et al. 2014), sustained threat (Ladouceur 2016), dopaminergic system (Weinberg et al. 2015), rumination (Tanovic et al. 2017), among others. Nevertheless, the lack of self-reported measures as frustration, sadness and anxiety, after the task, limits the understanding of our data. We can only hypothesize that negative affect, as measured by PANAS, has played a role in the blunted ERN. However, we have no index of how participants were feeling after the task, which might impact the understanding of our results.

There is also moderate evidence pointing out the Angry Rumination Scale (ARS) as a suitable explanation of the variance in ΔERN1. If we consider this model, it is possible to infer that verbal rumination, as proposed by Moser et al. (2013) is indeed a sub-component of anxiety and leads to enhanced ERN waves. On the other hand, both Bayesian Analyses (ΔERN1 and ΔERN2) present the null model as a candidate to explain the variance, in which case there would be no real difference between ERN amplitudes in both phases. Anecdotal and moderate BF10, as we found, might be indicating misleading differences due to the small sample size. Nonetheless, our data are not strong enough to prove those assumptions.

The lower reaction times can also be explained in terms of habituation and facility of the task, as well as fatigue. It is possible that reaction times were longer in phase 1 due to the novelty of the task, and subjects were requiring more time to process information. In block 4, after 180 trials, subjects may have become habituated to the task and started answering faster.

Another possibility is that the subjects started presenting fatigue, since the task was long and repetitive. The lack of positive outcomes and rewards in the task might have been responsible for the non-activation of the reward learning system (Weinberg et al. 2016), and subjects did not engage in the task. Studies suggest that ERN might reflect error-detection that is utilised for motivational ends (Olvet and Hajcak 2008).

Fatigue might also explain why errors increased in phase 2, since subjects' attention could be impaired by the length of the task (Boksem et al. 2005). Another possibility is that subjects were bored, due to a repetitive task, and have disengaged attention (Eastwood et al. 2012). We hypothesize that, due to the boredom of the task, participants were answering faster in order for it to be over. That could explain the less enhanced ERN (subjects were not monitoring errors), increase in the number of errors (attention disengagement) and faster reaction times. However, fatigue tends to

lead to an increase in reaction time (Matthews et al. 2017), and studies conducted with more trials than ours have not reported fatigue as a condition to be observed. It is, though, not likely that fatigue played a large role in our study. A third option is that the negative feedback provoked negative affect on subjects, especially rumination and depression, culminating in disengagement of the task and in more impulsive and incorrect answers (Tanovic et al. 2017).

Negative affect, defined by a proneness to experience distress, is a broad construct that embraces several mechanisms. In the age of RDoC, concepts of this order are no longer informative and require research on specific underlying mechanisms. In this study, we aimed to explore the effects of negative affect on ERN amplitudes and behavioural results. One could argue that a clinical population would be more appropriate for such studies. However, the RDoC framework proposes that units of analysis are evaluated in a transdiagnostic way, which justifies the choice for a community sample (Sanislow et al. 2010).

The heterogeneity of mental disorders represents difficulties for research, since an unlimited number of variables might be responsible for human behaviour and feelings. Due to these challenges, research focusing on neuroscience tools and techniques is essential to minimise diagnostic errors and to elucidate mechanisms underlying mental disorders (Insel et al. 2010). Endophenotypes, unobservable characteristics that mediate the relationship between genes and a given behavioural phenotype (Olvet and Hajcak 2008), are important tools as biomarkers and can help to assist premature diagnosis and posterior more effective treatment (Sanislow et al. 2010).

Despite its limitations concerning the sample size, that certainly affected our results, this exploratory study proposes tendencies to the literature of negative affect, suggesting that this construct is not homogeneous and possibly not suitable for the study of biomarkers. We have shown by literature review and our data that negative affect, as understood by irritability, anxiety and depression does not present consensus among researchers regarding ERN and performance monitoring. In order to elucidate endophenotypes, it is imperative that the scientific community starts focusing on mechanisms in a transdiagnostic way and abandon broad constructs that are uninformative. The use of the RDoC approach proves itself as a suitable way for the research of units of analysis. Our research is one of the first to try to throw light on a broad construct such as negative affect with this framework and our results suggest that negative affect might not be as clear as previously thought by research into anxiety. The relationship between negative affect and ERN, thus, remains unclear. Future studies should address these questions.

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