# State sadness reduces neural sensitivity to nonrewards versus rewards

Dan Foti and Greg Hajcak

**Both behavioral and neural evidence suggests that depression is associated with reduced sensitivity to rewards. Using the feedback negativity, a neural index of reward processing, an earlier study showed that depressive symptoms experienced over the previous week were associated with less differentiation between nonrewards and rewards in a gambling task. To directly test whether variability in state mood related to similar effects on neural correlates of reward, this study recorded the feedback negativity in individuals assigned to either a neutral or sad mood induction. Following the induction, individuals reporting greater sadness exhibited a reduced feedback negativity. This finding indicates that fluctuation in state negative affect moderates how environmental feedback is processed by reducing neural sensitivity to nonrewards versus rewards. *NeuroReport* 21:143–147 © 2010 Wolters Kluwer Health | Lippincott Williams & Wilkins.**

**NeuroReport** 2010, **21**:143–147

**Keywords: emotion, event-related potential, feedback negativity**

Department of Psychology, Stony Brook University, Stony Brook, New York, USA

Correspondence to Dan Foti, MA, Department of Psychology, Stony Brook University, Stony Brook, NY 11794-2500, USA
Tel: +1 845 234 8426; fax: +1 631 632 7876; e-mail: daniel.foti@stonybrook.edu

**Received** 29 October 2009 **accepted** 15 November 2009

## Introduction

Studies on neural activity elicited by environmental feedback have consistently identified an early response in the event-related potential that differentiates negative and positive outcomes. Termed the feedback negativity, this response is maximal at frontocentral recording sites approximately 300 ms after feedback presentation and is larger following negative outcomes, such as errors and monetary loss [1–7]. Source localization procedures indicate that the feedback negativity is likely generated within the anterior cingulate cortex [2,6], and it has been proposed that the observed response reflects dopaminergic disinhibition of neurons within this region [5]. As such, increases and decreases in the amplitude of the feedback negativity are thought to reflect phasic variation in dopamine signals to the anterior cingulate cortex when outcomes are worse or better than expected, respectively. Consistent with this possibility, a number of studies have showed that the feedback negativity is larger in response to negative feedback that is infrequent [8] or unexpected [5,7,9]. Furthermore, the feedback negativity seems to track the binary, contextual appraisal of feedback as either good or bad, rather than tracking its objective value *per se* [4,10,11].

In addition to providing basic information on how environmental feedback is processed within the brain, the feedback negativity may also be a useful measure for better understanding the individual differences in reward sensitivity. Indeed, in a recent study the feedback negativity recorded during a simple gambling task was used to identify abnormal feedback processing in individuals with current symptoms of depression [12]. In that study, increased scores on a depression measure were associated with reduced differentiation in the feedback negativity between nonrewards and rewards; that is, symptom severity was linked to less neural sensitivity to positive versus negative feedback. This finding complements earlier study showing that major depressive disorder is associated with reduced behavioral responsiveness to rewards [13], as well as patterns of asymmetrical prefrontal activation indicative of a deficit in goal-directed approach motivation [14]. Together, these multiple lines of research indicate that depressive symptomatology is associated with blunted positive affect that reduces early neural reactivity and subsequent behavioral responses to rewarding and nonrewarding stimuli.

A remaining question, however, is the degree to which these motivational, affective, and information-processing abnormalities in depression are driven by trait differences between individuals or state variation in mood. For example, the pattern of abnormal prefrontal activation mentioned above has also been observed in currently healthy individuals with a history of depression compared with individuals with no history of depression [15], suggesting that particular phenomenon is state independent. In contrast, neuroimaging studies have identified increased activity in the anterior cingulate cortex among currently depressed individuals [16] that normalizes following treatment [17]. Insofar as the feedback negativity is thought to be generated within the anterior cingulate cortex, this latter finding suggests that the reduction in the feedback negativity associated with depressive symptoms may be accounted for by state negative affect. In this study, we sought to directly test this possibility by inducing either a sad or neutral mood within a sample unselected for history of depression. We recorded the feedback negativity during a simple gambling task where individuals could win or lose a

0959-4965 © 2010 Wolters Kluwer Health | Lippincott Williams & Wilkins DOI: 10.1097/WNR.0b013e3283356448

nominal amount of money on a series of trials. We predicted that individuals assigned to the sad mood induction would exhibit a reduced feedback negativity, as evidenced by a smaller difference between nonrewards and rewards, compared with individuals assigned to the neutral mood induction.

## Methods

### Participants and measures

Forty-six undergraduate students participated in the study (19 females, 27 males). Twenty-three participants [10 females; age: mean=18.35, standard deviation (SD)=1.03] were randomly assigned to the neutral mood induction, and the remaining 23 (nine females; age: mean=18.57, SD=0.95) were assigned to the sad mood induction. The two groups did not differ in age or sex (both $P > 0.50$). No participants discontinued their participation in the experiment once the procedures had begun. All participants received course credit and \$5.00 (winnings from the gambling task) for their participation. Informed consent was obtained from participants before each experiment. This study was formally approved by the Stony Brook University Institutional Review Board.

To partially control for the effects of individual differences in psychological variables unrelated to the mood induction, the short-form version of the Depression Anxiety Stress Scale (DASS-21) [18] was administered to all participants at the beginning of the experiment. The DASS-21 is a self-report measure that captures symptoms of depression, anxiety, and stress reactivity over the previous week. The reliability and validity of the DASS-21 has been shown to be excellent in nonclinical samples [19].

### Mood induction

The sad and neutral mood induction paradigms were based on the guidelines provided by Rottenberg *et al.* [20] for using film clips to elicit discrete emotional states. Each mood induction consisted of two 5-min film clips and a song that was played in the background while participants completed a series of computer tasks. In the neutral mood induction, the film clips used were from 'Alaska's Wild Denali', and the song used was 'Robert Ronne's Meditation No. 19'. In the sad mood induction, the clips used were from 'The Champ and My Girl', and the song used was Gabriel Faure's Piano Quintet No. 1 in D Minor (Op. 89). Upon completing the computer tasks, a pleasant mood was induced in all participants using an amusing film clip.

To assess current mood throughout the experiment, the valence scale of the Self-Assessment Manikin was used [21]. Participants were asked to rate their current emotional state ranging from one (maximally pleasant) to nine (maximally unpleasant). This measure was administered at five points throughout the experiment: before and after each of the two film clips, and again at the conclusion of the experiment. For this study, the ratings of interest were those taken at baseline and immediately following the second film clip (i.e. immediately before the gambling task).

### Gambling task

The gambling task was administered using Presentation software (Neurobehavioral Systems, Inc., Albany, California, USA) to control the presentation and timing of all stimuli. On each trial, participants were shown a graphic displaying two doors (occupying 6° of the visual field vertically and 8° horizontally) and were told to choose which door they wanted to open. Participants were told to press the left mouse button to choose the left door or the right mouse button to choose the right door. Following each choice, a feedback stimulus appeared on the screen informing the participants whether they won or lost money on that trial. A green '↑' indicated a correct guess and a gain of \$0.50, while a red '↓' indicated an incorrect guess and a loss of \$0.25 (each occupying 3° of the visual field vertically and 1° horizontally). A fixation mark ( + ) was presented before the onset of each stimulus. At the end of each trial, participants were presented with the instruction 'Click for the next round'. The task consisted of 40 trials total, with positive feedback given on exactly 20 trials (i.e. 50%). Feedback was presented in a random order for each participant. The order and timing of all stimuli were as follows: (i) the graphic of two doors was presented until a response was made, (ii) a fixation mark was presented for 1000 ms, (iii) a feedback arrow was presented for 2000 ms, (iv) a fixation mark was presented for 1500 ms, and (v) 'Click for the next round' was presented until a response was made.

### Procedure

At the beginning of the laboratory session, participants completed the DASS-21. Following a brief description of the experiment, electroencephalograph sensors were attached. Participants viewed the first film clip (with premood and postmood ratings) and performed two computer tasks unrelated to this study. Participants then viewed the second film clip (with premood and postmood ratings) and were introduced to the gambling task. To familiarize participants with the gambling task, they were then given a practice block containing five trials. Participants then performed the main task, with the running total of money earned to that point presented after the first 20 trials. Finally, participants performed another unrelated computer task, completed a final mood rating, were paid their winnings (i.e. \$5.00), and watched an amusing film clip.

### Psychophysiological recording, data reduction, and analysis

The electroencephalogram was recorded using a custom cap (Cortech Solutions, Wilmington, North Carolina, USA)

and the ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands). The signal was preamplified at the electrode with a gain of 16×; electroencephalogram data was digitized at 64-bit resolution with a sampling rate of 512 Hz using a low-pass fifth order sinc filter with a half-power cutoff of 102.4 Hz. Recordings were taken from 64 scalp electrodes based on the 10/20 system, as well as two electrodes placed on the left and right mastoids. The electrooculogram was recorded from four facial electrodes: two 1 cm above and below the left eye, one 1 cm to the left of the left eye, and one 1 cm to the right of the right eye. Each electrode was measured online with respect to a common mode sense electrode that formed a monopolar channel. Off-line analysis was performed using Brain Vision Analyzer software (Brain Products, Munich, Germany). All data were rereferenced to the average of the two mastoids and band-pass filtered with cutoffs of 0.1 and 30 Hz. The electroencephalogram was segmented for each trial, beginning 200 ms before feedback onset and continuing for 800 ms following feedback onset. Each trial was corrected for blinks and eye movements using the method developed by Gratton and colleagues [22]. Specific channels were rejected in each trial using a semi-automated procedure, with physiological artifacts identified by the following criteria: a step of more than 50 μV between sample points, a difference of 300 μV within a trial, and a maximum difference of less than 0.5 μV within 100-ms intervals.

Stimulus-locked responses were averaged separately for nonrewards and rewards and the activity in the 200-ms window before feedback onset served as the baseline. The feedback negativity was quantified as the 50-ms window surrounding the peak negative deflection in the difference wave (nonreward minus reward) at a pooling of Fz/FCz for each participant. A difference wave approach was chosen owing to the fact that an apparent change in component magnitude can result instead from the onset of a second, opposite-going component [23]. This is particularly relevant in studies of the feedback negativity, insofar as feedback is thought to elicit phasic decreases and increases in dopamine signals to the anterior cingulate cortex [5]. By scoring the difference between nonreward and reward, variation in the feedback negativity may reflect abnormalities related to processing positive feedback, negative feedback, or both [1,9,10]. All statistical analysis was performed using SPSS (Version 17.0; SPSS, Inc., Chicago, Illinois, USA).

## Results

### Self-report ratings

Participants in the Sad (mean=23.83, SD=15.64) and Neutral (mean=18.89, SD=13.92) groups did not significantly differ on the total scores of the DASS-21 ($P=0.26$) or in their baseline mood ratings (Neutral: mean=3.17, SD=0.98; Sad: mean=3.74, SD=1.39; $P=0.12$). Following the induction, individuals in the Sad group (mean=6.00, SD=1.88) reported significantly higher mood ratings (i.e. greater sadness) compared with the Neutral group (mean=3.78, SD=0.80; $t(44)=5.20$, $P<0.001$).

### Feedback negativity

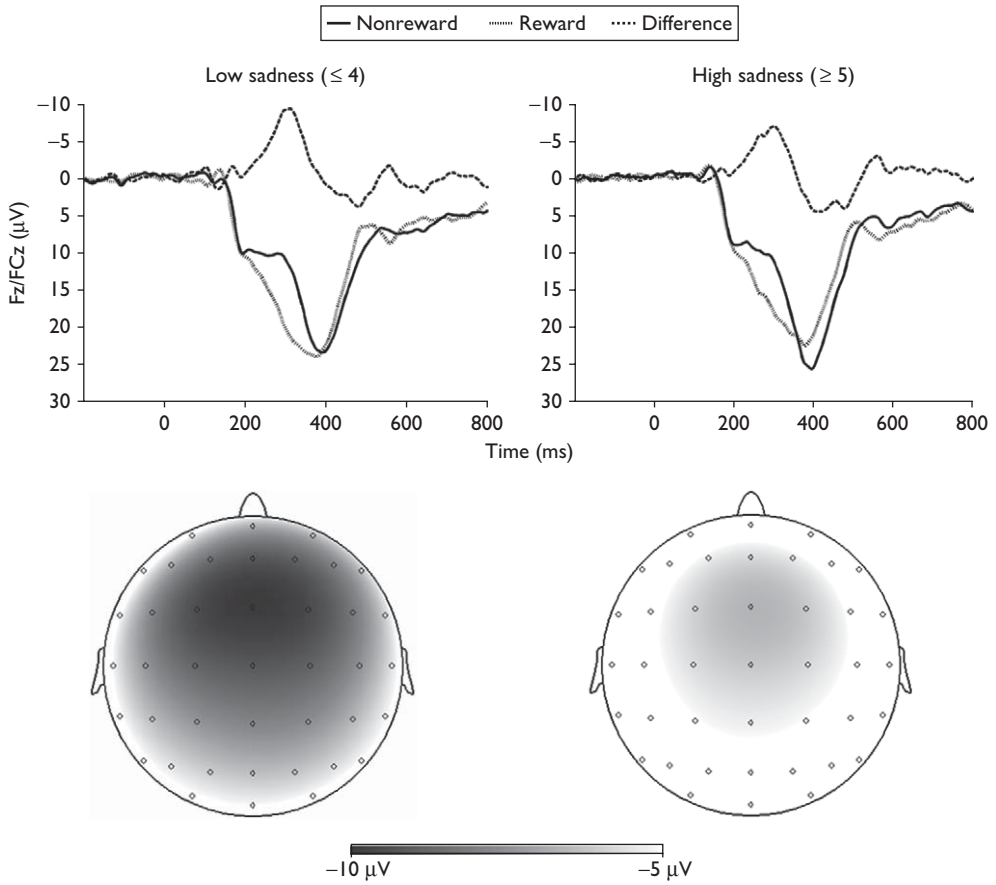
The feedback negativity was somewhat reduced within the Sad group (mean=−9.56, SD=5.93) compared with the Neutral group (mean=−8.15, SD=4.92), although this difference was not statistically significant ($P=0.39$). Across the entire sample, however, a significant inverse association was found between the FN and postinduction mood ratings ($r=0.30$, $P<0.05$; Figs 1 and 2). The feedback negativity is a numerically negative response, so this positive correlation value indicates that greater reported sadness was associated with less differentiation between nonrewards and rewards. After adjusting for the effects of baseline sadness, total DASS-21 score, age, and sex using multiple linear regression, the inverse association between postinduction sadness and the feedback negativity remained significant ($\beta=0.36$, $P<0.05$). None of these other predictors were significantly related to the feedback negativity (all $P>0.50$).

## Discussion

In this study, state mood was shown to influence early neural responses to nonrewards and rewards in a simple gambling task. Greater reported sadness predicted a reduced feedback negativity, calculated as the difference between monetary losses and gains, and this relationship was also present after controlling for the effects of baseline sadness, age, sex, and psychological distress. By demonstrating a direct relationship with variation in state mood, this result builds upon an earlier finding where the feedback negativity was shown to be inversely related to the severity of depressive symptoms over the previous week [12].

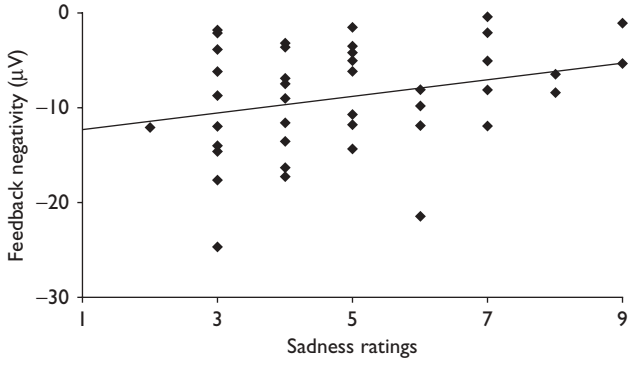
It should be noted that, although the sad mood induction produced the expected changes in mood ratings compared with the neutral induction, group status alone was not sufficient to capture the inverse association between the feedback negativity and sadness. A more robust effect was identified when the feedback negativity was instead related directly to self-report ratings. A closer inspection of the mood ratings across participants revealed that, of the 23 people assigned to the sad induction, 19 (83%) reported an increase in sadness. In the neutral induction, meanwhile, 13 out of the 23 people (57%) reported an increase in sadness, which could be explained in part by participant fatigue or boredom. In other words, although there was a greater increase in negative affect among individuals who were randomly assigned to the sad mood induction, a number of individuals in the neutral mood induction also reported such an increase. For this study, therefore, the key predictor of feedback negativity amplitude was the propensity to experience negative emotions such as sadness, of which there is considerable

**Fig. 1**




Top: event-related potentials locked to the presentation of feedback for nonreward and reward trials, as well as the difference. Bottom: scalp distribution of the difference between nonreward and reward trials from 275 to 325 ms, where the feedback negativity is maximal.

**Fig. 2**




Scatterplot depicting the relationship between the feedback negativity (nonreward minus reward) and postinduction mood ratings.

variability across individuals. In particular, there is evidence that sad mood inductions may activate attentional and recall biases in populations that are vulnerable to experiencing depression, such as currently healthy individuals with a history of depression [24] and never-depressed children with a parental history of depression [25]. It stands to reason, therefore, that the feedback negativity may be a suitable measure for detecting abnormal processing of rewards and nonrewards in these groups, a direction which merits further investigation.

## Conclusion

This study provides evidence that state variation in mood predicts the amplitude of early neural responses to nonrewards compared with rewards. Individuals reporting greater sadness immediately before performing a simple gambling task exhibited reduced neural differentiation between monetary losses and gains, suggesting that propensity to experience negative affect may be a critical component in how negative and positive outcomes are processed in the brain.

## References

1 Dunning JP, Hajcak G. Error-related negativities elicited by monetary loss and cues that predict loss. *Neuroreport* 2007; **18**:1875–1878.

2 Gehring WJ, Willoughby AR. The medial frontal cortex and the rapid processing of monetary gains and losses. *Science (New York, NY)* 2002; **295**:2279–2282.
3 Hajcak G, Holroyd CB, Moser JS, Simons RF. Brain potentials associated with expected and unexpected good and bad outcomes. *Psychophysiology* 2005; **42**:161–170.
4 Hajcak G, Moser JS, Holroyd CB, Simons RF. The feedback-related negativity reflects the binary evaluation of good versus bad outcomes. *Biol Psychol* 2006; **71**:148–154.
5 Holroyd CB, Coles MG. The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. *Psychol Rev* 2002; **109**:679–709.
6 Miltner WH, Braun CH, Coled MGH. Event-related brain potentials following incorrect feedback in a time-estimation task: evidence for a generic neural system for error detection. *J Cogn Neurosci* 1997; **9**:788–798.
7 Nieuwenhuis S, Ridderinkhof KR, Talsma D, Coles MG, Holroyd CB, Kok A, *et al*. A computational account of altered error processing in older age: dopamine and the error-related negativity. *Cogn Affect Behav Neurosci* 2002; **2**:19–36.
8 Holroyd CB, Nieuwenhuis S, Yeung N, Cohen JD. Errors in reward prediction are reflected in the event-related brain potential. *Neuroreport* 2003; **14**:2481–2484.
9 Hajcak G, Moser JS, Holroyd CB, Simons RF. It's worse than you thought: the feedback negativity and violations of reward prediction in gambling tasks. *Psychophysiology* 2007; **44**:905–912.
10 Holroyd CB, Larsen JT, Cohen JD. Context dependence of the event-related brain potential associated with reward and punishment. *Psychophysiology* 2004; **41**:245–253.
11 Holroyd CB, Hajcak G, Larsen JT. The good, the bad and the neutral: electrophysiological responses to feedback stimuli. *Brain Res* 2006; **1105**:93–101.
12 Foti D, Hajcak G. Depression and reduced sensitivity to non-rewards versus rewards: evidence from event-related potentials. *Biol Psychol* 2009; **81**:1–8.
13 Pizzagalli DA, Iosifescu D, Hallett LA, Ratner KG, Fava M. Reduced hedonic capacity in major depressive disorder: evidence from a probabilistic reward task. *J Psychiatr Res* 2008; **43**:76–87.
14 Davidson RJ. Affective style and affective disorders: perspectives from affective neuroscience. *Cogn Emotion* 1998; **12**:307–330.
15 Henriques JB, Davidson RJ. Regional brain electrical asymmetries discriminate between previously depressed and healthy control subjects. *J Abnorm Psychol* 1990; **99**:22–31.
16 Drevets WC. Neuroimaging studies of mood disorders. *Biol Psychiatry* 2000; **48**:813–829.
17 Drevets WC, Bogers W, Raichle ME. Functional anatomical correlates of antidepressant drug treatment assessed using PET measures of regional glucose metabolism. *Eur Neuropsychopharmacol* 2002; **12**:527–544.
18 Lovibond SH, Lovibond PF. *Maual for the depression anxiety stress scales*. Sydney: Psychology Foundation; 1995.
19 Henry JD, Crawford JR. The short-form version of the depression anxiety stress scales (DASS-21): construct validity and normative data in a large non-clinical sample. *BrJ Clin Psychol* 2005; **44**:227–239.
20 Rottenberg J, Ray RD, Gross JJ. Emotion elicitation using films. In: Coan JJ, Allen JJB, editors. *Handbook of emotion elicitation and assessment*. New York: Oxford University Press; 2007. pp. 9–28.
21 Lang PJ. *Self-assessment manikin*. Gainesville, FL: The Center for Research in Psychophysiology, University of Florida; 1980.
22 Gratton G, Coles MG, Donchin E. A new method for off-line removal of ocular artifact. *Electroencephalogr Clin Neurophysiol* 1983; **55**:468–484.
23 Luck SJ. *An introduction to the event-related potential technique*. Cambridge, Massachusetts: MIT Press; 2005.
24 Scher CD, Ingram RE, Segal ZV. Cognitive reactivity and vulnerability: empirical evaluation of construct activation and cognitive diatheses in unipolar depression. *Clin Psychol Rev* 2005; **25**:487–510.
25 Joormann J, Talbot L, Gotlib IH. Biased processing of emotional information in girls at risk for depression. *J Abnorm Psychol* 2007; **116**:135–143.