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Reduced sensitivity to neutral feedback versus negative feedback in subjects with mild depression: Evidence from event-related potentials study

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ABSTRACT

Many previous event-related potential (ERP) studies have linked the feedback related negativity (FRN) component with medial frontal cortex processing and associated this component with depression. Few if any studies have investigated the processing of neutral feedback in mildly depressive subjects in the normal population. Two experiments compared brain responses to neutral feedback with behavioral performance in mildly depressed subjects who scored highly on the Beck Depression Inventory (high BDI) and a control group with lower BDI scores (low BDI). In the first study, the FRN component was recorded when neutral, negative or positive feedback was pseudo-randomly delivered to the two groups in a time estimation task. In the second study, real feedback was provided to the two groups in the same task in order to measure their actual accuracy of performance. The results of experiment one (Exp. 1) revealed that a larger FRN effect was elicited by neutral feedback than by negative feedback in the low BDI group, but no significant difference was found between neutral condition and negative condition in the High BDI group. The present findings demonstrated that depressive tendencies influence the processing of neutral feedback in medial frontal cortex. The FRN effect may work as a helpful index for investigating cognitive bias in depression in future studies.

1. Introduction

Our complex and dynamic living environment requires human beings to learn how to predict and evaluate the consequences of actions that lead to rewards and punishments. For this reason accurate processing of external feedback is essential if humans are to optimize their behavior (Miltner, Braun, & Coles, 1997). Much research has focused on the brain activity associated with the evaluation of outcomes by examining the timing of electroencephalography (EEG) responses (Holroyd & Coles, 2002; Li, Han, Lei, Holroyd, & Li, 2011; Li et al., 2010; Ullsperger, Fischer, Nigbur, & Endrass, 2014; Walsh & Anderson, 2012). Investigations into feedback evaluation have consistently found negative deflection in the Event-Related Potential (ERP) following the presentation of negative feedback, an effect that has been termed Feedback Related Negativity (FRN, Cohen, Wilmes, & van de Vijver, 2011; Holroyd & Coles, 2002; Li et al., 2009; Miltner et al., 1997). This time-domain trial-averaged FRN component peaks at 250–300 ms after feedback and has a fronto-central distribution. Convergent findings from multiple methodologies suggest that the FRN is probably generated in the anterior cingulate cortex (ACC) in medial frontal cortex (Hauser et al., 2014; Warren, Hyman, Seamans, & Holroyd, 2014; but see Nieuwenhuis, Slagter, Alting von Geusau, Heslenfeld, & Holroyd, 2005).

Theories concerning the functional significance of FRN have been constantly updated in the last two decades. The current most influential theoretical account of FRN comes from Holroyd and Coles's "reinforcement learning theory of the error-related negativity (RL-ERN theory)". According to this theory, the FRN amplitude reflects reward prediction error, i.e. a signed value corresponding to the difference between the obtained reward and the expected reward (Holroyd & Coles, 2002; Nieuwenhuis, Holroyd, Mol, &

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Coles, 2004). This explanation has been supported by abundant evidence in FRN studies (e.g. Holroyd, Krigolson, Baker, Lee, & Gibson, 2009; Krigolson, Pierce, Holroyd, & Tanaka, 2009; Walsh & Anderson, 2012). However, several studies in the last ten years have provided evidence that the FRN conveyed an unsigned prediction error or "salience" encoding signal (Donkers, Nieuwenhuis, & Van Boxtel, 2005; Hauser et al., 2014; Oliveira, McDonald, & Goodman, 2007; Talmi, Atkinson, & El-Deredy, 2013). More recently, Sambrook and Goslin (2015) utilized the "great grand averages" approach in a meta-analysis study and showed strong effects of magnitude and likelihood on the FRN, which provided new evidence to support the RL_ERN theory.

Based on the predictions of RL_ERN theory, researchers proposed that the feedback processing system, putatively indexed by the FRN component, reveals a binary way to evaluate current outcomes, i.e. whether the current outcome is worse than expected or not (e.g. Hajcak, Moser, Holroyd, & Simons, 2006; Yeung & Sanfey, 2004). However, several studies have explored the phenomenon by introducing neutral feedback and showed that neutral feedback elicited a relatively larger FRN than negative feedback (Gu, Ge, Jiang, & Luo, 2010; Hirsh & Inzlicht, 2008; Li, Baker, Warren, Li, submitted for publication; Müller, Möller, Rodriguez-Fornells, & Münte, 2005). Müller et al. (2005) first found that the FRN occurred earlier and had a higher peak in the neutral condition than in the negative feedback condition. This discrepancy between neutral and negative feedback was also observed in two studies with negative affective states (Gu et al., 2010; Hirsh & Inzlicht, 2008). To systematically explore the effect of neutral feedback on the amplitude of the FRN, Holroyd and his colleagues (2006) conducted five experiments, which included an intermediate reward condition in the first three experiments and a neutral feedback condition in the later two experiments. Their results were consistent with the now widely accepted proposals that the evaluation system classifies outcomes into two categories: the satisfied outcome and unsatisfied outcome. Hence, it remains unclear how the evaluation system works when it comes to the neutral feedback. Thus investigating the FRN effect elicited by neutral feedback may also contribute to the above-mentioned arguments concerning the theoretical account of the FRN phenomenon because neutral feedback is a special case in terms of valence and magnitude.

According to the RL_ERN theory, the FRN manifests the dopamine signal transferred from the basal ganglia to the medial prefrontal cortex, more specifically, the ACC (Holroyd & Coles, 2002). So far, accumulating evidence shows that the patterns of activation in the ACC during performance monitoring vary as a function of individual differences in personality (Van Noordt & Segalowitz, 2012). Major depressive disorder (MDD) is a highly prevalent multifactorial psychiatric disorder and has been characterized as an abnormal tendency to engage in negative mood states, together with difficulty in disengaging from negative mood states (Holtzheimer & Mayberg, 2011). The impairment of ACC function by depression has been demonstrated in a number of ERP studies reporting that participants with severe MDD showed hyperactivation to internal or external error compared with the control group (Holmes & Pizzagalli, 2008; Santesso et al., 2008; but not in Foti & Hajcak, 2009; Ruchsow et al., 2004). However, it is still unclear whether or not depression influences the reward processing of neutral feedback. In fact, two FRN studies focused on individual differences in personality have found larger FRN effects following neutral feedback in subjects scoring high on neuroticism and high on trait anxiety scales (Gu et al., 2010; Hirsh & Inzlicht, 2008). Given that depression shares the same underlying biases of information processing with trait anxiety (Mathews & MacLeod, 2005) and that such biases might be predicted by neuroticism (Miller & Pilkonis, 2006), it is plausible that depression may also affect the FRN effect with neutral feedback.

To our knowledge, no study to date has focused on the FRN elicited by neutral feedback in depression. The most relevant study came from Mies et al. (2011), in which they investigated both behavioral and electrophysiological responses to feedback validity in non-medicated depressed patients. Their results found that non-medicated depressed in-patients showed a more pronounced FRN amplitude regardless of feedback validity. It is worth noting that the invalid feedback still contained valence information in Mies et al.'s study, and thus it differed from what we call "neutral feedback" here. Moreover, in previous studies the severity of depressive symptoms or neuroticism drives different neural responses toward reward (or correct) and non-reward (or error) feedback (Foti & Hajcak, 2009; Hirsh & Inzlicht, 2008; Tucker, Luu, Frishkoff, Quiring, & Poulsen, 2003). Tucker et al. (2003) found enhanced FRN responses in moderately depressed, but not in more severely depressed patients. Foti and Hajcak (2009) showed an enhancement of the FRN to non-rewards relative to rewards that was inversely related to depression. The FRN effect of neutral feedback has also been found to vary linearly as neuroticism scores change (Hirsh & Inzlicht, 2008). The current studies seek to extend this body of research by linking the neutral FRN phenomenon with mild depression in undergraduate students to complement the studies of clinical MDD (e.g. Mies et al., 2011).

Using a time estimation task, the present study investigated the outcome evaluation of neutral feedback in two groups varying in non-clinical depression. We also compared the behavioral performance of two groups in a subsequent time estimation task with real feedback. The aim of this study was to investigate whether depression attenuates or increases the performance-monitoring processing of neutral and negative feedback in medial prefrontal cortex. Given that depression is frequently associated with negatively biased information processing (cf. Mathews & MacLeod, 2005), the depressive group tested here was more likely to treat neutral feedback as negative feedback. Therefore, we hypothesized that neutral feedback would elicit comparable FRN amplitudes to negative feedback in the depressive group. We also predicted that distinctly different FRNs would be observed between neutral and negative feedback in the control group as it was in a previous study with random samples (Li et al., submitted for publication). In addition, we intended to compare the behavioral pattern of a depressive group and a control group in Exp. 2, in which real feedback was provided.

2. Methods

2.1. Participants

Participants were recruited from a large group (769) of students in two universities. 38 Participants aged 18–25 years were selected for the present study. All of them were assessed for depressive tendencies using the Beck Depression Inventory (BDI; Hautzinger, Bailer, & Worall, 1994) and were measured for trait anxiety using the trait section of the State Trait Anxiety Inventory (STAI; Spielberger, Gorssuch, Lushene, Vagg, & Jacobs, 1983) one week before the experiment was conducted. 19 participants (9 females, mean age 20.6) with a BDI score higher than 10 were identified as the high BDI group and another 19 participants (10 females, average age 20.7) with BDI scores lower than 10 were selected as the low BDI group. A chi-square analysis provided no evidence of significant differences in gender ratio between these two groups, $X^2(1, N = 38) = .11$, $p = .75$. The depression scores between the high BDI group (16 ± 4.49) and low BDI group (4.58 ± 2.87) were significantly different, $t(36) = 9.35$, $p < .001$, *Cohen's d* = 3.03. The anxiety scores between the high BDI group (49.95 ± 7.39) and the low BDI group (32.58 ± 5.53) were also significantly different, $t(36) = 8.20$, $p < .001$, *Cohen's d* = 2.66. Since no correlation was found between