Psychophysiology, 52 (2015), 449–459. Wiley Periodicals, Inc. Printed in the USA.

DOI: 10.1111/psyp.12370

EARLY CAREER AWARD ADDRESS, 2012

The reward positivity: From basic research on reward to a biomarker for depression

GREG HAJCAK PROUDFIT

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

Abstract

Feedback indicating monetary loss elicits an apparent negative deflection in the event-related potential (ERP) that has been referred to as the feedback error-related negativity, medial frontal negativity, feedback-related negativity, and feedback negativity—all conceptualizations that suggest a negative ERP component that is greater for loss than gain. In the current paper, I review a programmatic line of research indicating that this apparent negativity actually reflects a reward-related positivity (RewP) that is absent or suppressed following nonreward. I situate the RewP within a broader nomological network of reward processing and individual differences in sensitivity to rewards. Further, I review work linking reductions in the RewP to increased depressive symptoms and risk for depression. Finally, I discuss future directions for research on the RewP.

Descriptors: EEG/ERP, Psychopathology, Motivation, Individual differences, Emotion, Feedback negativity, Reward positivity

A (Very) Brief History

People tend to avoid mistakes. But in the early 1990s, errors became an exciting new focus of research in the event-related potential (ERP) world. Errors were found to elicit a robust negative deflection in the ERP time-locked to the response that was discovered nearly simultaneously in Germany and the United States, where it was referred to as the error negativity (Ne) and error-related negativity (ERN), respectively (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, & Coles, 1993; Gehring, Liu, Orr, & Carp, 2012). These initial papers, and many subsequent ones from Mike Coles' group, gave shape to the emerging science of the ERN.

For instance, Miltner and his colleagues published a paper in 1997 that reported a negative deflection, which resembled the ERN but occurred following feedback that an error was made (Miltner, Braun, & Coles, 1997). Miltner and colleagues utilized a time estimation task in which participants had to press a button 1 s after the presentation of an auditory cue on each trial. Importantly, the time window for a correct response was lengthened and shortened based on performance, such that participants were correct about half of the time. Thus, participants did not know when they made a mistake—they required feedback to know whether their response was correct or not. Approximately 250–350 ms after negative performance feedback, the ERP was characterized by a negative-going ERP that had a similar scalp distribution and source solution as the ERN; Miltner and colleagues suggested that both the response- and feedback-locked negativities might reflect, in the words of their paper's title, the activity of a generic error monitoring system (Miltner et al., 1997).

This possibility was further pursued by Clay Holroyd in a series of studies using reinforcement learning paradigms. Indeed, the response-locked ERN and the feedback ERN seemed functionally related: when participants did not yet know stimulus-response mappings, negative feedback elicited an ERN (i.e., the feedback ERN or fERN), whereas erroneous responses did not However, once stimulus-response mappings were learned, response errors elicited an ERN, whereas negative performance feedback did not (Holroyd & Coles, 2002). It appeared that the system that generated the ERN used either response- or feedback-related information to determine whether an error had been committed. These were critical data to the reinforcement learning model of the ERN (RL-ERN model; Holroyd & Coles, 2002). To test further predictions from the RL-ERN model, Holroyd used gambling experiments to examine how factors like the frequency of positive and negative feedback impacted the magnitude of the fERN (Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003).

This work was supported in part by grants from the NIMH (R01 MH097767; R03 MH094518; R01 MH069942), as well as NRSA training grants (MH090658; MH095307). I am incredibly grateful to my graduate mentor, Bob Simons—my time at the University of Delaware was a wonderful reward prediction error. My appreciation is also expressed to Ed Bernat, Josh Carlson, Joe Dien, Eddie Harmon-Jones, Clay Holroyd, Dan Klein, Roman Kotov, Christian Luhmann, Jason Moser, and Lillianne Mujica-Parodi. Most of the work was conducted with graduate students, former and present; working with them is the most rewarding part of my job: Jenny Bress, Dan Foti, Ellen Kessel, Autumn Kujawa, Alex Meyer, Anja Riesel, Colin Sauder, Ezra Smith, and Anna Weinberg. I also benefited from comments on earlier drafts of this manuscript from many of the people listed above, as well as Brady Nelson and Emily Kappenman.

Address correspondence to: Greg Hajcak Proudfit, Department of Psychology, Stony Brook University, Stony Brook, NY 11794-2500, USA. E-mail: greg.hajcak@stonybrook.edu

PSYCHOPHYSIOLOGY

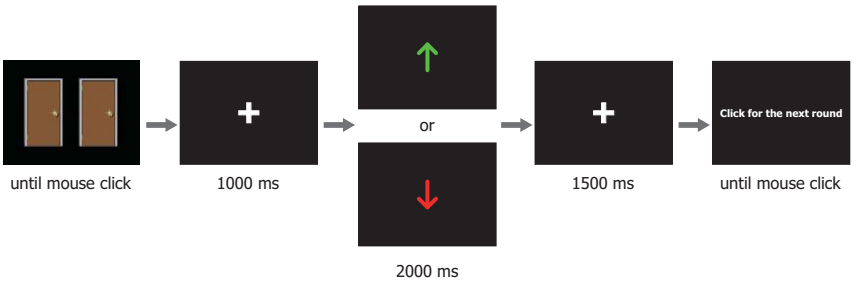

Figure 1. The doors task. On each trial, participants are shown two doors and pick one by clicking the left or right mouse button. A fixation cross is presented for 1,000 ms and followed by either gain (i.e., upward green arrow) or loss (i.e., downward red arrow) feedback—which are presented for 2,000 ms. A 1,500-ms fixation cross is next presented, and then the participant clicks either mouse button to start the next trial. Gain feedback is presented on exactly 50% of trials.

In 2002, Gehring and Willoughby utilized a clever manipulation in a gambling paradigm to create situations in which participants could win money and have made the "wrong" choice (e.g., the alternative choice would have led to a larger gain); they found that losses elicited a negativity around 250 ms after feedback—regardless of whether the feedback indicated the choice was correct or an error (Gehring & Willoughby, 2002). Since this negativity did not appear to track performance errors, Gehring and Willoughby referred to it as the medial frontal negativity (MFN).[1] Subsequent papers using gambling paradigms referred to the feedback-related negativity (FRN) or feedback negativity (FN; Hajcak, Moser, Holroyd, & Simons, 2006; Yeung, Holroyd, & Cohen, 2005). In the span of less than a decade, this feedback-locked ERP itself became a hot topic of research, and has been referred to as the fERN, MFN, FRN, and FN.

In what follows, I'll argue that the fERN/MFN/FRN/FN is actually a reward-related positivity (i.e., reward positivity, or RewP).[2] To be clear, I'm not the first person to make this suggestion (Holroyd, Pakzad-Vaezi, & Krigolson, 2008). Even though we have argued this conceptual point elsewhere (Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011; Foti, Weinberg, Dien, & Hajcak, 2011a; Kujawa, Smith, Luhmann, & Hajcak, 2013), I have resisted referring to it by a new name—in part, because doing so seemed almost comical, and potentially more confusing than useful or helpful. Given the evidence, however, I believe referring to it as the reward positivity (RewP) is both appropriate and accurate—and that this nomenclature more closely reflects the functional significance of variability in the ERP following gain versus loss feedback.

ERP Componentry

In all of the data I will describe, participants play a simple guessing task (i.e., the doors task, see Figure 1): on each trial, they are shown two doors; participants then pick a door and subsequently receive feedback indicating either a monetary gain (i.e., upward-pointing green arrow) or loss (i.e., downward-pointing red arrow). Because probability has a large impact on stimulus-locked ERPs, gains and losses are presented on exactly 50% of trials. In addition, because losses are subjectively about twice as valuable as gains (Tversky & Kahneman, 1992)—and to ensure that participants accrue money over the course of the experiment—rewards are twice as large in magnitude as losses (e.g., 50 and 25 cents, respectively).

Figure 2 (top left) presents ERPs at FCz elicited by gain and loss feedback in our doors task averaged across 32 college students. Approximately 300 ms after feedback onset, the ERP waveforms for losses and gains are maximally different, such that losses are more negative compared to gains. Indeed, when you create a difference waveform and subtract gains from losses, the resulting difference is a negativity maximal at frontocentral sites (Figure 2, top right). However, if you instead subtract losses from gains, the resulting difference waveform is a positivity (Figure 2, top right). Figure 2 presents the scalp distribution of the loss-gain (bottom left) and gain-loss (bottom right) differences from 250 to 350 ms after feedback. Whether there is a loss-related negativity or a gain-related positivity depends on the subtraction performed. The key question is whether variation in the ERP reflects the addition of a process that is positive in polarity on gain trials, or the addition of a process that is negative in polarity on loss trials. Alternatively, which ERP is the "baseline" response?

One seemingly reasonable approach to answer this question is to consider the ERP elicited by feedback with an intermediate meaning (i.e., breaking even). That is, we could compare neural activity elicited by winning money, breaking even, and losing money. Indeed, when we did this—across five experiments—we found that breaking even elicited a relative negativity compared to gains; breaking even was just like losing (Holroyd, Hajcak, & Larsen, 2006). It is interesting to note that, at the time, we didn't even consider the possibility that the ERP response to losses and breaking even was the baseline response, and that rewards elicited a relative positivity.[3] Rather, we suggested that losses and breaking even might be similarly categorized as unfavorable outcomes by the system that generates the negativity (i.e., a negativity was elicited by the absence of gain).

In a more recent study, we modified our doors task so that each trial was preceded by a cue. The cue indicated one of two trial types: on half of the trials, the two possible outcomes were breaking even or losing; on the other half, the possible outcomes were

1. A subsequent study showed that whether a relative negativity was elicited by the performance or utilitarian aspect of feedback depended on what was made more salient to participants (Nieuwenhuis, Yeung, Holroyd, Schurger, & Cohen, 2004).

2. RewP is utilized because RP is an existing abbreviation for the readiness potential. Although I argue that the time-domain difference between gains and losses is due to a RewP, time-frequency approaches suggest increases in both loss- and gain-related neural activity in the time range of the RewP—a topic I will return to later.

3. I was not wondering about this possibility—but Clay Holroyd was (raised on p. 3166 of Nieuwenhuis, Slagter, von Geusau, Heslenfeld, & Holroyd, 2005). When I say "we," I refer only to what appears in the published paper.

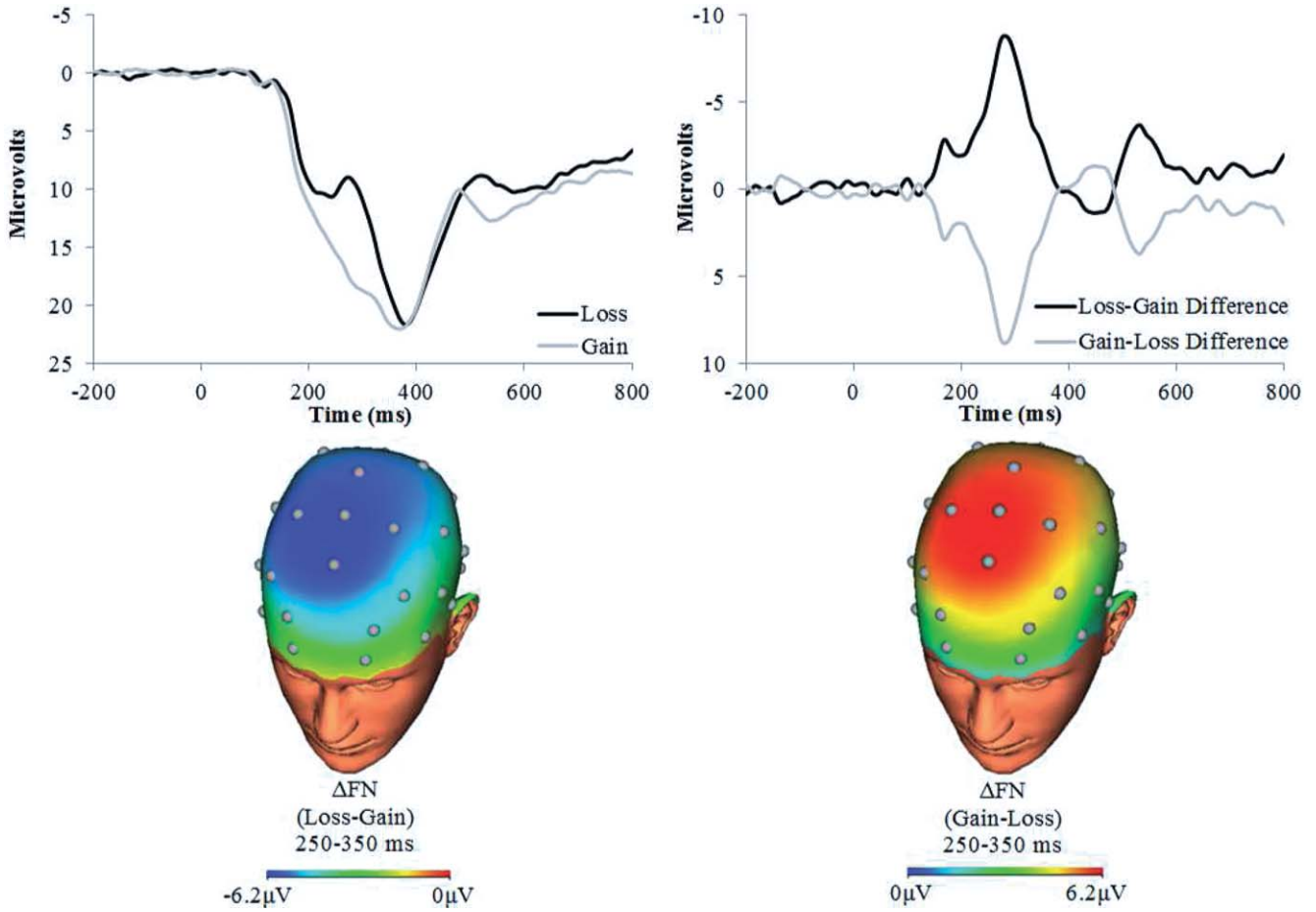

Figure 2. Feedback-locked ERPs at FCz (top, left) for losses (dark) and gains (light). Difference waveforms (top, right) at FCz suggest a relative negativity or positivity depending on whether gains are subtracted from the losses (dark) or losses are subtracted from gains (light), respectively. Negative is plotted up. In both ERP figures, the reward positivity (RewP) is evident as a relative positivity maximal between 250 and 350 ms following feedback indicated gain compared to loss. Scalp distribution of the loss minus gain difference (bottom, left) and gain minus loss difference (bottom, right) in the time range of the RewP.

breaking even or winning. In this way, breaking even could either be the best or worst possible outcome on a given trial. In this experiment, breaking even was always associated with a negativity—even when it was the best possible outcome on a given trial (Kujawa et al., 2013). Only gains were different from the other outcomes—again suggesting the addition of a positivity on gain trials rather than a negativity on loss trials.

Why is it so challenging to see modulation of the feedback-related ERPs in terms of a reward-related positivity? Part of the answer is undoubtedly the historical precedent described above. However, I suspect the answer is also partially visual: for losses, there is a negative-looking something in the ERP; for gains, there appears to be nothing there. However, Holroyd pointed out that the apparent loss-related negativity has a striking resemblance to the N200 in terms of timing, morphology, and scalp distribution (Holroyd, 2004). In fact, when feedback contains no information, it generates an N200 rather than the absence of an N200—suggesting that the presence of an N200 is itself a baseline response (Baker & Holroyd, 2009). Thus, one possibility is that all informative feedback in a gambling task elicits an N200 that is suppressed by a reward-related positivity in this time range; indeed, this is what Holroyd and colleagues later went on to suggest (Holroyd et al., 2008).

Factor Analysis, Source Localization, and Correlations with fMRI-Based Measures

My own view of the underlying ERP componentry of winning and losing is further informed by temporal-spatial principal component analysis (PCA). PCA is a factor analytic approach that can be used to parse the observed ERP waveform into underlying constituent components (Dien, 2010; Donchin & Heffley, 1978). The utility of PCA is emphasized when one appreciates that the negative and positive deflections in the scalp-recorded ERP reflect overlapping neural processes and do not themselves index specific brain processes (Kappenman & Luck, 2012). As an example, there are occasions when PCA can indicate something about the underlying componentry that may not be obvious from the observed ERPs (e.g., Spencer, Dien, & Donchin, 2001). While still a graduate student in my lab, Dan Foti examined ERPs elicited by gains and losses in a sample of 85 college students using temporal-spatial PCA. Although it was not the focus of the paper, we reported that

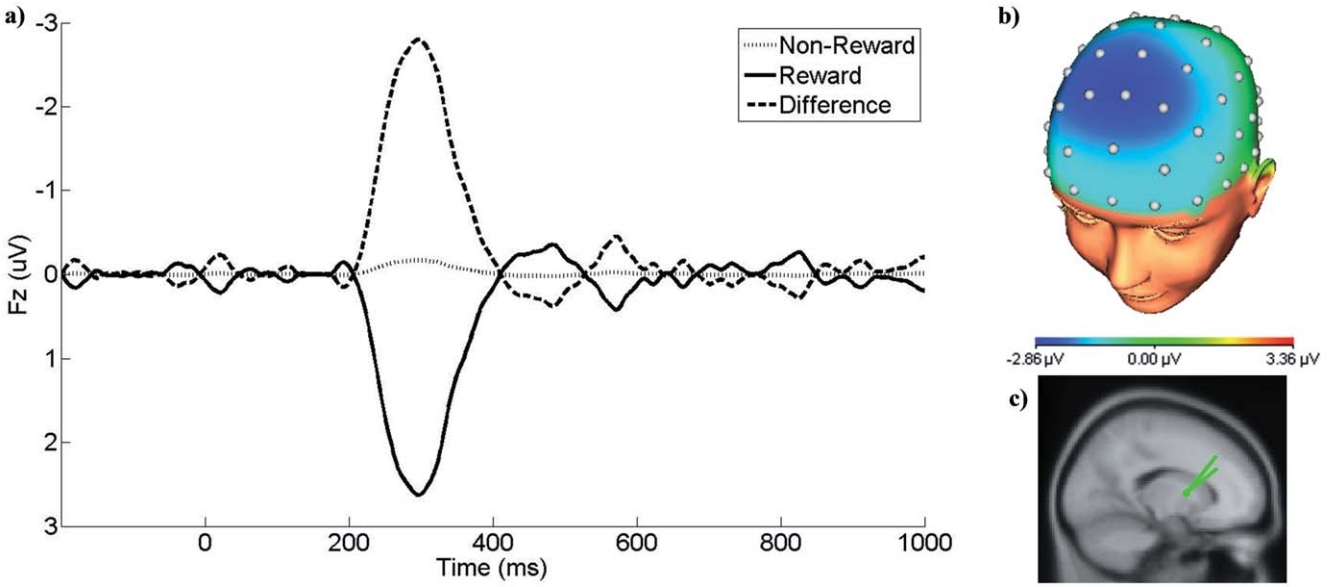

Figure 3. a: The waveforms at Fz representing the portion of the ERP associated with TF3/SF1, the PCA factor corresponding to the feedback negativity. Waveforms are presented for nonreward and reward trials, as well as the difference. b: The scalp topography of the difference between nonreward and reward trials at 297 ms, where the temporal loading is maximal. c: The dipole source associated with TF3/SF1. Reprinted from Foti, Weinberg, Dien, & Hajcak (2011a) with permission.

the PCA factor combination that accounted for the difference between gains and losses was actually a frontocentral positivity following gains that was maximal around 300 ms after feedback, and reduced following losses (Foti & Hajcak, 2009). Again, these PCA data suggested the addition of a positivity on gain trials that was reduced or absent on nonreward trials.

In a follow-up paper, we reported more details on this PCA-derived factor that differentiated rewards from losses (Foti et al., 2011a). These data are presented in Figure 3, and the factor that differentiated gains from losses was a positivity that was absent on nonreward trials. In comments on the first draft of that paper, a positive but suspicious reviewer wondered how it was possible for the absence of a reward-related positivity to create an apparent negativity on loss trials. To address this concern, we reconstructed the observed ERPs from a broader set of PCA factors (Foti et al., 2011a). As can be seen in Figure 4, we found that both rewards and nonrewards elicited an equivalent frontocentral P200 and slow positive wave that peaked around 200 and 300 ms, respectively; the reward-related positivity peaked just between these components, and its absence on nonreward trials—like a valley between two mountains—produced an apparent negativity in the ERP.

Furthermore, our source localization results suggested that this reward-related component was potentially generated in the putamen, a region of the basal ganglia (BG) implicated in reward (Foti et al., 2011a; see Figure 3c). Although a possible source in the BG linked our reward-related positivity to neural structures and circuits implicated in reward processing, it was a controversial suggestion because the prevailing wisdom is that subcortical structures such as the putamen do not contribute to scalp-recorded ERPs (see commentary by Cohen, Cavanagh, & Slagter, 2011; and our response: Foti, Weinberg, Dien, & Hajcak, 2011b).

In a subsequent study, we examined both ERP and fMRI measures among 42 participants who completed our doors task twice, in counterbalanced order (Carlson et al., 2011). The fMRI data confirmed that a number of regions implicated in reward processing were more active for gains compared to losses (e.g., ventral striatum, amygdala, orbital frontal cortex, medial frontal cortex). These fMRI data are indicative of increased neural activity on gain compared to loss trials. Using PCA, we replicated our previous factor structure, and found that the most likely neural generator of the RewP was in the dorsal striatum—a source that was consistent with our initial report. Moreover, we found that the RewP correlated with hemodynamic activity across the reward circuit (Carlson et al., 2011). Figure 5 presents data from Carlson et al. (2011), in which greater reward-related neural activity measured with fMRI in both the ventral striatum and medial prefrontal cortex was related to an increased RewP.

Evidence for a RewP derived using PCA has been replicated in other samples and studies from our group (Foti, Carlson, Sauder, & Proudfit, 2014; Weinberg, Riesel, & Proudfit, 2014) and other groups (Liu et al., 2014); moreover, the correspondence between the RewP and activity in the BG using fMRI has also been replicated (Becker, Nitsch, Miltner, & Straube, 2014; Foti, Carlson et al., 2014). Becker and colleagues used simultaneously recorded EEG and fMRI, and found that variation in the trial-to-trial ERP to reward predicted BOLD (blood oxygen level-dependent) signal change in the ventral striatum, midcingulate, and midfrontal cortex (Becker et al., 2014). Although these data further highlight the possibility that the RewP is generated by neural activity in the BG, an alternative possibility is that the RewP reflects the impact of BG activity on other structures. That is, the RewP could reflect either direct or indirect activation of the BG; regardless, the critical point is that variability in the ERP following gains versus losses is generated by a reward positivity (i.e., the RewP) that reflects mesocorticolimbic reward circuit activation.

Reward Positivity and Other Reward-Related Constructs

If the reward positivity (RewP) indexes reward-related neural activity, then it ought to relate to other reward-related constructs, in both

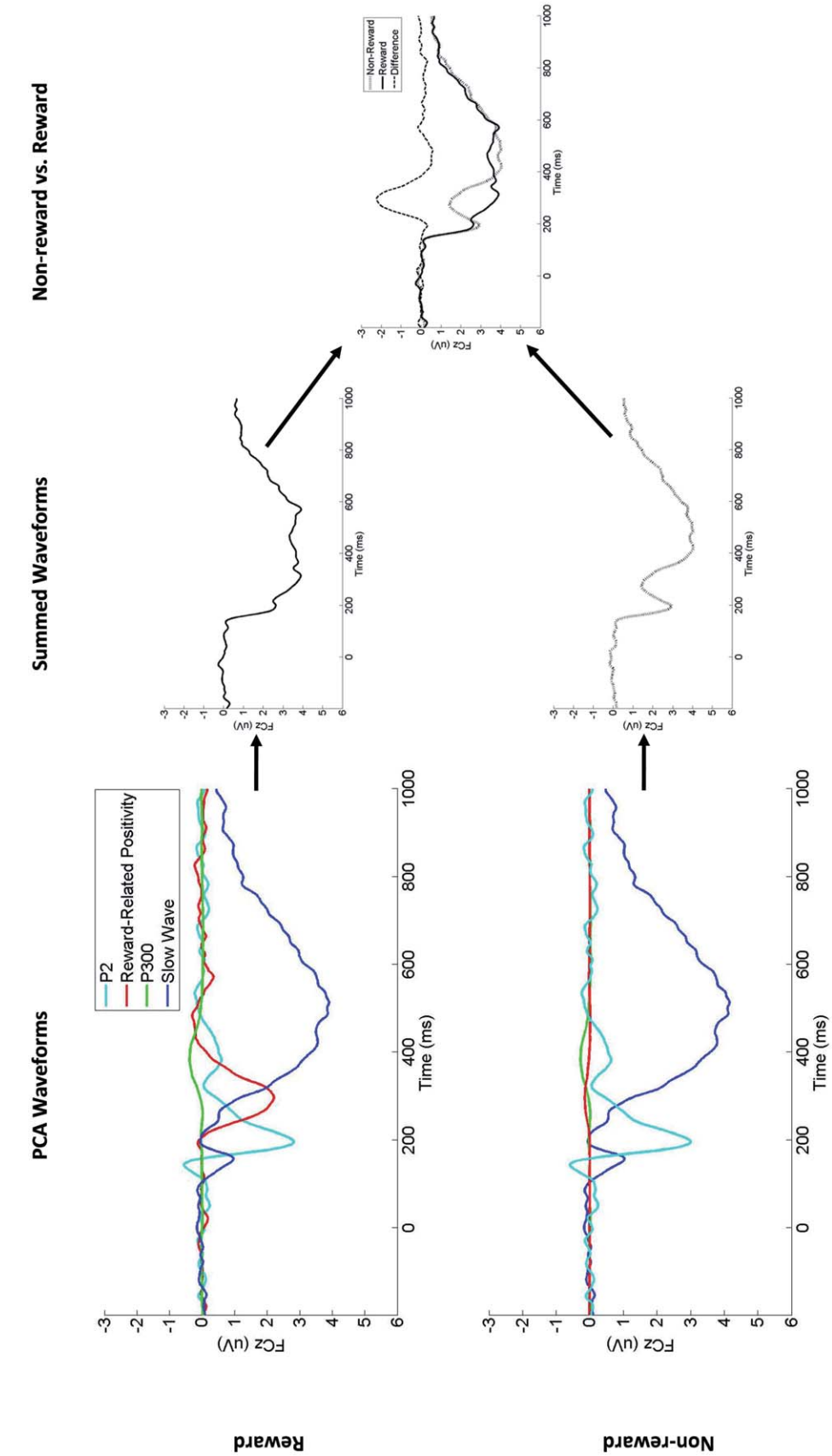

Figure 4. The waveforms of four temporospatial factors at FCz, their algebraic sum, and the comparison between nonreward and reward trials. Reprinted from Foti, Weinberg, Dien, & Hajcak (2011a) with permission.

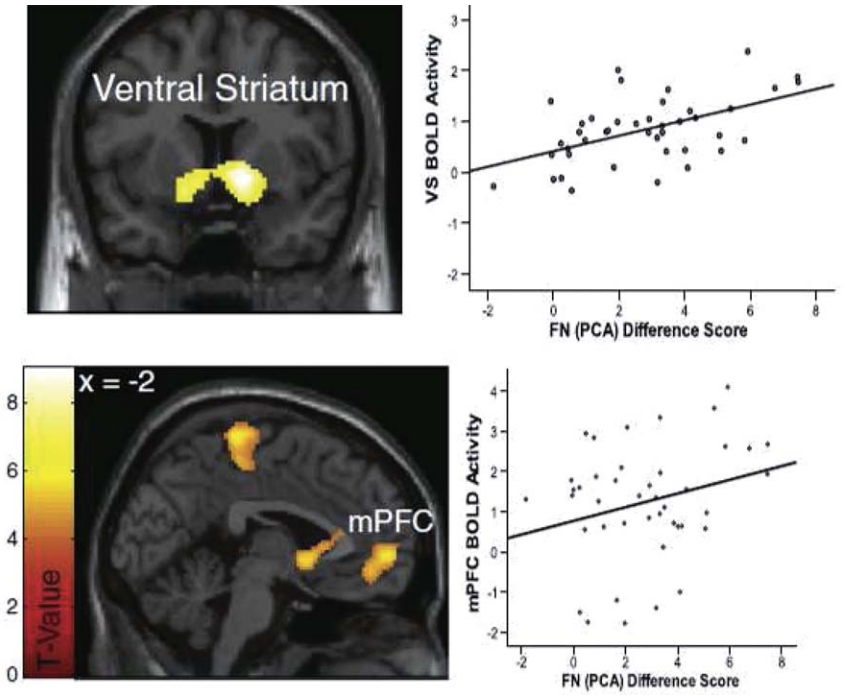

Figure 5. Greater activation was observed for monetary gain compared to loss bilaterally in the ventral striatum (top left) and medial prefrontal cortex (mPFC; bottom left). Scatter plot depicting the relationship between the FN and BOLD responses in the right ventral striatum (top left) and mPFC (bottom right). The FN was reverse-scored such that a more positive FN difference score reflected a greater difference between gains and losses (i.e., a larger RewP). Reprinted from Carlson et al. (2011) with permission.

within- and between-subjects designs. In a simple manipulation, we had participants perform our doors task under two conditions. In some blocks, participants were told that feedback indicated actual gains and losses—and following these blocks, participants were given their winnings in cash to emphasize the veracity of the instructions; in other blocks, participants were told that they would play the doors task, but that no actual money would be won or lost. The RewP—and the ERP response to gains in particular—was larger in blocks in which feedback was associated with actual rewards (Weinberg et al., 2014).

The magnitude of the RewP also predicts individual differences in sensitivity to reward as measured by both self-report and behavioral measures. Among 46 college students, we found that higher self-report scores on the Reward Responsiveness Scale (Van den Berg, Franken, & Muris, 2010) was related to a larger RewP (Bress, Smith, Foti, Klein, & Hajcak, 2012). That is, individuals who said that they respond more to potential rewards (e.g., "When I see an opportunity for something I like, I get excited right away") were also characterized by a larger RewP. Additionally, subjects with a larger RewP were also more biased in their behavior as a function of positive, rewarding feedback. We utilized Diego Pizzagalli's signal detection task—a behavioral task in which participants must make a difficult perceptual decision on each trial (i.e., decide if a mouth that is briefly presented on a schematic face is either short or long). In this task, participants only receive positive feedback and rewards (i.e., there is no negative feedback). Crucially, one type of correct response is rewarded three times more frequently than the other—and this creates a response bias that develops over the course of the task: because one response is rewarded more frequently, this response is chosen more frequently when the correct response is uncertain. Among individuals who developed a response bias, a larger RewP predicted a greater response bias to make richly rewarded decisions (Bress & Hajcak, 2013).

I have argued that the RewP indexes reward-related neural activity based on sensitivity to experimental context, factor analytic approaches (i.e., PCA), potential neural generators, and correlations with self-report, behavioral, and fMRI measures. We have found that the RewP is characterized by excellent psychometric properties in terms of both high internal reliability and test-retest reliability over 2 years (Bress, Meyer, & Proudfit, in press)—consistent with the view that it is a neurobehavioral trait (Patrick & Bernat, 2010; Patrick et al., 2013). The construct validity of the RewP as a neural indicator of reward sensitivity is, therefore, founded on its place within a broader nomological network (Cronbach & Meehl, 1955). Specifically, we have conceptualized reward sensitivity as a latent construct and the RewP as one indicator or manifestation. To the extent that the RewP indexes individual differences in reward sensitivity, it may be particularly relevant to conceptualizations of depression that highlight low positive affect and emotionality as a feature that distinguishes depression from anxiety (for a review, see Shankman & Klein, 2003).

Consistent with these models, we have found that major depressive disorder (MDD) and increased depressive symptoms are related to a reduced RewP. Among the unselected sample of 85 college undergraduates described above, we found that increased depressive symptoms predicted a smaller RewP (Foti & Hajcak, 2009). We subsequently found a reduced RewP among adults with diagnosed MDD (Foti, Carlson et al., 2014)—a finding that was recently reported by another lab (Liu et al., 2014). Moreover, we found a similar pattern among 64 unselected children aged 8–13:

increased self- and parent-reported depressive symptoms related to a smaller RewP (Bress et al., 2012). A reduced RewP appears to relate to depressive, but not anxious, symptoms (Bress, Meyer, & Hajcak, 2013; Bress et al., 2012). These data suggest some specificity of the relationship between an attenuated RewP and increased depressive symptoms, and that this relationship emerges relatively early in development.

The Reward Positivity and Risk for Depression

We have been particularly interested in whether a blunted RewP, as a neural indicator of reduced reward sensitivity, precedes increases in depression. That is, does a smaller RewP signal risk for subsequent increases in depression? This specific question is important as the field attempts to identify both neural correlates and predictors of depression. If a reduced RewP is a vulnerability marker, it should be abnormal among healthy individuals who are at increased risk for depression. That is, the RewP should relate to known risk factors even among those who have never been depressed (Ingram & Luxton, 2005). To address this issue, we examined the RewP among a sample of 407 never-depressed 9-year-olds in relation to maternal history of MDD—one of the best risk factors for depression (Gotlib & Goodman, 1999). Consistent with the notion that a reduced RewP indexes risk for depression, maternal history of MDD, but not anxiety, was associated with a smaller RewP; moreover, the RewP was most reduced among children who had mothers with more severe depressive histories (Kujawa, Proudfit, & Klein, 2014).

We are currently following these participants in ongoing and longitudinal studies that will ultimately allow us to examine whether the RewP can prospectively predict increases in depressive symptoms and onset of MDD. In the meantime, we already have preliminary data from two smaller studies suggesting that the RewP does, in fact, predict increases in depressive symptoms and the onset of depression prospectively. Above, I described a study on the RewP in relation to depressive symptoms in 64 children aged 8–13 (Bress et al., 2012); we brought 47 of these individuals back to the lab approximately 2 years after their initial visit and again measured the RewP using the doors task. We found that the relationship between the RewP and depressive symptoms was reproduced at the second testing session (i.e., higher depressive symptoms were again associated with a reduced RewP); in addition, a reduced RewP at the first testing session predicted increased depressive symptoms 2 years later (Bress et al., in press).

In a separate study of never-depressed adolescent girls, we similarly found that a smaller RewP prospectively predicted both first onset major depressive episodes and increased depressive symptoms 2 years after the RewP was measured in the lab (Bress, Foti, Kotov, Klein, & Hajcak, 2013). Data from Bress and colleagues' paper is presented in Figure 6. In this study, the RewP predicted increases in depressive symptoms beyond baseline depressive symptoms; indeed, in all analyses, the RewP was associated with a comparable effect size as baseline depressive symptoms—one of the best predictors of subsequent risk for increases in depression (Keenan, Feng, Hipwell, & Klostermann, 2009; Klein, Shankman, Lewinsohn, & Seeley, 2009). We are continuing to examine the RewP in larger and longitudinal studies that focus on adolescence—a key neurodevelopmental period for both changes in reward sensitivity (Galvan, 2013; Van Leijenhorst et al., 2010) and emerging symptoms of depression (Hankin et al., 1998).

Future Directions

Although the RewP appears relatively stable over time and is potentially traitlike (Bress et al., in press), the RewP is also sensitive to certain laboratory-based manipulations. For instance, the RewP is increased when outcomes signal actual rewards (Weinberg et al., 2014). A remaining question is the degree to which individual differences in the RewP can be shaped by environmental factors that impact reward sensitivity. Recently, we found that the relationship between a reduced RewP and parental history of depression was moderated by maternal parenting: supportive parenting appeared to buffer the impact of parental history of depression on children's RewP (Kujawa, Proudfit, Laptook, & Klein, in press). These data suggest the exciting possibility that experiences that shape response to reward can alter the RewP—and that the RewP may be a modifiable biomarker of risk for depression.

In the clinical science literature, there is increasing emphasis on moving beyond studies that compare a group of individuals with diagnosed psychopathology (e.g., MDD) to a group of healthy individuals. This approach is likely limited insofar as MDD is a heterogeneous disorder and highly comorbid with other disorders, which are also heterogeneous; furthermore, this dichotomous approach reifies extremes on what is most likely a continuum of variability in depressive symptoms. In recent years, the National Institute of Mental Health's Research Domain Criteria (RDoC) initiative has emphasized the need to understand dysfunction in core neural systems that may underlie dimensional individual differences that cut across disorders (Cuthbert & Insel, 2013; Insel et al., 2010; Sanislow et al., 2010). Along these lines, a recent report suggests that the RewP was reduced in MDD compared to healthy controls, and that reduction in the RewP was related to increased symptoms of anhedonia across both groups (Liu et al., 2014). Likewise, we recently found that MDD was characterized by a reduced RewP—and that this effect was driven by more specific deficits in mood reactivity to positive events in the MDD group (Foti, Carlson et al., 2014). Only MDD patients who reported impaired mood reactivity to positive events (i.e., they reported that their mood did not improve when something positive happens) were characterized by both a reduced RewP and blunted striatal response to reward measured using fMRI (see Figure 7). Collectively, these data point toward a relationship between the RewP and anhedonia (Foti, Carlson et al., 2014; Liu et al., 2014).

Along these lines, an important area for future research will be clarifying how the RewP relates to more specific phenotypic variation—as well as whether and how RewP abnormalities cross diagnostic boundaries. For instance, we have suggested that a reduced RewP characterizes increased depression, but not anxiety (Bress, Meyer, & Hajcak, 2013; Bress et al., 2012). One study found that schizophrenia was associated with an intact RewP (Horan, Foti, Hajcak, Wynn, & Green, 2012). These studies suggest some specificity of the relationship between the RewP and depressive symptoms. However, deficits in the RewP have also been found in externalizing disorders such as attention deficit hyperactivity disorder (Umemoto, Lukie, & Kerns, 2014; Van Meel, Heslenfeld, Oosterlaan, Luman, & Sergeant, 2011) and in relation to self-reported externalizing symptoms (Bernat, Nelson, Steele, Gehring, & Patrick, 2011). Thus, one possibility is that variation in core neural systems that support reward sensitivity, indexed by the RewP, is impacted by both depression and certain characteristics of externalizing disorders (e.g., impulsivity). That is, seemingly disparate disorders (e.g., internalizing and

externalizing) may have underlying commonalities in terms of neural dysfunction.

In terms of the RDoC matrix itself, the RewP appears to be a good candidate measure of the positive valence system construct of approach motivation—and it appears more related to reward consummation than anticipation. However, future research is needed to determine whether the RewP indexes more specific subconstructs (e.g., reward valuation, initial responsiveness to reward, sustained responsiveness to reward, reward learning). That is, we need to further parse reward sensitivity and understand more about the functional significance of the RewP. In all of the studies described above, we present gain and loss feedback 1 s after participants make a choice in the doors task. In one study, we found that when feedback was delayed (i.e., presented 6 s after response choice) the RewP was completely eliminated (Weinberg, Luhmann, Bress, & Hajcak, 2012). Based on these data, it seems unlikely that the RewP simply reflects the receipt of reward. Rather, these data suggest boundary conditions in which the RewP may integrate information about actions and outcomes, as might be the case for a reward prediction error signal (Holroyd, Krigolson, & Lee, 2011).

If simply delaying feedback can eliminate the RewP, it stands to reason that other task-related differences may similarly alter the RewP. At the beginning of this paper, I described the first published paper on the RewP from Miltner and colleagues (1997), who presented participants with positive and negative performance feedback in a time estimation task. As part of Dan Foti's dissertation, we had participants perform both our doors task and a time estimation task. Both tasks elicited a robust RewP—although they were uncorrelated with one another. Although many tasks can be used to elicit a RewP, aspects of reward sensitivity and individual differences may be task specific.

All of the research discussed to this point has focused on the time-domain representation of the RewP. Studies that have examined the difference between gains and losses using time-frequency analyses have reported both gain-related increases in the delta frequency band and loss-related increases in theta (Bernat, Nelson, Holroyd, Gehring, & Patrick, 2008; Bernat et al., 2011). We recently reanalyzed data from our large sample of undergraduates (Foti & Hajcak, 2009) using time-frequency decomposition of gain- and loss-related ERPs. In this study, we found that the time-domain measure of the RewP—both scored as the average activity in the ERP and using PCA—was predicted independently by loss-related theta and gain-related delta (Foti, Weinberg, Bernat, & Proudfit, in press). Moreover, we found that loss-related theta and gain-related delta had unique neural generators in the anterior cingulate cortex (ACC) and BG, respectively—and that depressive symptoms were only related to gain-related delta (Foti, Weinberg et al., in press). These time-frequency data suggest a possible rec-

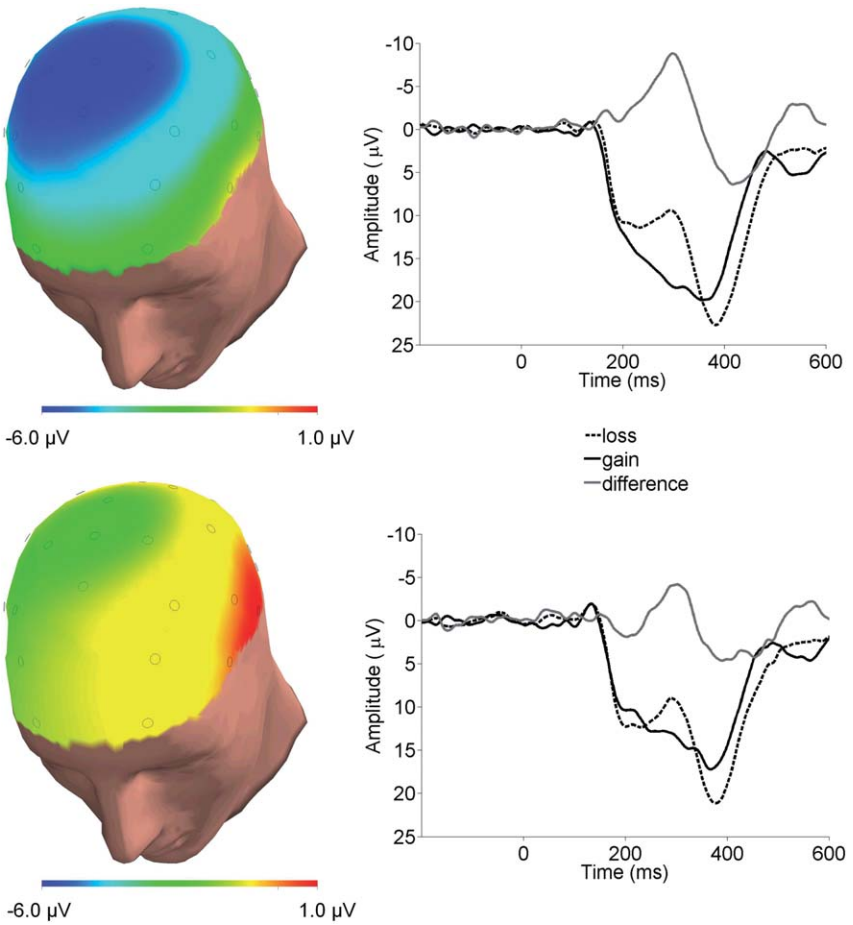

Figure 6. Scalp distribution of the difference between losses and gains from 250 to 350 ms after feedback presentation (left) and feedback-locked ERPs at a pooling of Fz and FCz electrodes in response to losses and gains, as well as the loss–gain difference (right). Results are shown for participants who did not later develop an MDE (top) and for participants who did (bottom). Reprinted from Bress, Foti, Kotov, Klein, & Hajcak (2013) with permission.

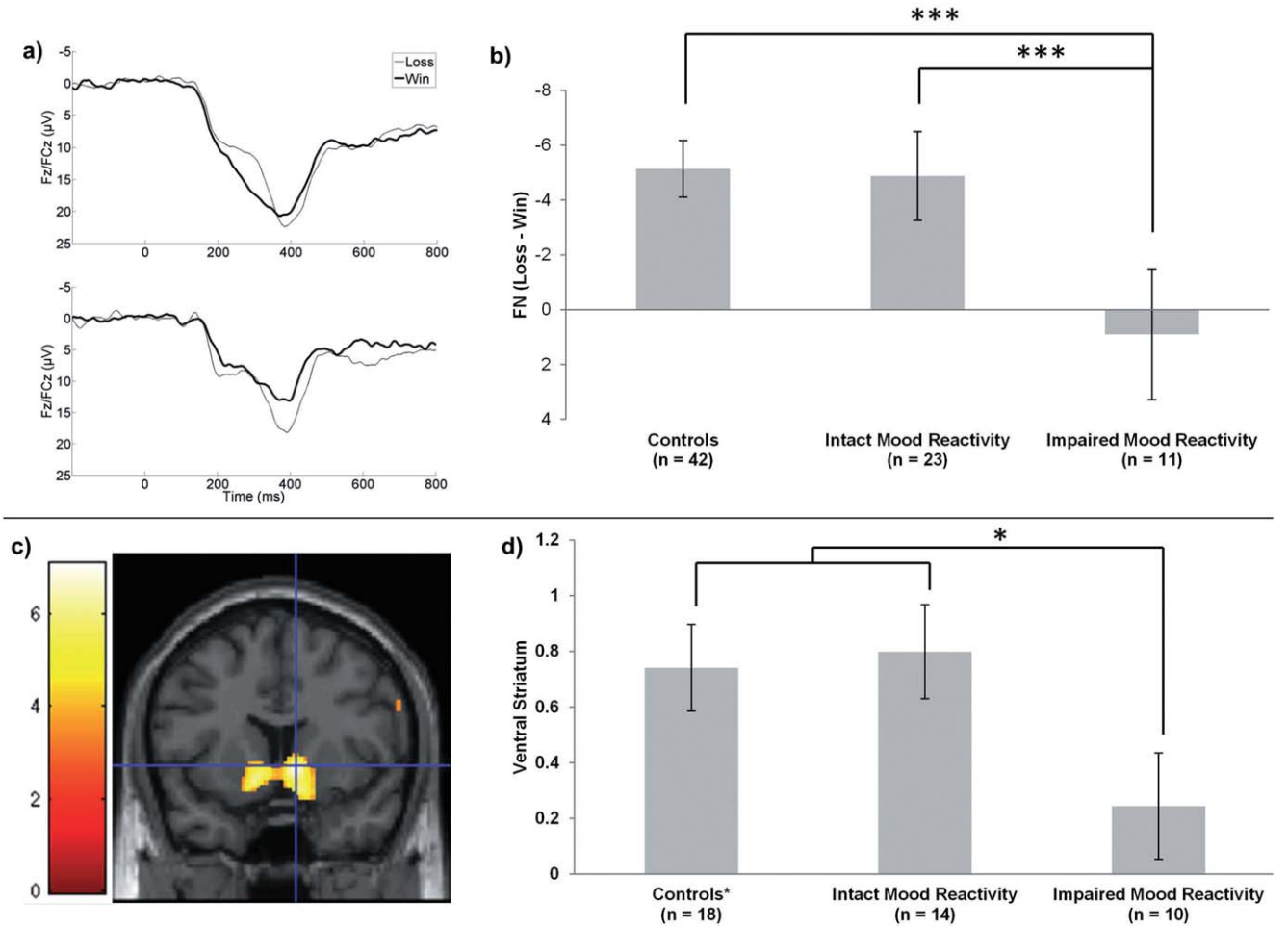

Figure 7. Reduced reward-related neural activity among depressed individuals reporting impaired mood reactivity. a: ERPs among depressed individuals with intact (top) and impaired (bottom) mood reactivity. b: Group means and standard errors for FN amplitude. c: Ventral striatal (VS) activation across the full sample (y = 10). d: Group means and standard errors for VS activation; controls include 12 nondepressed individuals not represented in part b. *$p < .05$; ***$p < .001$. Reprinted from Foti, Carlson, Sauder, & Proudfit (2014) with permission.

onciliation between research on the RewP and past research that highlights the role of the ACC in processing negative feedback (Gehring & Willoughby, 2002; Miltner et al., 1997). That is, loss-related ACC activity may contribute to the scalp-recorded ERP in the theta range. The key point here is that this activity is dissociable from gain-related neural activity in the delta range. Thus, time-frequency decompositions provide an important tool for isolating and representing reward-related neural activity and individual differences. It will be important for future studies to further utilize time-frequency analyses in studies of depression and risk.

Summary

In *Romeo and Juliet*, Juliet asks rhetorically, "What is in a name?" and goes on to argue that language does not change the referent and the fundamental experience of reality ("That which we call a rose by any other name would smell as sweet"). Having changed my own last name, I am inclined to agree with Juliet. And yet, the naming convention of ERPs is intended to be descriptive: ERP names denote information about polarity (i.e., negativity versus positivity), timing (i.e., the P300), scalp distribution (e.g., frontal slow wave), and function (e.g., error-related negativity). In this way, the fERN, MFN, FRN, and FN are all appropriate descriptions of the scalp-recorded difference between the ERP elicited by gain and loss. Referring to the reward positivity (RewP) represents a shift in the conceptualization of the underlying neural processes that give rise to scalp-recorded ERPs. Moreover, the relationship between the RewP and related measures and constructs further suggests greater precision of reward positivity.

References

Baker, T., & Holroyd, C. (2009). Which way do I go? Neural activation in response to feedback and spatial processing in a virtual T-maze. *Cerebral Cortex*, *19*, 1708–1722.

Becker, M. P. I., Nitsch, A. M., Miltner, W. H. R., & Straube, T. (2014). A single-trial estimation of the feedback-related negativity and its relation to BOLD responses in a time-estimation task. *Journal of Neuroscience*, *34*, 3005–3012. doi: 10.1523/JNEUROSCI.3684-13.2014

Bernat, E. M., Nelson, L. D., Holroyd, C. B., Gehring, W. J., & Patrick, C. J. (2008). Separating cognitive processes with principal components

analysis of EEG time-frequency distributions. *Proceedings of SPIE*, *7074*, 70740S. doi: 10.1117/12.801362

Bernat, E. M., Nelson, L. D., Steele, V. R., Gehring, W. J., & Patrick, C. J. (2011). Externalizing psychopathology and gain-loss feedback in a simulated gambling task: Dissociable components of brain response revealed by time-frequency analysis. *Journal of Abnormal Psychology*, *120*, 352–364. doi: 10.1037/a0022124

Bress, J. N., Foti, D., Kotov, R., Klein, D. N., & Hajcak, G. (2013). Blunted neural response to rewards prospectively predicts depression in adolescent girls. *Psychophysiology*, *50*, 74–81. doi: 10.1111/j.1469-8986.2012.01485.x

Bress, J. N., & Hajcak, G. (2013). Self-report and behavioral measures of reward sensitivity predict the feedback negativity. *Psychophysiology*, *50*, 610–616. doi: 10.1111/psyp.12053

Bress, J. N., Meyer, A., & Hajcak, G. (2013). Differentiating anxiety and depression in children and adolescents: Evidence from event-related brain potentials. *Journal of Clinical Child and Adolescent Psychology*, *7*, 37–41. doi: 10.1080/15374416.2013.814544

Bress, J. N., Meyer, A., & Proudfit, G. H. (in press). The stability of the feedback negativity and its relationship with depression during childhood and adolescence. *Child Development*.

Bress, J. N., Smith, E., Foti, D., Klein, D. N., & Hajcak, G. (2012). Neural response to reward and depressive symptoms in late childhood to early adolescence. *Biological Psychology*, *89*, 156–162. doi: 10.1016/j.biopsycho.2011.10.004

Carlson, J. M., Foti, D., Mujica-Parodi, L. R., Harmon-Jones, E., & Hajcak, G. (2011). Ventral striatal and medial prefrontal BOLD activation is correlated with reward-related electrocortical activity: A combined ERP and fMRI study. *NeuroImage*, *57*, 1608–1616. doi: 10.1016/j.neuroimage.2011.05.037

Cohen, M., Cavanagh, J., & Slagter, H. (2011). Event-related potential activity in the basal ganglia differentiates rewards from nonrewards: Temporospatial principal components analysis and source localization of the feedback negativity. *Human Brain Mapping*, *32*, 2270–2272.

Cronbach, L., & Meehl, P. (1955). Construct validity in psychological tests. *Psychological Bulletin*, *52*, 281–302.

Cuthbert, B. N., & Insel, T. R. (2013). Toward the future of psychiatric diagnosis: the seven pillars of RDoC. *BMC Medicine*, *11*, 126. doi: 10.1186/1741-7015-11-126

Dien, J. (2010). Evaluating two-step PCA of ERP data with Geomin, Infomax, Oblimin, Promax, and Varimax rotations. *Psychophysiology*, *47*, 170–183. doi: 10.1111/j.1469-8986.2009.00885.x

Donchin, E., & Heffley, E. F. (1978). Multivariate analysis of event-related potential data: A tutorial review. In D. Otto (Ed.), *Multidisciplinary perspectives in event-related brain potential research* (pp. 555–572). Washington, DC: U.S. Government Printing Office.

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. *Electroencephalography and Clinical Neurophysiology*, *78*, 447–455. doi: 10.1016/0013-4694(91)90062-9

Foti, D., Carlson, J. M., Sauder, C. L., & Proudfit, G. H. (2014). Reward dysfunction in major depression: Multimodal neuroimaging evidence for refining the melancholic phenotype. *NeuroImage*, *101*, 50–58.

Foti, D., & Hajcak, G. (2009). Depression and reduced sensitivity to nonrewards versus rewards: Evidence from event-related potentials. *Biological Psychology*, *81*, 1–8.

Foti, D., Weinberg, A., Bernat, E., & Proudfit, G. H. (in press). Anterior cingulate activity to monetary loss and basal ganglia activity to monetary gain uniquely contribute to the feedback negativity. *Clinical Neurophysiology*.

Foti, D., Weinberg, A., Dien, J., & Hajcak, G. (2011a). Event-related potential activity in the basal ganglia differentiates rewards from nonrewards: Temporospatial principal components analysis and source localization of the feedback negativity. *Human Brain Mapping*, *2216*, 2207–2216. doi: 10.1002/hbm.21182

Foti, D., Weinberg, A., Dien, J., & Hajcak, G. (2011b). Event-related potential activity in the basal ganglia differentiates rewards from nonrewards: response to commentary. *Human Brain Mapping*, *32*, 2267–2269. doi: 10.1002/hbm.21357

Galvan, A. (2013). The teenage brain: Sensitivity to rewards. *Current Directions in Psychological Science*, *22*, 88–93. doi: 10.1177/0963721413480859

Gehring, W. J., Goss, B., & Coles, M. G. H. (1993). A neural system for error detection and compensation. *Psychological Science*, *4*, 385–390. doi: 10.1111/j.1467-9280.1993.tb00586.x

Gehring, W. J., Liu, Y., Orr, J. M., & Carp, J. (2012). The error-related negativity (ERN/Ne). In S. J. Luck & E. S. Kappenman (Eds.), *Event related potential components* (pp. 231–291). New York, NY: Oxford University Press. doi: 10.1093/oxfordhb/9780195374148.013.0120

Gehring, W. J., & Willoughby, A. R. (2002). The medial frontal cortex and the rapid processing of monetary gains and losses. *Science*, *295*, 2279–2282. doi: 10.1126/science.1066893

Gotlib, I. H., & Goodman, S. H. (1999). Children of parents with depression. In W. K. Silverman & T. H. Ollendick (Eds.), *Developmental issues in the clinical treatment of children* (pp. 415–432). Needham Heights, MA: Allyn & Bacon.

Hajcak, G., Moser, J. S., Holroyd, C. B., & Simons, R. F. (2006). The feedback-related negativity reflects the binary evaluation of good versus bad outcomes. *Biological Psychology*, *71*, 148–154. doi: 10.1016/j.biopsycho.2005.04.001

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology*, *107*, 128–140. doi: 10.1037/0021-843X.107.1.128

Holroyd, C. B. (2004). A note on the oddball N200 and the feedback ERN. In M. Ullsperger & M. Falkenstein (Eds.), *Errors, conflicts, and the brain. Current opinions on performance monitoring* (pp. 211–218). Leipzig, Germany: MPI of Cognitive Neuroscience.

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, *109*, 679–709. doi: 10.1037//0033-295X.109.4.679

Holroyd, C. B., Hajcak, G., & Larsen, J. T. (2006). The good, the bad and the neutral: Electrophysiological responses to feedback stimuli. *Brain Research*, *1105*, 93–101. doi: 10.1016/j.brainres.2005.12.015

Holroyd, C. B., Krigolson, O. E., & Lee, S. (2011). Reward positivity elicited by predictive cues. *NeuroReport*, *22*, 249–252. doi: 10.1097/WNR.0b013e328345441d

Holroyd, C. B., Nieuwenhuis, C. A. S., Yeung, N., & Cohen, J. D. (2003). Errors in reward prediction are reflected in the event-related brain potential. *NeuroReport*, *14*, 2481–2484. doi: 10.1097/01.wnr.0000099601.41403.a5

Holroyd, C. B., Pakzad-Vaezi, K. L., & Krigolson, O. E. (2008). The feedback correct-related positivity: Sensitivity of the event-related brain potential to unexpected positive feedback. *Psychophysiology*, *45*, 688–697. doi: 10.1111/j.1469-8986.2008.00668.x

Horan, W. P., Foti, D., Hajcak, G., Wynn, J. K., & Green, M. F. (2012). Impaired neural response to internal but not external feedback in schizophrenia. *Psychological Medicine*, *42*, 1637–1647. doi: 10.1017/S0033291711002819

Ingram, R., & Luxton, D. (2005). Vulnerability-stress models. In B. L. Hankin & J. R. Z. Abela (Eds.), *Development of psychopathology: A vulnerability-stress perspective* (pp. 32–46). Thousand Oaks, CA: Sage Publications, Inc.

Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K., . . . Wang, P. (2010). Research domain criteria (RDoC): Toward a new classification framework for research on mental disorders. *American Journal of Psychiatry*, *167*, 748–751. doi: 10.1176/appi.ajp.2010.09091379

Kappenman, E., & Luck, S. (2012). ERP components: The ups and downs of brainwave recordings. In S. J. Luck & E. S. Kappenman (Eds.), *Oxford handbook of ERP components*. (pp. 3–30). New York, NY: Oxford University Press.

Keenan, K., Feng, X., Hipwell, A., & Klostermann, S. (2009). Depression begets depression: Comparing the predictive utility of depression and anxiety symptoms to later depression. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *50*, 1167–1175. doi: 10.1111/j.1469-7610.2009.02080.x

Klein, D. N., Shankman, S. A., Lewinsohn, P. M., & Seeley, J. R. (2009). Subthreshold depressive disorder in adolescents: Predictors of escalation to full-syndrome depressive disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *48*, 703–710. doi: 10.1097/CHI.0b013e3181a56606

Kujawa, A., Proudfit, G. H., & Klein, D. N. (2014). Neural reactivity to rewards and losses in offspring of mothers and fathers with histories of

depressive and anxiety disorders. *Journal of Abnormal Psychology*, *123*, 287–297. doi: 10.1037/a0036285

Kujawa, A., Proudfit, G. H., Laptook, R., & Klein, D. N. (in press). Early parenting moderates the association between parental depression and neural reactivity to rewards and losses in offspring. *Clinical Psychological Science*.

Kujawa, A., Smith, E., Luhmann, C., & Hajcak, G. (2013). The feedback negativity reflects favorable compared to nonfavorable outcomes based on global, not local, alternatives. *Psychophysiology*, *50*, 134–138. doi: 10.1111/psyp.12002

Liu, W. H., Wang, L. Z., Shang, H. R., Shen, Y., Li, Z., Cheung, E. F., & Chan, R. C. (2014). The influence of anhedonia on feedback negativity in major depressive disorder. *Neuropsychologia*, *53*, 213–220. doi: 10.1016/j.neuropsychologia.2013.11.023

Miltner, W. H. R., Braun, C. H., & Coles, M. G. H. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a "generic" neural system for error detection. *Journal of Cognitive Neuroscience*, *9*, 788–798. doi: 10.1162/jocn.1997.9.6.788

Nieuwenhuis, S., Slagter, H. A., von Geusau, N. J. A., Heslenfeld, D. J., & Holroyd, C. B. (2005). Knowing good from bad: Differential activation of human cortical areas by positive and negative outcomes. *European Journal of Neuroscience*, *21*, 3161–3168. doi: 10.1111/j.1460-9568.2005.04152.x

Nieuwenhuis, S., Yeung, N., Holroyd, C. B., Schurger, A., & Cohen, J. D. (2004). Sensitivity of electrophysiological activity from medial frontal cortex to utilitarian and performance feedback. *Cerebral Cortex*, *14*, 741–747. doi: 10.1093/cercor/bhh034

Patrick, C. J., & Bernat, E. M. (2010). Neuroscientific foundations of psychopathology. In T. Milton, R. F. Krueger, & E. Simonsen (Eds.), *Contemporary directions in psychopathology: Scientific foundations of the DSM-V and ICD-II* (pp. 419–452). New York, NY: Guilford Press.

Patrick, C. J., Venables, N. C., Yancey, J. R., Hicks, B. M., Nelson, L. D., & Kramer, M. D. (2013). A construct-network approach to bridging diagnostic and physiological domains: Application to assessment of externalizing psychopathology. *Journal of Abnormal Psychology*, *122*, 902–916.

Sanislow, C. A., Pine, D. S., Quinn, K. J., Kozak, M. J., Garvey, M. A., Heinssen, R. K., . . . Cuthbert, B. N. (2010). Developing constructs for psychopathology research: Research domain criteria. *Journal of Abnormal Psychology*, *119*, 631–639. doi: 10.1037/a0020909

Shankman, S., & Klein, D. (2003). The relation between depression and anxiety: An evaluation of the tripartite, approach-withdrawal and valence-arousal models. *Clinical Psychology Review*, *23*, 605–637.

Spencer, K., Dien, J., & Donchin, E. (2001). Spatiotemporal analysis of the late ERP responses to deviant stimuli. *Psychophysiology*, *38*, 343–358.

Tversky, A., & Kahneman, D. (1992). Advances in prospect theory: Cumulative representation of uncertainty. *Journal of Risk and Uncertainty*, *5*, 297–323.

Umemoto, A., Lukie, C., & Kerns, K. (2014). Impaired reward processing by anterior cingulate cortex in children with attention deficit hyperactivity disorder. *Cognitive, Affective, & Behavioral Neuroscience*, *14*, 698–714.

Van den Berg, I., Franken, I. H. A., & Muris, P. (2010). A new scale for measuring reward responsiveness. *Frontiers in Psychology*, *1*, 239. doi: 10.3389/fpsyg.2010.00239

Van Leijenhorst, L., Moor, B. G., Op de Macks, Z. A., Rombouts, S. A. R. B., Westenberg, P. M., & Crone, E. A. (2010). Adolescent risky decision-making: Neurocognitive development of reward and control regions. *NeuroImage*, *51*, 345–355. doi: 10.1016/j.neuroimage.2010.02.038

Van Meel, C. S., Heslenfeld, D. J., Oosterlaan, J., Luman, M., & Sergeant, J. A. (2011). ERPs associated with monitoring and evaluation of monetary reward and punishment in children with ADHD. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *52*, 942–953. doi: 10.1111/j.1469-7610.2010.02352.x

Weinberg, A., Luhmann, C. C., Bress, J. N., & Hajcak, G. (2012). Better late than never? The effect of feedback delay on ERP indices of reward processing. *Cognitive, Affective, & Behavioral Neuroscience*, *12*, 671–677.

Weinberg, A., Riesel, A., & Proudfit, G. H. (2014). Show me the money: The impact of actual rewards and losses on the feedback negativity. *Brain and Cognition*, *87*, 134–139. doi: 10.1016/j.bandc.2014.03.015

Yeung, N., Holroyd, C. B., & Cohen, J. D. (2005). ERP correlates of feedback and reward processing in the presence and absence of response choice. *Cerebral Cortex*, *15*, 535–544. doi: 10.1093/cercor/bhh153

(Received July 15, 2014; Accepted September 1, 2014)