Performance Monitoring in the Anterior Cingulate is Not All Error Related: Expectancy Deviation and the Representation of Action–Outcome Associations

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Abstract

■ Several converging lines of evidence suggest that the anterior cingulate cortex (ACC) is selectively involved in error detection or evaluation of poor performance. Here we challenge this notion by presenting event-related potential (ERP) evidence that the feedback-elicited error-related negativity, an ERP component attributed to the ACC, can be elicited by positive feedback when a person is expecting negative feedback and vice versa. These results suggest that performance monitoring in the ACC is not limited to error processing. We propose that the ACC acts as part of a more general performance-monitoring system that is activated by violations in expectancy. Further, we propose that the common observation of increased ACC activity elicited by negative events could be explained by an overoptimistic bias in generating expectations of performance. These results could shed light into neurobehavioral disorders, such as depression and mania, associated with alterations in performance monitoring and also in judgments of self-related events. ■

INTRODUCTION

Errors are an integral part of human life, a fact perhaps best described by the words of Melchior de Polignac: *Errare humanum est* (To err is human). Fittingly, strong interest has been devoted to the study of the neural basis of performance monitoring and error detection. This has been facilitated by the discovery of the error-related negativity (ERN), a component of the event-related brain potential (ERP) elicited when people commit errors and when feedback about such errors is provided (Gehring, Goss, Coles, Meyer, & Donchin, 1993; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990; for a review, see Nieuwenhuis, Holroyd, Mol, & Coles, 2004). The ERN is a negative-going voltage measured over the fronto-central scalp that appears to reflect increased activation in the anterior cingulate cortex (ACC) (Miltner, Braun, & Coles, 1997; Dehaene, Posner, & Tucker, 1994). This is supported by converging lines of evidence from functional magnetic resonance imaging (fMRI) (Holroyd, Nieuwenhuis, Yeung, et al., 2004; Kiehl, Liddle, & Hopfinger, 2000), magnetoencephalography (Miltner et al., 2003), and single-cell recordings (Wang, Ulbert, Schomer, Marinkovic, & Halgren, 2005; Niki & Watanabe, 1979), suggesting that the ACC is critically involved in performance monitoring and error detection.

Currently, there are three primary views of how performance monitoring leads to increased ACC activity. One view is that the ACC acts as part of a dedicated error-detection system that is activated by information signaling errors in performance (Miltner et al., 1997). An alternative view is that the ACC acts as part of a conflict-monitoring system that is activated by response competition rather than error detection per se (Botvinick, Cohen, & Carter, 2004; Yeung, Cohen, & Botvinick, 2004; Botvinick, Braver, Barch, Carter, & Cohen, 2001). A third view is that the ACC acts as part of a reward-prediction system that is activated when events are worse than expected (Holroyd & Coles, 2002).

Although the current views of ACC function link ACC activity to different types of performance-monitoring processes, they all predict increased ACC activity, and thus, increased ERN amplitude, to occur only when performance is poor or when response conflict is present. However, positive feedback can elicit a small negative ERP deflection that is similar to the feedback-elicited ERN (Hajcak, Holroyd, Moser, & Simons, 2005; Müller, Moller, Rodriguez-Fornells, & Munte, 2005; Yeung, Holroyd, & Cohen, 2005; Yeung & Sanfey, 2004). Furthermore, evidence from fMRI and single-cell recordings suggests that ACC activity is not elicited only in response to errors, but also in response to rewards (Walton, Devlin, & Rushworth, 2004; Ito, Stuphorn, Brown, & Schall, 2003; Akkal, Bioulac, Audin, & Burbaud, 2002; Niki & Watanabe, 1979). These findings indicate that the feedback-elicited ERN and the performance-monitoring functions of the ACC may not be directly related to error processing or the evaluation of poor performance.

Here we tested whether increased ACC activity, as measured by the feedback-elicited ERN, can be elicited

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when performance is good. Specifically, we predicted that positive as well as negative feedback would elicit a negative ERP component comparable to the ERN when the performance indicated by feedback is unexpectedly good or bad. We refer to this component as the *feedback-related negativity* (FRN) (Luu, Tucker, Derryberry, Reed, & Poulsen, 2003) because it might not be restricted to errors. The driving hypothesis was that the ACC acts as part of a system that detects deviations from expectancy rather than a system that detects errors or performance that is worse than expected. According to this *expectancy-deviation hypothesis*, the FRN is an outcome of a monitoring system that compares the participant's expected feedback to the actual feedback and is elicited when a mismatch between the two is detected. We also hypothesized that FRNs elicited by negative feedback are typically larger than those elicited by positive feedback because humans tend to be overly optimistic when judging their performance (Miller & Ross, 1975). Such a bias would result in a larger proportion of trials on which negative feedback followed positive self-evaluations than vice versa, hence, a larger FRN in the negative feedback condition.

EXPERIMENT 1

To test the expectancy-deviation hypothesis for feedback-related ACC activity, we asked participants to judge their own performance on each trial after making a response and before receiving feedback. Participants performed an anticipation timing task that involved pressing a button when a horizontally moving visual stimulus reached a prespecified target location. Immediately following their response, participants estimated whether they thought they responded early, late, or on target by pressing a corresponding button. The trial ended with the presentation of feedback indicating the actual performance (Figure 1A). We used participants' estimations as indices of their expectancies and computed ERPs separately for trials on which the expectancies matched the feedback or differed from it. We predicted that large FRNs would be elicited by feedback that mismatched the participants' expectancies and small FRNs would be elicited by feedback that matched the participants' expectancies, irrespective of the valence of the feedback. Furthermore, we predicted that participants would overestimate the number of trials they responded correctly and underestimate the number of trials they responded erroneously, consistent with an overoptimistic bias.

Methods

Participants

Fourteen volunteers (7 men, mean age = 22.1 years, range = 19–28 years) participated in Experiment 1. Participants were all healthy, right-handed, experiment naive, and were paid to participate after giving informed written consent. Data from one participant were excluded from analyses due to excessive noise in the electroencephalogram (EEG).

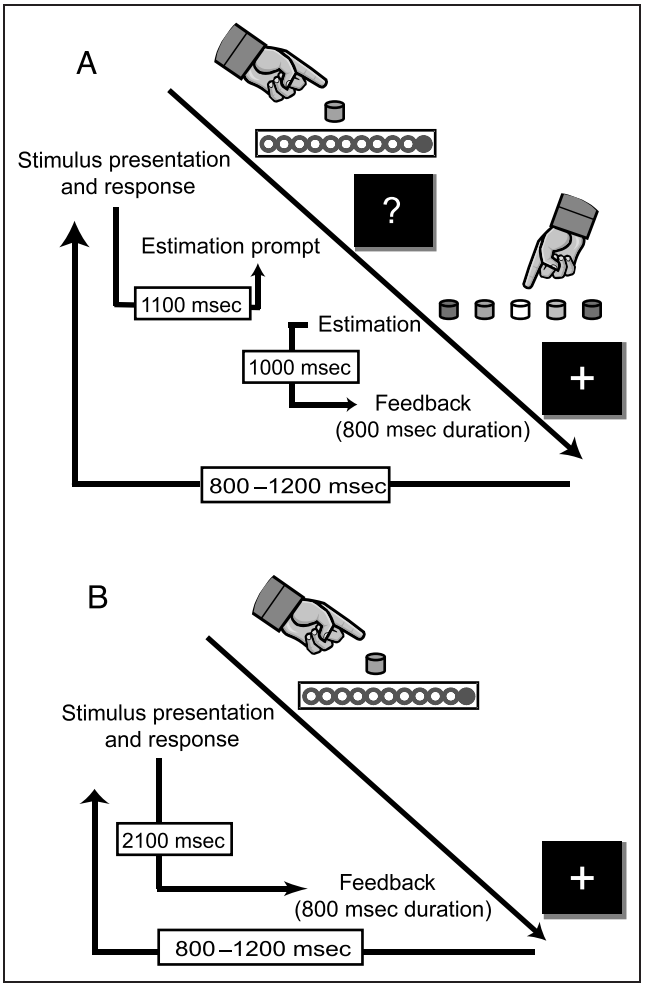

Figure 1. Schematic illustration of the sequence of events on each trial. (A) Experiment 1. Participants performed an anticipation-timing task that involved pressing a button when a moving visual stimulus reached a target location. Following their response, participants were prompted to estimate their performance and then received feedback about the performance. (B) Experiment 2. Participants performed the same anticipation-timing task as in Experiment 1 but did not have to estimate their performance. Instead, participants received feedback directly following their response.

Apparatus and Procedure

Participants sat in a dark room and faced a computer monitor and a light display consisting of 24 light-emitting diodes (LEDs) mounted side-to-side 3 cm apart from each other. Either the leftmost or rightmost LED was illuminated throughout a block of trials to serve as the target endpoint. On each trial, the first 12 LEDs on the opposite end of the target endpoint were illuminated sequentially in the direction of the target endpoint. This gave the impression of a moving visual stimulus that disappeared

prior to reaching the target. The participants' primary task was to press a mouse button with their left hand when the moving stimulus reached the target endpoint. A question mark then appeared on the computer screen to prompt the participants to estimate their performance by pressing one of five buttons on an estimation panel using their right hand. The buttons represented: "I am very confident that I responded early," "I think I responded early," "I think I responded on target," "I think I responded late," and "I am very confident that I responded late." After the estimation, participants received feedback about their performance signaling that they had responded early ("−"), late ("+"), or on target ("0") (Figure 1A). There were four different velocities for the moving stimulus. The time between the onset of the first LED in the sequence and the target time was either 480, 600, 720, or 840 msec (representing velocities of 144, 115, 96, and 82 cm/sec, respectively). Two velocities were randomly selected for a given block of trials. The direction of the moving stimulus was randomly assigned to each block and remained constant throughout the trials of the block. The experimental session consisted of 17 blocks of 50 trials with self-controlled rest breaks every 25 trials. During trials participants were asked to blink as little as possible and were instructed not to move their eyes by fixating their vision on a marked central location on the computer screen.

Feedback was based on the following procedure. Error magnitude was computed on-line as the time difference between the response and the time the moving stimulus reached the target endpoint (target time). Responses were considered *on-target* (i.e., correct) if the error fell within a tolerance window centered on the target time and *off-target* (i.e., erroneous) if they fell outside of this window. The tolerance window was initially 250 msec; that is, responses were considered on-target if they occurred within 125 msec of the illumination of the last LED. It decreased by a factor of 0.85 after every on-target response, and increased by a factor of 1.10 after every off-target response. The adaptive procedure stopped once the window size decreased below 120 msec, which occurred, on average, after 29 trials, remaining constant to the end of the experiment.

During initial practice, participants were instructed on how to perform the task and were told that their performance estimation was being used only as an index of how they thought they were doing. It was stressed that their primary task was to respond to the moving stimuli, and that they would be evaluated on this task. They were further instructed that every time they responded on-target they would win a bonus of 4 cents and every time they responded early or late they would lose 4 cents—this procedure was only motivational as participants would unknowingly receive the same bonus at the end of the testing session. All experimental procedures were approved by the university ethics board.

Behavioral Recording and Measures

We calculated the percentage of trials participants responded on-target, early, and late, as well as the percentage of trials that participants estimated their responses to be on-target, early, and late, and the percentage of trials in each condition that were accurately estimated. Due to limited number of trials in each condition, we collapsed "I am very confident that I responded early (or late)" with "I think I responded early (or late)" estimations, such that we only used three levels of estimation (i.e., early, on-target, and late). We further used paired *t* tests to compare the percentage of trials that participants responded early and late, and the percentage of trials that participants estimated as early and late, and found no differences ($p > .2$). Given this, we collapsed the two conditions into a single error condition.

Electrophysiological Recording and Measures

EEG activity was recorded using tin electrodes placed at FPz, Fz, FCz, Cz, Pz, Oz, and the left mastoid. An electrode on the right mastoid was used as reference for all of these recordings. Electrodes were also placed near the external canthi of both eyes to record the horizontal electrooculogram (EOG). Electrode impedances were kept below 10 kΩ. EEG and EOG signals were amplified by a gain of 20,000 and a band pass of 0.1–80 Hz, and were digitized at 500 Hz. Automated artifact rejection was done off-line to discard trials contaminated by eye movements and blinks, or when amplifier blocking occurred. The EEG was averaged in 1000 msec epochs (200 msec baseline) time-locked to the feedback stimulus. These averages were digitally low-pass filtered at 20 Hz and re-referenced to the average of the mastoids.

We measured FRN amplitude base-to-peak at electrodes Fz, FCz, and Cz, where FRN is reported to be maximal, according to the following procedures: First, we identified the most positive peak 160–270 msec following feedback presentation. We then found the most negative peak that followed this positivity in a window extending to 350 msec after feedback onset, and the most positive peak following the negativity up to 500 msec from feedback presentation. Peaks were defined as samples that were more positive/negative than the preceding and following samples and of the average of the five preceding and following samples. FRN amplitude was quantified as the voltage difference between the negative-going peak and the average of the preceding and following positive-going peaks (Yeung & Sanfey, 2004). If no negative-going peaks were found in the time window defined above, the ERP component was defined as a positivity and FRN amplitude was considered 0 μV. The same patterns of results were found for early and late responses in a preliminary analysis; therefore, we combined these trials to create a general error condition in the analysis reported here.

Statistical Analysis

We used two-tailed paired-samples t tests for all behavioral analyses. FRN analysis was done through a 3 (electrode: Fz, FCz and Cz) × 2 (mismatch: present and absent) × 2 (feedback valence: positive or negative) repeated-measures ANOVA. Whenever a violation of the assumption of sphericity was detected, we used the Greenhouse–Geisser correction of degrees of freedom, and to break down any significant main effects, we performed uncorrected pairwise comparisons.

Results

Overoptimistic Bias

As predicted, participants were overly optimistic about their performance (Figure 2). Not only did they judge their responses to be correct (on-target) more often than erroneous [early or late; $t(12) = 5.88, p < .0001$] but they also judged their responses to be correct more often than was actually the case [$t(12) = 5.55, p < .001$] and to be erroneous less often than was actually the case [$t(12) = 5.55, p < .001$]. Moreover, participants accurately estimated correct performance more often than erroneous performance [$t(12) = 7.38, p < .00001$]. As a result, the proportion of erroneous trials with mismatches between expected and actual feedback was larger than the proportion of correct trials with mismatches between expected and actual feedback.

Expectancy Deviation Elicits FRN

We first examined the ERP waveforms elicited in response to positive and negative feedback without regard to the estimated performances. Consistent with prior work, we found an FRN 200–350 msec after the onset of negative feedback and a smaller FRN 200–350 msec after the onset of positive feedback (Figure 3A). To test the predictions stemming from the expectancy-deviation hypothesis, we next sought to determine whether such negative-going ERP deflections were present when participants received positive or negative feedback that mismatched their expectation. As hypothesized, the FRN was larger when the expected feedback and actual feedback mismatched (5.6 μV) than when they matched (2.7 μV), regardless of the type of feedback that was received [$F(1, 12) = 26.44, p < .001$]. Moreover, the absence of a significant feedback-valence main effect [$F(1, 12) < 1$] showed that FRNs elicited to positive (4.4 μV) and negative (4.0 μV) feedback were no different when collapsed over whether there was a match or a mismatch between the expected and actual outcome.[1]

FRNs differed in amplitude across the electrodes of interest [$F(2, 24) = 6.39, p < .05; \varepsilon = .69$]. FRN amplitude was smaller at Cz than at Fz and FCz ($p < .05$ for both comparisons) and not reliably different at Fz and FCz ($p > .1$).

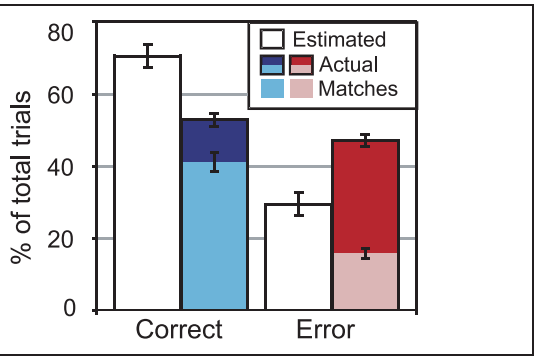

Figure 2. Estimated and actual performance. White bars show percentages of trials on which participants estimated their response to be correct or erroneous, and colored bars show percentages of trials on which responses were actually correct or erroneous. Light and dark colors denote the percentages of trials on which the estimated and actual outcomes matched or mismatched, respectively. Matches represent 77.9% of correct trials and 33.6% of erroneous trials. The standard error of the mean is shown at the top of each section.

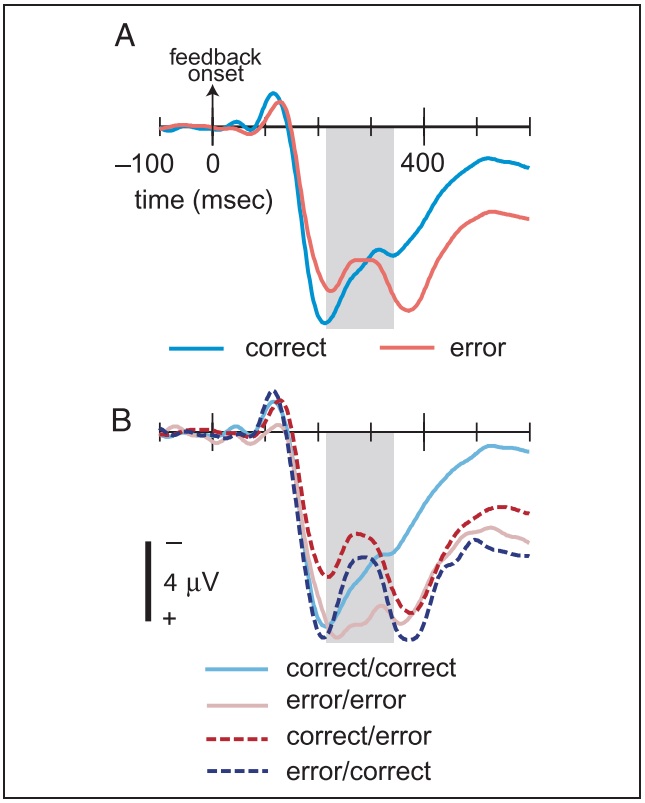

Figure 3. Grand-average ($n = 13$) ERP waveforms recorded from electrode Fz, time-locked to feedback onset. (A) ERPs plotted separately for error and correct feedback, (B) ERPs plotted separately for expected/actual feedback pairs. Dark-dashed lines and light-solid lines represent conditions with mismatches and matches between expected and actual feedback, respectively. The colors match those of Figure 2. The gray shaded area represents the range in which the FRN peaks were calculated. The FRN is seen as the negative-going deflection observed within the shaded area. Negative voltages are plotted up.

Summary. Negative feedback elicited a large FRN when participants estimated they had responded correctly but not when they estimated they had responded erroneously (Figure 3B). This result was predicted by the expectancy-deviation hypothesis, which suggests that FRNs are the result of a mismatch between the expected and actual feedback, and with the reinforcement-learning theory, which suggests that FRNs occur only when events are worse than expected (Holroyd & Coles, 2002). The ERPs elicited by positive feedback, however, were consistent with only the expectancy-deviation hypothesis. Specifically, positive feedback elicited a large FRN when participants estimated their performance to be erroneous but not when they estimated their performance to be correct.

EXPERIMENT 2

In Experiment 2, we addressed an alternative (error-related) account of the results obtained in Experiment 1. Although the results of Experiment 1 suggested that the FRN is not error-specific, Experiment 1 had a potential confound in the fact that participants were instructed to estimate their performance prior to receiving feedback. This could have influenced participants' perception of the goal of the task. It is possible that participants considered an error in estimation, and not an error in the primary task, to ultimately constitute an error. To test this possibility, we eliminated performance estimation as part of the task and concentrated on trials on which responses were considerably off-target (Figure 1B). We assumed that participants would be aware of most of these large errors and would thus expect to receive negative feedback on the majority of these large-error trials. To violate participants' expectancies, we provided false-positive feedback on half of the large-error trials.

If the requirement that participants overtly estimated their performance was responsible for the FRN to positive feedback in Experiment 1, then false-positive feedback would not elicit any FRN in Experiment 2. We hypothesized that if expectancy deviation is the main factor in the generation of the FRN, then false-positive feedback would elicit a large FRN on large-error trials.

Methods

Participants

Thirteen volunteers (9 men, mean age = 25.1 years, range = 20–35 years) participated in Experiment 2. Participants were all healthy, right-handed, experiment naive, and were paid to participate after giving informed written consent. Data from one participant were excluded from analyses due to unusually poor performance. This participant's behavioral performance (as measured by root mean square error) deviated by more than 8 standard deviations from the group mean.

Apparatus and Procedures

The apparatus and procedure were the same as those for Experiment 1 with the following exceptions. All 24 LEDs were illuminated to provide participants with full vision of the moving visual stimulus. The time between the onset of the first LED in the sequence and the target time was either 432, 600, 768, or 936 msec (representing velocities of 160, 115, 90, and 74 cm/sec, respectively). Participants did not have to estimate their performance (Figure 1B). The adaptive procedure for determining feedback stopped when the window decreased below 80 msec, which occurred, on average, after 24 trials. Participants performed 15 blocks of 60 trials. We presented participants with feedback according to their performance on every trial of the first three blocks. On the remaining 12 blocks, there was a .5 probability that participants would receive a "0" as the feedback instead of "−" for early responses and "+" for late responses that were off-target by more than 1.8 times the tolerance window. If participants did not have at least 10 trials with an error greater than 1.8 times, the tolerance window by the end of Block 6, false feedback was at that point on presented with a probability of .5 whenever the error was greater than 1.5 times the tolerance window. This adjustment was necessary for two participants. To make the analyses consistent across participants and to ensure that the error was substantive, false feedback in trials that had an error of less than 90 msec was excluded from all statistical analyses. The reason for providing false feedback only on half of the large-error trials was such that the participants would not disbelieve the feedback.

All experimental procedures were approved by the university ethics board.

Electrophysiological Recording and Measures

EEG was recorded and averaged using similar procedures as those described for Experiment 1. To calculate the FRN amplitude, we measured the first positive peak between 150 and 290 msec from feedback onset. The negative peak was measured following this positivity up to 380 msec after feedback onset, and the second positive peak was measured following the negativity up to 500 msec after feedback onset. In Figure 4, the grand-average ERP was synchronized to each participant's FRN following the procedures used by Frank, Woroch, and Curran (2005). This latency-compensation procedure (Picton et al., 2000) was used because between-participant variability in the latency to FRN peak caused smearing of the FRN when the waveforms were synchronized to the onset of feedback. Four of the participants did not show an FRN in response to true positive feedback. Given this absence of an FRN peak, we synchronized these participants' true positive feedback waveforms using the time of the FRN peak for the false-positive feedback. Qualitatively similar ERPs were obtained when synchronizing these partic-

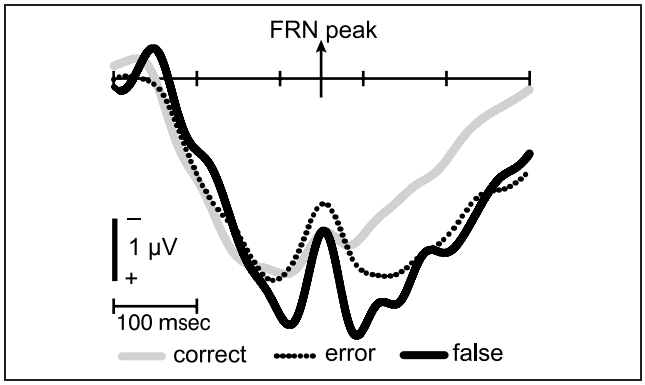

Figure 4. Grand-average (n = 12) ERP waveforms recorded from electrode Fz, time-locked to feedback onset and synchronized to each participant's FRN (see Experiment 2: Methods). ERPs are plotted separately for feedback indicating errors, correct responses, and false feedback indicating correct responses in erroneous trials. The arrow indicates the FRN peak. Negative voltages are plotted up.

ipants' waveforms to the onset of feedback, and also when excluding the four participants from the grand average.

Statistical Analysis

FRN analysis was done through a 3 (electrode: Fz, FCz, and Cz) × 3 (feedback: positive, negative, and false positive) repeated-measures ANOVA. All other statistical procedures followed those of Experiment 1.

Results

False-positive Feedback Elicits FRN

As predicted, we found significant differences in the amplitude of the FRN across the different types of feedback [$F(2, 22) = 9.71$, $p < .005$, $\varepsilon = .68$] showing that false-positive feedback presented after participants made large errors (2.5 μV) elicited a larger FRN than true positive feedback (1.2 μV; $p < .005$). The FRNs elicited by negative feedback (1.8 μV) were smaller than those elicited by false-positive feedback ($p = .04$) but larger than those elicited by true positive feedback ($p < .01$) (Figure 4). FRNs did not differ in amplitude across the electrodes of interest [$F(2, 22) < 1$].

Summary. Experiment 2 provided converging evidence for the expectancy-deviation hypothesis without the requirement that participants overtly estimate their performance. False-positive feedback after erroneous trials elicited a significantly larger FRN than true positive feedback and than negative feedback.

Discussion

In the present study, increased ACC activity, as measured by the FRN, was elicited by *positive* and negative feedback, whenever the valence of the feedback mismatched participants' estimated performance. Little or no FRN was observed when the feedback matched the participants' estimated performance, even when the feedback indicated that an error had been made. These results indicate that the FRN is not specifically related to errors or poor performance. We suggest that the FRN is an outcome of a more general performance-monitoring system that detects violations of expectancies (Luu & Pederson, 2004; Pritchard, Shappell, & Brandt, 1991).

Expectancy Deviation and Previous Findings

At the outset, we predicted that the observation that FRNs elicited by negative feedback are typically larger than FRNs elicited by positive feedback was due to the tendency for people to be overly optimistic when judging their own performance (Miller & Ross, 1975). We found evidence for such an overoptimistic bias. As a consequence, there were three times more mismatches between the expected feedback and actual feedback for erroneous trials than for correct trials (Figure 2). This resulted in a larger FRN to negative feedback than to positive feedback when the ERPs were averaged without regard for the participants' expectations (Figure 3A), but it obscured the fact that positive feedback elicited a similarly large FRN when the actual feedback and the expected feedback mismatched (Figure 3B).

The expectancy-deviation hypothesis also accounts for some previous findings that are incompatible with present theories of the FRN and ACC function. For example, whereas present theories predict FRNs and ACC activity to be elicited only by feedback of negative valence, feedback of positive valence has been shown to elicit small FRNs (Hajcak et al., 2005; Müller et al., 2005; Yeung et al., 2005; Yeung & Sanfey, 2004). Moreover, increased ACC activity in response to rewards has been observed with fMRI in humans (Walton et al., 2004) and with single-cell recordings in nonhuman primates (Ito et al., 2003; Matsumoto, Suzuki, & Tanaka, 2003; Akkal et al., 2002; Niki & Watanabe, 1979). More importantly, single-cell recordings in monkeys have shown ACC neurons to be responsive to *unexpected* rewards (Ito et al., 2003), and single-cell recordings in humans have shown ACC neurons to be responsive to *increases* in rewards (Williams, Bush, Rauch, Cosgrove, & Eskandar, 2004).

Additionally, the FRN has been found to be largely reduced or absent when participants have reliable information about the error prior to the presentation of feedback (de Bruijn, Mars, & Hulstijn, 2004; Holroyd & Coles, 2002). This finding led Holroyd and Coles (2002) to propose that the ERN is produced upon the first detection of a prediction error that occurs when consequences of actions are worse than expected. Our evidence is consistent with the notion that the FRN is elicited by the first evidence of a prediction error but suggests that the FRN is not elicited when events are *worse* than expected, but rather when events are simply *different* than expected.

This view is corroborated by the results of Müller et al. (2005), which showed that feedback that did not provide any information about performance or reward, but was unexpected, elicited an FRN that was thrice the amplitude of the FRN elicited by negative feedback. Corroboration also comes from single-cell recordings in monkeys and humans showing that ACC neurons were activated by the presentation of an infrequent form of feedback that indicated standard reward but required a change in response (Amiez, Joseph, & Procyk, 2005; Williams et al., 2004).

At first glance, the expectancy-deviation hypothesis seems incompatible with the results of previous studies that used guessing and gambling tasks to investigate how expectation of feedback modulates the amplitude of the FRN. Negative feedback elicited larger FRNs compared to positive feedback when the probability of each feedback was equal (Yeung et al., 2005), and also when contrasting FRNs when the probabilities of each positive and negative feedback were low (Hajcak et al., 2005; Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003). However, in guessing and gambling tasks, positive and negative feedback are presented randomly at predetermined frequencies and it is possible that participants are biased to expect rewards regardless of whether rewards or penalties are more likely overall (Gilovich, 1983). This would be analogous to the case of lottery players, who generate expectations of winning every lottery draw despite knowing the odds of winning are minimal.

Here we used a task that enabled participants to generate expectations of feedback based on their performance of the task and we directly measured their expectation. This procedure enabled us to examine FRNs to expected and unexpected negative and positive feedback separately. One caveat, however, is that participants may have perceived an error to have occurred whenever feedback did not match their own evaluation of performance. This alternative is unlikely for three reasons. First, participants were told that the estimation task was merely used to monitor their perception of accuracy and that they were evaluated on their responses to the moving stimuli. Second, to emphasize the valence of the feedback, we told participants they would earn money whenever they received positive feedback and would lose money whenever they received negative feedback. Finally, we observed similar FRNs to positive feedback in the absence of the performance-estimation requirement when we provided false-positive feedback after erroneous trials in Experiment 2.

Expectancy Deviation and Current Theories of the FRN and ACC Function in Performance Monitoring

The expectancy-deviation hypothesis is consistent with error, conflict, and reinforcement-learning accounts of the ERN and of ACC function, only in revised form. Expectancy deviation can be seen as a form of error (an error in expectancy, or a meta-error), of conflict (conflict between internal and external information about performance), and of reinforcement learning (reinforcement of the internal model used to generate estimations of performance). However, these accounts would have to be expanded to explain the present results. To date, error-related accounts of the ERN have focused on errors in performance (Miltner et al., 1997). According to conflict-monitoring theory, conflict at the response level triggers the ERN (Yeung et al., 2004; van Veen, Cohen, Botvinick, Stenger, & Carter, 2001), whereas according to reinforcement-learning theory, reward-prediction errors are the trigger to the response-elicited ERN and the FRN (Holroyd & Coles, 2002).

A Mechanism for Increased ACC Activity in Performance Monitoring

A possible mechanism for the generation of the ERN is through the interaction between the ACC and the mesencephalic dopamine system (Holroyd & Coles, 2002). However, given dopamine's complicated modulatory function (Seamans & Yang, 2004), it is unclear how it influences the ACC in performance monitoring. One possibility is that dopamine tonically inhibits ACC neurons, and phasic decreases in dopaminergic activity allow for a disinhibition that would give rise to the ERN (Holroyd & Coles, 2002).

Although this view seems at odds with the present results, the response of neurons to dopamine is dependent on the neurons' state of activity (Lavin & Grace, 2001; Lewis & O'Donnell, 2000; Yang & Seamans, 1996). Thus, albeit unlikely, the possibility exists that dopamine has an excitatory effect in the case of unpredicted errors and an inhibitory effect in the case of unpredicted correct responses. Another possibility is that, in humans, a higher-level reward-prediction error influences the dopamine system in similar ways (phasically decreasing its activity) whether the error is represented by the inability to predict a negative event or the inability to predict a reward.

A more plausible alternative, however, is that the mesencephalic dopamine system has an excitatory effect in the generation of the FRN. It has recently been proposed that the short-latency phasic response observed in the dopamine system is not specifically related to rewards, but is rather related to a more general process of switching attention to unexpected, behaviorally relevant stimuli [including, but not restricted to, rewards] (Redgrave & Gurney, 2006; Horvitz, 2000; Redgrave, Prescott, & Gurney, 1999). Based on this attentional-gating view of the dopamine system (and the FRN), we speculate that feedback indicating unexpectedly good or bad performance elicits a phasic increase in the activity of mesencephalic dopamine neurons which, in turn, induces increased excitability in the ACC (Lavin et al., 2005; Seamans & Yang, 2004; Seamans, Nogueira, & Lavin, 2003; Lewis & O'Donnell, 2000), or perhaps

''sharpens'' or ''focuses'' the effects of synaptic input to ACC neurons (Yang & Seamans, 1996; Servan-Schreiber, Printz, & Cohen, 1990), thereby giving rise to the FRN. This view is supported by single-cell recordings showing that dopamine neurons are activated by salient stimuli that signal the need for behavioral change regardless of the valence of the stimuli (Horvitz, 2000; also see Ungless, 2004; Ungless, Magill, & Bolam, 2004), by evidence from microdialysis showing that unexpected aversive stimuli increase extracellular dopamine levels (Young, 2004) and by fMRI studies showing that both the striatum and the ACC are activated by unexpected nonrewarding stimuli (Zink, Pagnoni, Chappelow, Martin-Skurski, & Berns, 2006; Davidson et al., 2004; Zink, Pagnoni, Martin-Skurski, Chappelow, & Berns, 2004).

Expectancy Deviation, Learning, and the Representation of Action–Outcome Associations

The expectancy-deviation account of the FRN can be seen as a mechanism for error detection, learning, and adaptive behavior. We propose that the ACC acts as part of a system that drives learning by identifying errors in the internal representation used in comparison with intrinsic task-related information to generate predictions of success and failure. This would signal an increased need for cognitive control (Brown & Braver, 2005; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004; Botvinick et al., 2001) to allow an update in the internal model of ''what to do'' and ''what not to do'' (Cohen, Aston-Jones, & Gilzenrat, 2004; Miller & Cohen, 2001; Braver & Cohen, 2000), as well as improve the use of external information to judge performance.

Indeed, Matsumoto and Tanaka (2004) and Rushworth, Walton, Kennerley, and Bannerman (2004) have proposed that the ACC plays a role in the representation of action–outcome associations even when the outcome is a reward. The ACC may act as part of a generic performance-monitoring system that is sensitive to a multitude of inputs (Ridderinkhof, van den Wildenberg, et al., 2004; Bush et al., 2002; Bush, Luu, & Posner, 2000), linked by their unexpected behavioral saliency (see above), which signal the need for adjustments to achieve intended consequences. According to this view, an evaluative signal from the mesencephalic dopamine system activates the ACC (Holroyd, Nieuwenhuis, Mars, & Coles, 2004; Holroyd & Coles, 2002). ACC activity then signals other brain regions the increased need for (physical or cognitive) resources to: (i) seek behaviors that lead to positive consequences or avoid adverse consequences immediately, or (ii) to improve the chances of generating behaviors that lead to favorable consequences in the future. The results of the present study could be explained by (ii) above. Delayed feedback about performance provides a signal that can be used to evaluate the amount of effort needed to achieve the intended outcome. Thus, it allows for an update in the associations formed between actions and outcomes needed to guide behavior.

According to this proposed mechanism, the ACC is not directly involved in the implementation of control, but rather relays information about the need for increased resources. A candidate region responsible for implementing control is the lateral prefrontal cortex (Ridderinkhof, Ullsperger, et al., 2004; Paus, 2001; Carter et al., 2000). Another intriguing possibility is that the ACC signals the need for increased resources to subcortical areas associated with homeostasis and autonomic control (Critchley, 2005; Critchley, Tang, Glaser, Butterworth, & Dolan, 2005; Critchley et al., 2003), perhaps more specifically to the locus coeruleus (Aston-Jones & Cohen, 2005). Locus coeruleus-mediated release of norepinephrine would then act as a signal that increases arousal, and adjusts the gain in task-related processing units (including the lateral prefrontal cortex), thereby improving performance.

Although more work is needed to determine whether this is a viable alternative to the present theories of ACC function, it provides a single interpretation for the main functional roles that have been ascribed to the ACC; namely, conflict monitoring (Botvinick et al., 2001, 2004; Yeung et al., 2004), error processing (Miltner et al., 1997), reinforcement learning (Holroyd & Coles, 2002), and pain processing (Vogt, Derbyshire, & Jones, 1996). It is also capable of explaining two sources of evidence that present difficulties for existing theories of ACC function in performance monitoring: the observation that ACC activity can precede performance (Brown & Braver, 2005; Shidara & Richmond, 2002; Oliveira, Hickey, & McDonald, unpublished data) and the present observation that unpredicted positive events can elicit ACC activations.

Conclusions

This study demonstrated that the FRN is not directly related to performance errors. We propose that the FRN is instead related directly to the recognition of violations in expectancy. This explains why the FRN can occur when performance is better than expected as well as when it is worse and could help reconcile conflicting findings from neuroimaging (Donkers, Nieuwenhuis, & van Boxtel, 2005; Walton et al., 2004) and single-cell recordings (Williams et al., 2004; Ito et al., 2003; Matsumoto et al., 2003; Akkal et al., 2002; Niki & Watanabe, 1979) with the bulk of literature showing an error-related ACC function. Taken together, these evidence suggest that the ACC does not act as part of a specialized performance-monitoring system that is selectively activated by information indicating errors in performance. Our results are therefore compatible with the idea that the FRN might represent the activity of the same general cognitive process as other ERP components that have been attributed to the ACC (Holroyd, 2004; Mai, Luo,

Wu, & Luo, 2004; Bokura, Yamaguchi, & Kobayashi, 2001; Eimer, Goschke, Schlaghecken, & Sturmer, 1996; Pritchard et al., 1991).

We further propose that the common observation that negative feedback elicits increased ACC activity when compared to positive feedback could be explained by the overoptimistic nature by which humans judge their own performance. These results could illuminate the understanding of pathological disorders affecting performance monitoring and behavioral adaptability. For instance, the neurobehavioral deficiencies associated with depression and bipolar mania are closely related to those that would be predicted based on the present study. Depression is characterized by pessimistic expectations of self-related events (American Psychiatric Association, 2000), and hypoactivity in the ACC (Pizzagalli, Peccoralo, Davidson, & Cohen, 2006; Ruchsow et al., 2004, 2006; Davidson et al., 2002; Mayberg et al., 1997; also see Ullsperger, 2006), along with difficulty in behavioral switching, in generation of goal-directed actions and in engagement of conscious cognitive effort (Pizzagalli et al., 2006; Murphy et al., 1999; Lafont et al., 1998). Mania is characterized by overconfidence and increased overoptimism in generating expectations of self-related events (American Psychiatric Association, 2000) along with hyperactivity in the ACC (Blumberg et al., 2000), and difficulty in the inhibition of inappropriate responses and in the maintenance of attentional focus (Minassian, Paulus, & Perry, 2004; Perry, Minassian, Feifel, & Braff, 2001; Murphy et al., 1999). Although the specific relation between these behavioral and neurological dysfunctions is a question to be addressed by future research, the present work suggests that the study of this relation may prove beneficial to the understanding and treatment of such diseases.

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Note

1. To exclude the possibility that these results stemmed from the fact that the correct/error and the error/correct conditions had different proportion of trials, we randomly sampled from the correct/error trials so that we created a new correct/error condition with an equal number of trials to the error/correct condition. Our analysis showed no differences between these two conditions [$t(12) < 1$]. We opted to keep the original analysis in the article because, although the subaveraging provided a useful safeguard against spurious findings, it did not improve the signal-to-noise ratio in the critical condition.

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