Error-related negativities elicited by monetary loss and cues that predict loss

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Event-related potential studies have reported error-related negativity following both error commission and feedback indicating errors or monetary loss. The present study examined whether error-related negativities could be elicited by a predictive cue presented prior to both the decision and subsequent feedback in a gambling task. Participants were presented with a cue that indicated the probability of reward on the upcoming trial (0, 50, and 100%). Results showed a negative deflection in the event-related potential in response to loss cues compared with win cues; this waveform shared a similar latency and morphology with the traditional feedback error-related negativity. *NeuroReport* 18:1875–1878 © 2007 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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Introduction

People adapt to changing environments by evaluating the positive and negative consequences of their actions. Many researchers have used event-related potentials (ERPs) to investigate how the brain processes positive and negative feedback from the environment. For instance, research has identified two negative deflections in the ERP [termed error-related negativities (ERNs)] that are uniquely associated with erroneous or otherwise undesirable events. The ERN was first reported when participants made errors in reaction time tasks or learning tasks [1–3]. This response-locked ERN is maximal at frontocentral electrode sites, and peaks approximately 50 ms following the execution of an erroneous response [3].

A similar ERN has been found following the receipt of feedback indicating undesirable outcomes, such as error commission or monetary loss [1,4–10]. This feedback-locked ERN occurs at frontocentral electrode sites, and is maximal approximately 250 ms following the onset of negative compared with positive feedback. Several investigations have identified the neural source of both ERNs in the anterior cingulate cortex using source-localization techniques [1,4,9,11].

One theory that attempts to explain both ERNs is the reinforcement learning theory (RL-ERN). Holroyd and Coles [1] argue that both response-ERNs and feedback-ERNs reflect the activity of a general error-processing system that influences reinforcement learning. More specifically, RL-ERN theory states that ERNs are generated when a mesencephalic dopamine signal disinhibits neurons in the anterior cingulate cortex [1]. The anterior cingulate cortex then uses the error signal to modify performance on the task at hand. A crucial point demonstrated by Holroyd and Coles [1] was that over the course of trial-and-error learning, ERNs are elicited by feedback when participants have not learned the correct response. Once participants, however, have leaned the appropriate stimulus–response associations, the ERN is then elicited by the participant's incorrect response. Thus, the ERN is produced at the very first indication that outcomes are worse than expected [1].

Certain predictions of the RL-ERN theory have been tested through both trial-and-error learning tasks and gambling tasks. For instance, several studies sought to determine whether ERNs are sensitive to expectation and probability. Findings suggest that larger feedback-ERNs are elicited by more unexpected negative feedback [1,12–14] and by more infrequent negative feedback as well [15].

It is important to note that for some stimuli in reinforcement learning tasks, the participant is always correct or always incorrect, regardless of how they respond. Hence, there is no opportunity for the participant to learn the correct stimulus–response association on some trials. In designs such as these, the actual stimulus itself then signals the correct/error outcome. Some gambling studies have also included cues that predict the probability of gains and losses on the upcoming trial (e.g. [5,12]). No studies to date, however, have examined whether early predictive cues can also elicit an ERN. In other words, it is unclear whether a cue that predicts loss, like an erroneous response and negative feedback, can elicit an ERN.

Presumably, this was not originally investigated because RL-ERN theory claims that the ERN reflects the evaluation of actions. Yet, several studies have recently found that ERNs can be elicited in the absence of an active response or overt motor response [8,16]. For instance, Yeung *et al.* [8] found that ERNs were elicited by negative feedback in a roulette game in which participants simply attended to the outcomes of the game; participants were not required to make active choices or perform any motor responses.

As RL-ERN theory states that ERNs are the first indication that outcomes are worse than expected, and considering that ERNs have been elicited in the absence of responding, it stands to reason that a cue predicting an undesirable outcome might also elicit an ERN. To examine this possibility, this study utilized a simple gambling experiment in which participants were told to guess which of two doors hid a prize. Prior to each trial, participants were presented with a cue indicating the number of doors that hid a prize on the upcoming trial (i.e. 0, 1, or 2). Thus, '0' and '2' cues predict losses and gains, respectively, with 100% accuracy. Consistent with earlier studies, we expected that loss compared with gain feedback on 1-cue (50% probability of gain) trials would elicit a feedback ERN. On the basis of the notion that the ERN, however, reflects the evaluation of unfavorable events, we also hypothesized that 0 compared with 2 cues would elicit a similar negativity.

Method

Participants

Sixteen undergraduates (seven women, nine men) participated in this study; none of whom withdrew from the experiment once it had begun. The average age of the participant sample was 19.13 years (SD=0.96). Each participant received course credit and $5.00 (winnings from the gambling task) for participating in the study. Informed consent was obtained from participants prior to each experiment. This research was formally approved by the Stony Brook University Institutional Review Board.

Task and materials

The present task was administered on a Pentium D class computer, using Presentation software (Neurobehavioral Systems Inc., Albany, California, USA) to control the presentation and timing of all stimuli. During the task, participants were shown a graphic displaying two doors horizontally adjacent and were told to choose which door they wanted to open (the graphic occupied approximately 6° of the visual field vertically and 8° horizontally). Participants were told to press the left mouse button to choose the left door or the right mouse button to choose the right door. Following each choice, a feedback stimulus appeared on the screen informing the participants whether they won or lost money on that trial. A green '↑' feedback indicated a correct guess and a red '↓' indicated an incorrect guess. Prior to each trial, a white '0', '1', or '2' cue was presented to inform participants how many of the doors would contain a prize on the upcoming trial; therefore, '0', '1', or '2' indicated that the probability of reward on the upcoming trial was 0, 0.50, or 1, respectively. All cues and feedback were presented against a black background and occupied approximately 3° of the visual field vertically and 1° horizontally. A fixation mark (+) was presented prior to the onset of each stimulus. At the end of each trial, participants were presented with the instruction to 'Click for next round'.

Stimuli appeared in the following order and remained on screen for the following durations: (i) cues were presented for 2000 ms, (ii) a fixation mark was presented for 500 ms, (iii) the graphic of two doors was presented indefinitely until a response was made, (iv) a fixation mark was presented for 1000 ms, and finally (v) a feedback arrow was presented for 2000 ms. The intertrial interval between feedback stimulus and the following 'Click to continue' instruction was 1500 ms.

Participants were told that they would gain $0.20 each time they opened a door that hid a prize, and lose $0.10 each time they opened a door without a prize. Participants received rewards on exactly 50% of the trials; negative feedback was presented on 100% of 0-cue trials, 50% of 1-cue trials, and 0% of 2-cue trials.

Procedure

Following a brief description of the experiment, EEG sensors were attached and the participant was given detailed task instructions. To familiarize participants with the task, they were given a practice block containing five trials and told to choose which door hid the prize. The actual experiment consisted of 100 trials (25 0-cue trials, 50 1-cue trials, and 25 2-cue trials) that were presented in random order. Every 20 trials, a running total of money earned was presented on the screen. At the end of the experiment, participants were paid $5.00.

Psychophysiological recording, data reduction and analysis

The continuous EEG was recorded using an ECI electrocap and the ActiveTwo BioSemi system (BioSemi, Amsterdam, The Netherlands). Recordings were taken from 64 scalp electrodes on the basis of the 10/20 system. In addition, two electrodes were placed on the left and right mastoids (M1 and M2, respectively). The electrooculogram generated from blinks and eye movement was recorded from four electrodes: two approximately 1 cm above and below the participant's left eye, one approximately 1 cm to the left of the left eye, and one approximately 1 cm to the right of the right eye. As designed by BioSemi, the ground electrode during acquisition was formed by the common mode sense active electrode and the driven right leg passive electrode.

All bioelectric signals were digitized on a laboratory microcomputer using ActiView software (BioSemi, Amsterdam, The Netherlands) and analyzed off-line using Brain Vision Analyzer (Brain Products, Gilohing, Germany). The EEG was sampled at 512 Hz. All data were rereferenced to the numeric mean of the mastoids and band-pass filtered between 0.1 and 30 Hz. Offline, the EEG was corrected for blinks and eye movements using the method developed by Gratton *et al.* [17]. In addition, a semiautomated procedure was used to identify and reject physiological artifacts according to the following criteria: a voltage step of more than 50.0 μV between sample points, a voltage difference of 300.0 μV within a trial, and a maximum voltage difference of less than 0.50 μV within 100 ms intervals.

Stimulus-locked ERPs were averaged separately for each type of feedback (gain or loss) and each predictive cue (0, 1, or 2). For ERP averages, the average activity in the 200 ms window prior to stimulus onset served as the baseline. Two separate difference waves were then created: first, the feedback-ERN on the basis of the difference between loss and gain feedback on 1-cue trials; second, the cue-locked ERN on the basis of the difference between the 0 and 2-cues. ERNs were then defined as the most negative point in the difference waves within a 200–600 ms window following the feedback or the cue. On the basis of the earlier research that has demonstrated a midline distribution of the ERN, the feedback-related and cue-related negativities were evaluated at the 10 midline

electrode sites (i.e. Fpz, AFz, Fz, FCz, Cz, CPz, Pz, POz, Oz, and Iz). The two negativities were analyzed using SPSS (14.0; SPSS Inc., Chicago, Illinois, USA) statistical software with Greenhouse–Geisser corrections applied to P values associated with multiple d.f. repeated measures comparisons.

Results

Figure 1 presents the average amplitude of the cue-locked and feedback-locked negativities across all midline electrode sites. A 2 (Trial Type: Cue, Feedback) $\times$10 (Electrode Site) factorial repeated-measures analysis of variance indicated that ERN amplitude did not differ overall between cues and feedback [$F(1,15)=2.73$, $P>0.10$]. The magnitude of the ERN varied across electrode sites [$F(9,135)=10.22$, $P<0.001$], however, consistent with the impression from Fig. 1, this was qualified by a significant interaction between trial type and electrode site [$F(9,135)=3.96$, $P<0.05$]. Post-hoc t-tests indicated that the feedback ERN was reliably larger than the cue ERN only at Cz after Bonferoni correction [$t(15)=3.73$, $P<0.005$].

Figures 2 and 3 present feedback-locked and cue-locked data at Cz, where the feedback ERN was maximal. As evident in Figs 2 and 3, both loss feedback and loss cues elicited a negative deflection in the ERP with a similar morphology and peak latency. A t-test confirmed that the peak latency of the feedback-ERN (M=371.9 ms, SD=109.9) did not differ from the cue-ERN [M=417.2 ms, SD=122.7; $t(15)=0.86$, $P>0.40$].

Discussion

Consistent with earlier literature, this study showed a centrally maximal negative deflection in the ERP that peaked approximately 400 ms after the receipt of feedback indicating monetary loss – the feedback-ERN [1,4–10]; furthermore, a similar negative deflection was elicited by predictive cues presented prior to completing the task and receiving feedback. Specifically, compared with cues that indicated impending gains, cues that predicted subsequent losses were associated with a negative deflection in the ERP at approximately the same latency as the feedback-ERN (see Fig. 3). It is important to note, however, that the cue-ERN in this study had a broader scalp distribution than the feedback-ERN. It is possible then that processing impending losses engaged somewhat different neural circuitry than processing actual losses. It will be important for future studies to further evaluate the similarity of the cue-ERNs and feedback-ERNs using source localization techniques.

The presence of a cue-elicited ERN is broadly consistent with the reinforcement learning theory of the ERN. Specifically, Holroyd and Coles [1] detailed how, over the course of reinforcement learning tasks, the ERN is initially elicited by negative feedback, but becomes elicited by the erroneous response once the appropriate stimulus–response associations are learned. Consistent with the RL-ERN theory's claim that ERNs are produced at the very first indication that outcomes are worse than expected, the present data demonstrates that an ERN-like component can

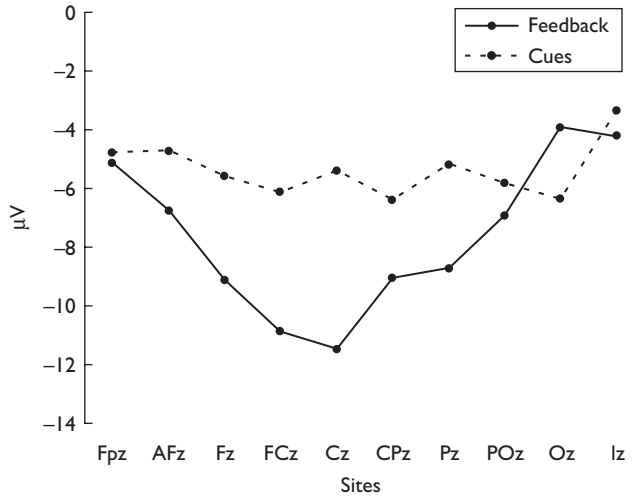

Fig. 1 Amplitude of the feedback (loss minus gain) and cue (0 minus 2) difference wave peaks at each midline electrode site, from front (left) to back (right) of the head.

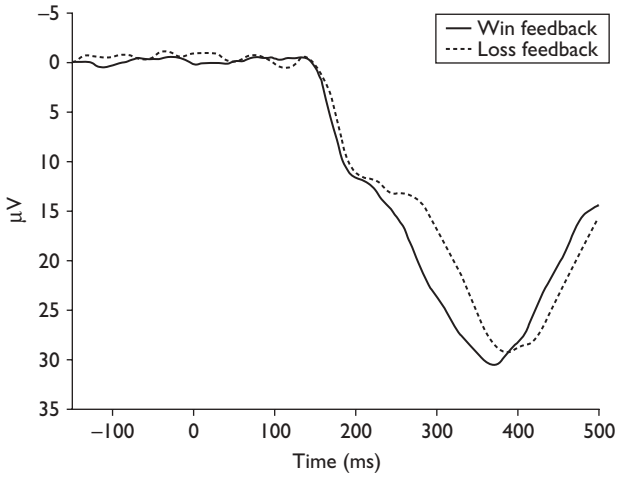

Fig. 2 Event-related potential for gain and loss feedback at site Cz; stimulus onset occurred at 0 ms.

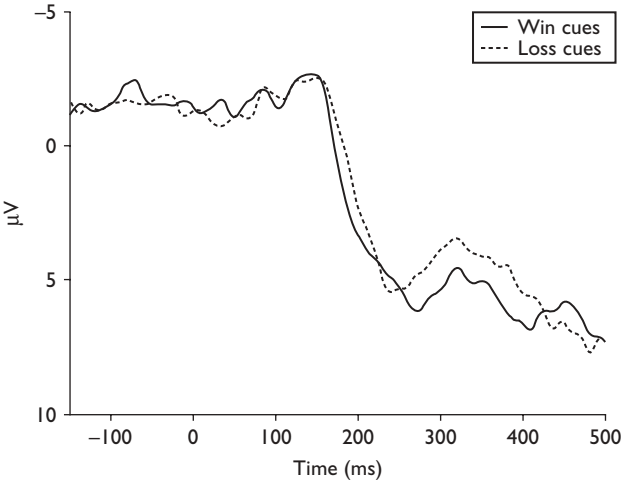

Fig. 3 Event-related potential for 0 and 2-cue stimuli at site Cz; stimulus onset occurred at 0 ms.

be elicited by early, predictive cues that signal impending loss.

According to the original interpretation of the RL-ERN theory [1], learning would be expected to take place only when feedback is dependent on behavior. In spite of this, several investigations have elicited ERNs in the absence of active choices or overt responses [8,16]. In addition, more recent studies have even found ERNs elicited by simply observing the behavior of others [18,19]. This study, then, fits well in the context of these studies, and further suggests that responses are not required to elicit an ERN.

Taken together, the RL-ERN theory may need to be expanded to include learning that is not strictly contingent on recent behavior [8]. Considering this, arguments have been made that, rather than simply being linked to error commission, the anterior cingulate cortex might be more generally involved in processing motivationally significant information regarding rewards and punishments [4,8]. For instance, Yeung *et al*. [8] identified a correlation between feedback-ERN amplitude and subjective ratings of involvement in tasks not requiring responses, indicating that motivational factors influence processing in the anterior cingulate cortex [20].

Conclusion

The elicitation of an ERN-like negative deflection to a predictive cue is consistent with the RL-ERN theory, which states that ERNs reflect the first indication that events are worse than expected. This study also provides evidence that these negativities may reflect the evaluation of distal events and serve a cautionary or predictive function in the brain's error processing system.

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