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ERN and the Placebo: A Misattribution Approach to Studying the Arousal Properties of the Error-Related Negativity

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Performance monitoring in the anterior cingulate cortex (ACC) has largely been viewed as a cognitive, computational process devoid of emotion. A growing body of research, however, suggests that performance is moderated by motivational engagement and that a signal generated by the ACC, the error-related negativity (ERN), may partially reflect a distress response to errors. Although suggestive, this past work is hampered by use of correlational designs or by designs that confound affect and cognitive performance. Here we use the misattribution of arousal paradigm—an experimental paradigm that pilot research shows can dissociate affect from cognitive performance—to investigate the extent to which the ERN has arousal properties. Forty university students completed a misattribution of arousal paradigm by consuming a beverage they believed would either increase their anxiety or would have no side effects and then completed a go/no-go task while we recorded ERNs. Results indicate that participants who were given the opportunity to misattribute arousal exhibited a reduced ERN compared with participants who were not given any misattribution cues. This occurred despite no measurable differences in performance on the go/no-go task. In addition, correlations between the ERN and behavior were observed only for participants who did not misattribute their arousal to the placebo beverage. Taken together, these results suggest that the ERN is dissociable from cognitive performance but not negative affect.

Keywords: cognitive control, anterior cingulate cortex, error-related negativity, misattribution paradigm, performance monitoring

Monitoring one's performance, particularly for errors, is essential for successful day-to-day functioning. Errors provide the impetus for correcting one's behavior and guiding it toward a desired goal state. Certain occupations, such as air traffic controllers, surgeons, and airport screeners, have a low tolerance for errors and thus require a high degree of cognitive control to perform their duties effectively. How are these individuals capable of monitoring their performance with such precision? Does performance monitoring result exclusively from cognitive processes in the brain, or are other factors, such as affect, also involved?

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Performance Monitoring

Over the past 20 years, a growing body of research has provided insight into the neural basis of performance monitoring. One of the best-studied neural correlates of performance monitoring is an event related potential (ERP) called the error-related negativity (ERN; Falkenstein, Hohnsbein, & Hoormann, 1990; Gehring, Goss, Coles, & Meyer, 1993). The ERN is a pronounced negative deflection on electroencephalography (EEG) that occurs within 100 ms of making an error on a task, and is thought to be generated by the anterior cingulate cortex (ACC; Dehaene, Posner, & Tucker, 1994).

Several theories have attempted to explain the function of the ERN. Holroyd and Coles (2002) hypothesized that the ERN reflects a discrepancy between an expected outcome (e.g., a correct response) and an actual outcome (e.g., an error). When expectancy violation occurs, dopaminergic neurons in the midbrain that project to the ACC temporarily cease firing, resulting in the disinhibition of the ACC and the consequent generation of an ERN. According to Holroyd and Coles's (2002) computational model, the ERN acts as a reinforcement learning signal to adjust ongoing behavior closer to expected behavior. In support of this theory, studies have found that administration of dopamine agonists and antagonists resulted in larger and smaller ERN amplitudes, respectively (de Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004; de Bruijn, Sabbe, Hulstijn, Ruigt, & Verkes, 2006).

Another computational model (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Yeung, Botvinick, & Cohen, 2004) posits that the ERN, and ACC activity more generally, signals conflict

between competing responses. When an error occurs, the motor programs for the correct and incorrect responses are activated simultaneously, resulting in enhanced ACC activity and, consequently, an ERN. According to this view, increased ACC activity should be observed not only following errors but also immediately preceding correct responses with a high degree of response conflict. This prediction has been supported by functional neuroimaging studies showing an increase in ACC activity preceding correct responses characterized by high conflict, such as incongruent trials on the Stroop task (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Carter et al., 1998).

ERN: Affective Significance of Detected Errors?

Although this predominantly cognitive perspective of performance monitoring has received much support, recent developments suggest that such computational models may provide an incomplete picture of the ERN because they do not account for emotion. Recent research, for example, suggests that the ERN is related to affective and defensive reactions to errors (Gehring & Willoughby, 2002; Hajcak & Foti, 2008; Luu, Collins, & Tucker, 2000) and that it may represent a more general evaluative system processing the motivational significance of events (Gehring & Fencsik, 2001) or even a "distress signal" (Bartholow et al., 2005, p. 41).

There is mounting evidence, then, that the ERN is influenced by—and perhaps partially reflects—an emotional response to errors (e.g., Luu, Tucker, Derryberry, Reed, & Poulsen, 2003). The ERN, as the name implies, is time-locked to errors, and errors are not affectively neutral events. Rather, errors are distressing because of the negative consequences typically associated with them. Errors, for example, prompt increased skin conductance, greater heart rate deceleration, increased pupil dilation, and larger startle reflexes compared with correct responses (Critchley et al., 2003; Hajcak & Foti, 2008; Hajcak, McDonald, & Simons, 2003b).

The ERN may thus reflect not only the detection of an error but also the negative affect that accompanies such detection. If this is the case, then individual differences in negative affect should moderate the amplitude of the ERN. And indeed, studies have repeatedly observed enhanced ERN amplitudes in patients with anxiety disorders (e.g., Gehring, Himle, & Nisenson, 2000) and among healthy participants high in anxious apprehension (Moser, Moran, & Jendrusina, 2012) or high in trait negative affect (e.g., Hajcak, McDonald, & Simons, 2003a, 2004; Luu et al., 2000). The ERN is also associated with defensive reactions. Hajcak and Foti (2008) found that greater ERN amplitude predicted enhanced eyeblink startle after errors, a measure of defensiveness and aversion.

These correlations between ERN amplitude and negative affect are also consistent with studies demonstrating involvement of the ACC in negative affect and arousal (Bush, Luu, & Posner, 2000; Shackman et al., 2011). Similar to the ERN, the ACC is implicated in a number of mood disorders, including anxiety and depression (Drevets et al., 1997; Pezawas et al., 2005). In addition to serving as a target for anxiolytic agents (Albrechet-Souza, Borelli, Carvalho, & Brandao, 2009), the ACC is associated with sympathetic modulation of heart rate (Critchley et al., 2003), pain (Rainville, Duncan, Price, Carrier, & Bushnell, 1997), and distress (Eisenberger & Lieberman, 2004). Finally, primate studies indicate that the ACC is richly interconnected with subcortical regions involved in negative affect, including the periaqueductal gray (An, Bandler, Ongur, & Price, 1998), the basolateral nucleus of the amygdala (Ghashghaei, Hilgetag, & Barbas, 2007), and the ventral striatum and nucleus accumbens (Kunishio & Haber, 1994). The ERN and ACC more generally, then, are associated with negative affect.

While this work highlights how the ERN is associated with negative affect and arousal, because many of these studies are correlational it is often difficult to know what role affect plays—does the ERN reflect a negative emotional response to errors or does a negative emotional response merely moderate aspects of error detection? A more serious problem for this past work is that it often confounds affective and cognitive aspects of performance monitoring. That is, even though this work suggests that the ERN is related to negative affect, because affective and cognitive aspects of error detection often go hand in hand in this past work, it is difficult to conclude that these observed effects reflect affect and not cognition (Yeung, 2004). For example, while recent work highlights how anxiolytic agents such as alcohol reduce the ERN because of drops in negative affect, this same work indicates that alcohol hampers cognitive task performance (Bartholow, Henry, Lust, Saults, & Wood, 2011). Similarly, while increasing the motivational significance of errors amplifies the ERN, it also improves cognitive task performance (Hajcak, Moser, Yeung, & Simons, 2005, Experiment 2; Gehring et al., 1993). It is thus possible that these between-group differences in task performance, and not differences in affect and motivation, drive changes to the ERN (Yeung, 2004). Because affect is so often confounded with performance in these past studies, it is difficult to know if the ERN reflects a negative emotional response to errors or if this negative emotional response is merely an epiphenomenon of conflict and error monitoring.

Thus, there is still considerable debate about what the ERN represents (e.g., Inzlicht & Bartholow, 2009), with purely cognitive models of the ERN still very much prevailing. For example, a recent literature search on PsycINFO using the keywords *error related negativity* generated a large number of articles, the vast majority of which described the ERN in purely cognitive terms.[1] Case in point, among the five most recent articles on the ERN (Arbel & Donchin, 2011; Hammer, Rautzenberg, Heldmann, & Munte, 2011; Hoffmann & Falkenstein, in press; Perez et al., 2011; Suzuki & Shinoda, 2011), only one of them (Arbel & Donchin, 2011) mentioned the motivational underpinning of the ERN, and even this was done only in passing; the remainder of the articles described the ERN in entirely cognitive terms. Despite clues to its affective qualities, then, the predominant view of the ERN is a purely cognitive one. Thus, more work is needed to understand how negative affect directly contributes to the amplitude of the ERN, with a special emphasis on work that can distinguish cognitive versus affective models of it (Yeung, 2004). The goal of the current article is to do just this, and we do so by using a classic method in social psychology: the misattribution of arousal paradigm.

[1] We conducted the PsycINFO search on December 28, 2011, using the words "error related negativity" in the keyword field. We then examined the first five articles that examined the ERN.

Misattribution of Arousal Paradigm

The pioneering work of Schachter and Singer (1962) on the labeling of emotion demonstrates that arousal can be misattributed. They reasoned that emotion was a combination of nonspecific physiological arousal and a cognitive label, which led them to propose that different emotional states—joy, fury, or jealousy—could be produced by the same arousal state labeled with different cognitions (cf. Reisenzein, 1983). This also suggests that arousal can be attributed to wrong sources, thus changing the nature of the emotional experience. One implication of this is that arousal can be "explained away," its effects reduced when attributed to benign external sources. Indeed, results from a large body of studies using the misattribution paradigm show that mislabeling the source of one's emotional state reduces the magnitude of that emotional state and the sequelae of that state (Ross, Rodin, & Zimbardo, 1969; Zanna & Cooper, 1974). In one study, Storms and Nisbett (1970) gave patients with insomnia a sugar pill. Some participants were told that the pill would make them feel tense and aroused, whereas others were told that the pill would make them feel calm and relaxed. Paradoxically, patients who took the "arousing" pill fell asleep faster than those who took the "relaxing" pill, presumably because participants in the former group attributed their arousal to an external agent—the pill—rather than to their own restlessness. In another study, participants who were fearful of electric shocks exhibited less fear and could tolerate more shocks if they were able to misattribute their naturally occurring arousal to the effects of a loud noise (Ross et al., 1969). By mislabeling naturally occurring arousal to an external source, then, misattribution appears to lessen negative affect.

One of the most famous uses of the misattribution paradigm was by Zanna and Cooper (1974), who used it to settle the then-raging debate about the nature of cognitive dissonance. As with the ERN today, there was once a dispute as to whether dissonance was a cognitive (Bem, 1967) or affective (Festinger, 1957) process. In their study, Zanna and Cooper induced dissonance and then gave participants a sugar pill. Some participants learned that the pill would make them feel anxious; others learned that the pill would have no side effects. Participants who took the arousing pill (i.e., those who could misattribute their endogenous arousal) showed no signs of dissonance, whereas those who took the no-effects pill (i.e., those who would correctly attribute their own arousal) showed typical dissonance. Zanna and Cooper's use of the misattribution paradigm provided strong evidence that dissonance is an affective phenomenon (see Harmon-Jones & Mills, 1999).

The misattribution paradigm may provide similarly strong evidence about the affective nature of the ERN. One advantage of using the misattribution paradigm is that it may allow for the manipulation of affect while keeping cognitive components of performance monitoring constant (i.e., cognitive detection of error and conflict should not be affected by mislabeling). The aim of the present study was to use the misattribution paradigm to determine whether the presence of an alternative source of one's arousal decreases the amplitude of the ERN. Importantly, because misattribution should not affect cognitive performance (e.g., speed or accuracy), it provides a strong test of the affective versus cognitive models of the ERN (Yeung, 2004).

Given the importance of using a manipulation that could dissociate affective from cognitive influences on the ERN, we first conducted a pilot test of our misattribution manipulation, measuring performance on a cognitive reaction time test, expectations for the reaction time test, and state anxiety. We predicted that our misattribution manipulation would lower state anxiety after the test yet keep cognitive performance (and expectation for cognitive performance) intact.

Pilot Study

Method

Participants and procedure. We conducted an initial pilot study to examine the affective and cognitive consequences of our misattribution of arousal manipulation. Fifty-eight participants (32 women) from the participant pool at the University of Toronto Scarborough participated for course credit. Participants were told that the goal of the experiment was to investigate the effects of an herbal supplement (which we called *Panax Senticosus*) on cognitive performance. Participants were randomly assigned to one of two conditions. Participants in the misattribution condition were told that the herbal supplement had minor side effects, including tenseness, anxiety, and increased heart rate. By contrast, participants in the control condition were told that the herbal supplement had no side effects. Participants were then asked to drink a solution of water with green food coloring. Immediately after ingesting the placebo beverage, participants completed the Spielberger 20-item, 4-point Likert State Anxiety Inventory (Spielberger, 1983), which is a reliable measure of state negative affect ($\alpha = .87$) and includes items relating to feeling nervous and feeling relaxed. Participants then completed the 21-item Situational Motivation Scale on a 7-point Likert scale (Guay, Vallerand, & Blanchard, 2000). Critically, this scale contained four items that assessed expectations for the upcoming cognitive reaction time task (e.g., "I think I will be pretty good at this task"; "I think I will be satisfied with my performance at the upcoming reaction time task"; $\alpha = .82$), and we used it to assess performance expectations.

Participants then completed the go/no-go task. Participants were required to press a button if they saw a "go" stimulus (i.e., the letter M) and to refrain from pressing the button if they saw a "no-go" stimulus (i.e., the letter W). Each trial consisted of a fixation cross ("+") presented randomly for 300–700 ms, followed by either a go or no-go stimulus for 100 ms. The maximum time allowed for a response was 500 ms. Participants first completed a practice block and then completed two experimental blocks, each consisting of 80 go trials and 20 no-go trials. Go and no-go trials were presented randomly. Average reaction time on correct and incorrect trials was measured, as was the number of errors of commission (going during a no-go trial) and omission (not going during a go trial). Preliminary analyses revealed that five participants acted as if they were not following instructions, withholding button presses on 52% or more of the simple go trials; they were outliers in terms of error rates (all Extreme Studentized Deviate statistics > 4.00, $p < .001$) and were excluded as a result. We note, however, that their inclusion did not affect the shape of our results.

After the go/no-go task, participants once again completed the State Anxiety Inventory (Spielberger, 1983). We measured state anxiety twice, once immediately after our manipulation and a second time after the go/no-go reaction time task. Although we

suspected that our misattribution manipulation might have immediate effects, we were more confident that differences in state anxiety would emerge after the natural, albeit mild, stress of taking a reaction time task. Participants were then carefully probed for suspicion with a funneled debriefing procedure (Bargh & Chartrand, 2000). Our misattribution of arousal paradigm used deception to convince participants that they were ingesting an herbal supplement that might have side effects, so it was important to ensure that participants believed our cover story. Funneled debriefing uncovered five participants who expressed suspicions about the true nature of the study, and they were thus excluded from analyses. As with the exclusions above, however, inclusion of these suspicious participants did not affect the shape of our results. We prefer these conservative inclusion criteria, however, because they offer the best test of the affective and cognitive consequences of misattribution.

Results and Discussion

Results confirmed that the misattribution of arousal paradigm had no effect on cognitive performance or on performance expectations, while significantly lowering state anxiety after the cognitive reaction time test. Table 1 indicates that participants who were told that the placebo beverage would have adverse side effects made as many errors of omission, $t(46) = -.37$, *ns*, and commission, $t(46) = -.33$, *ns*, as participants who were told that the beverage had no side effects; they also responded as quickly as one another when making a correct go response, $t(46) = .77$, *ns*, and an incorrect no-go response, $t(46) = 1.36$, $p = .18$. Participants further showed no differences in how they expected to perform, $t(46) = 1.18$, $p = .24$.

Importantly, we found differences when we examined the effects of misattribution on state anxiety. Although participants in the misattribution group showed only a trend of being less anxious than those in the control group immediately after the manipulation, $t(46) = -1.36$, $p = .18$, $d = .40$, this trend became significant on our second measure of anxiety, after participants completed the go/no-go task, $t(46) = 2.16$, $p < .05$, $d = .64$. Interestingly, all participants experienced more anxiety from pre- to postreaction time task, $t(46) = -2.82$, $p < .01$, $d = .41$, suggesting the experience of performing the go/no-go was (mildly) stressful. This counters suggestions that such cognitive reaction time tasks recruit only cold executive processes but not those related to negative affect (Amodio & Ratner, in press). All told, when participants were encouraged to misattribute their naturally occurring, task-induced arousal to a benign external source, their arousal was "explained away" and thereby reduced. As with past research, mislabeling the source of one's emotional state reduced the magnitude of that emotional state (Ross et al., 1969; Zanna & Cooper, 1974). Critically, it did so without affecting cognitive performance. This pilot test thus confirms that the misattribution paradigm may allow for the manipulation of affect while keeping cognitive components of performance monitoring intact. It should thus provide a strong test of the affective versus cognitive models of the ERN (Yeung, 2004).

EEG Study

Method

Participants. Forty introductory psychology students (22 women, 18 men) at the University of Toronto Scarborough (mean age = 19.4 years, $SD = 5.2$ years) participated for course credit. Three participants were excluded from analyses due to equipment malfunction.

Procedure. As with the pilot study above, participants were told that the goal of the experiment was to investigate the effects of an herbal supplement on cognitive performance. Half of the participants were assigned to the misattribution of arousal group and the other half to the control group. Participants were then asked to drink a solution of water with green food coloring. Participants waited about 20 min before performing the main go/no-go task, during which they were prepared for EEG recording and during which the placebo beverage could ostensibly take effect. The go/no-go task was the same as in the pilot, except that

Table 1
Means (SD) for Pilot Study and Electroencephalography (EEG) Study, With Cognitive Performance, Performance Expectations, and State Anxiety for Participants in the Misattribution and Control Groups

	Pilot study		EEG study	
Dependent variable	Misattribution	Control	Misattribution	Control
Omission error rate (%)	10.08_a (9.54)	11.23_a (11.53)	4.89_a (4.90)	3.83_a (3.27)
Commission errors (%)	36.63_a (16.88)	38.10_a (13.59)	47.79_a (14.97)	47.01_a (15.07)
Reaction time correct	261.19_a (44.84)	250.85_a (47.84)	283.04_a (38.46)	287.55_a (19.57)
Reaction time error	228.74_a (45.35)	212.15_a (38.97)	245.11_a (28.28)	245.47_a (16.68)
Performance expectations	3.12_a (1.48)	2.67_a (1.15)		
State anxiety (pretask)	1.82_a (.47)	2.01_a (.46)		
State anxiety (posttask)	1.97_a (.58)	2.29_b (.39)		
Correct-related negativity			-2.05_a (2.08)	-2.27_a (2.20)
Error-related negativity			-3.92_a (2.18)	-6.52_b (3.96)

Note. Within each experiment, means across rows with different subscripts differ significantly at $p < .05$ (two-tailed). For pilot study, $n = 23$ for the misattribution group and $n = 25$ for the control group. For EEG study, $n = 19$ for the misattribution group and $n = 17$ for the control group.

participants completed five experimental blocks, each consisting of 85 go trials and 15 no-go trials. The intertrial interval was also set to 50 ms, making for a very fast task. Participants were probed for suspicion at the end of the study using funneled debriefing (Bargh & Chartrand, 2000), and one participant expressed suspicion about the nature of the placebo beverage and was therefore excluded. Results, however, were virtually identical with this participant's inclusion.

Neurophysiological recording. Continuous EEG during the go/no-go task was recorded using a stretch Lycra cap embedded with 32 tin electrodes (Electro-Cap International, Eaton, OH). Recordings were digitized at 512 Hz using ASA acquisition software (Advanced Neuro Technology B.V., Enschede, The Netherlands) with average-ear reference and forehead ground. EEG was corrected for vertical electrooculogram artifacts (Gratton, Coles, & Donchin, 1983) and digitally filtered offline between 0.1 and 30 Hz (FFT implemented, 24 dB zero phase-shift Butterworth filter).[2] We baseline-corrected the signal by subtracting the average voltage 200 ms to 50 ms before key press. Artifacts were automatically detected with –75 μV and +75 μV thresholds. An epoch was defined as 200 ms before and 800 ms after the response. Data for these epochs were averaged within participants independently for correct (correct-related negativity; CRN) and incorrect trials (ERN), and then grand-averaged within the respective conditions. ERN and CRN were defined as the maximum negativity between 50 ms preresponse and 150 ms postresponse at the frontocentral midline electrode (FCz). ERN calculations were based on no fewer than five artifact-free error trials (Olvet & Hajcak, 2009).[3]

Results

Go/no-go task performance. As with the pilot study, results of this study revealed that the misattribution of arousal paradigm had no noticeable effect on performance. Table 1 indicates that participants who were told the placebo beverage would have adverse side effects made as many errors of omission, $t(34) = .75$, *ns*, and commission, $t(34) = .15$, *ns*, as participants who were told the beverage had no side effect; they also responded as quickly as one another when making correct go responses, $t(34) = -.44$, *ns*, and incorrect no-go responses, $t(34) = -.05$, *ns*.[4] Further analyses revealed typical effects for reaction time tasks. A 2 (group: misattribution vs. control) × 2 (response: error vs. correct) mixed-factor analysis of variance (ANOVA) with reaction time as the dependent variable revealed a significant main effect of response, $F(1, 34) = 287.24$, $p < .001$, $\eta_p^2 = .89$, such that reaction time on error trials, regardless of group, was significantly faster than reaction time on correct trials. A 2 (group: misattribution vs. control) × 2 (error type: omission vs. commission) mixed-factor ANOVA with error rate as the dependent variable revealed a significant main effect of error type, $F(1, 34) = 370.49$, $p < .001$, $\eta_p^2 = .92$, such that participants, regardless of group, committed significantly more commission than omission errors. Taken together, these data suggest that cognitive performance, as assessed by reaction time, number of errors, and type of errors, was equivalent across experimental groups. This is important because it allows for a dissociation between affective versus cognitive models of the ERN (Yeung, 2004).

The ERN. Inspection of the scalp topography of the ERN (see Figure 1D) reveals a clear frontocentral scalp distribution, which is consistent with past research (e.g., Hajcak & Foti, 2008) and justifies our use of FCz as our electrode of interest. A 2 (group: misattribution vs. control) × 2 (response: error vs. correct) mixed-factor ANOVA with minimum amplitude between −50 and 150 ms at FCz as the dependent variable revealed a significant main effect of response, $F(1, 34) = 46.02$, $p < .001$, $\eta_p^2 = .58$, such that for both groups the ERN was more negative ($M = -5.15$ μV, $SD = 3.37$) than the CRN ($M = -2.16$ μV, $SD = 2.10$; see Figure 1). Importantly, this main effect was subsumed under a significant interaction between group and response, $F(1, 34) = 6.96$, $p < .02$, $\eta_p^2 = .17$. Analysis of simple main effects revealed that, although the misattribution and control groups showed comparable CRNs, $F(1, 34) = .10$, *ns*, participants in the misattribution condition showed significantly reduced waveform amplitude on error trials compared with control participants, $F(1, 34) = 6.12$, $p < .02$, $\eta_p^2 = .15$. Importantly, these results were virtually unchanged after controlling for errors of commission, errors of omission, reaction time for correct responses, and reaction time for incorrect responses; the interaction between group and response was still significant, $F(1, 30) = 6.96$, $p < .02$, $\eta_p^2 = .19$, as was the simple effect of group on the ERN, $F(1, 30) = 5.58$, $p < .03$, $\eta_p^2 = .16$. In short, the misattribution manipulation affected the amplitude of the ERN separate of any cognitive performance difference. Given results of the pilot study indicating that misattribution lowered state anxiety but left cognitive control intact, results of this study offer strong evidence that the ERN has arousal properties that are independent of cognition.

Correlations between the ERN and behavior. The correlations between the ERN and performance on the go/no-go task revealed an interesting dissociation between error-related brain processing and measures of cognitive control for those who mislabeled their naturally occurring arousal to the placebo beverage. To examine these associations, we first computed an ERN minus

[2] Despite recommendations that only modest high-pass filters should be used (e.g., 0.1 or 0.5 Hz) for fear that they could change the morphology and latency of ERPs (Luck, 2005), we find that filtering at lower (0.1 Hz) or higher (1 Hz) frequencies yields practically identical results for peak amplitude, $r(34) = .82$, $p < .01$.

[3] All participants made at least 14 errors of commission; however, because of movement and other EEG artifacts, the number of usable error trials for calculation of the ERN was reduced. Nonetheless, every participant had at least five usable error trials for ERN calculation.

[4] We note that we found a different set of error rates in the pilot and main studies. Specifically, participants in the pilot study were more likely to omit pressing the button, hence more errors of omission and fewer errors of commission. In contrast, participants in the EEG study were more likely to press the button, hence fewer errors of omission and more errors of commission. We did not predict such differences, but we suspect that the nature of the EEG study (i.e., it involved wearing an elaborate EEG cap, used 2.5 times as many trials, contained a higher percentage of go trials, and had a short intertrial interval) motivated participants and pushed them to make more impulsive button-pressing errors. Nonetheless, the error rates and reaction times for the EEG study are in line with past results in our lab using EEG and the go/no-go task (Tullett & Inzlicht, 2010).

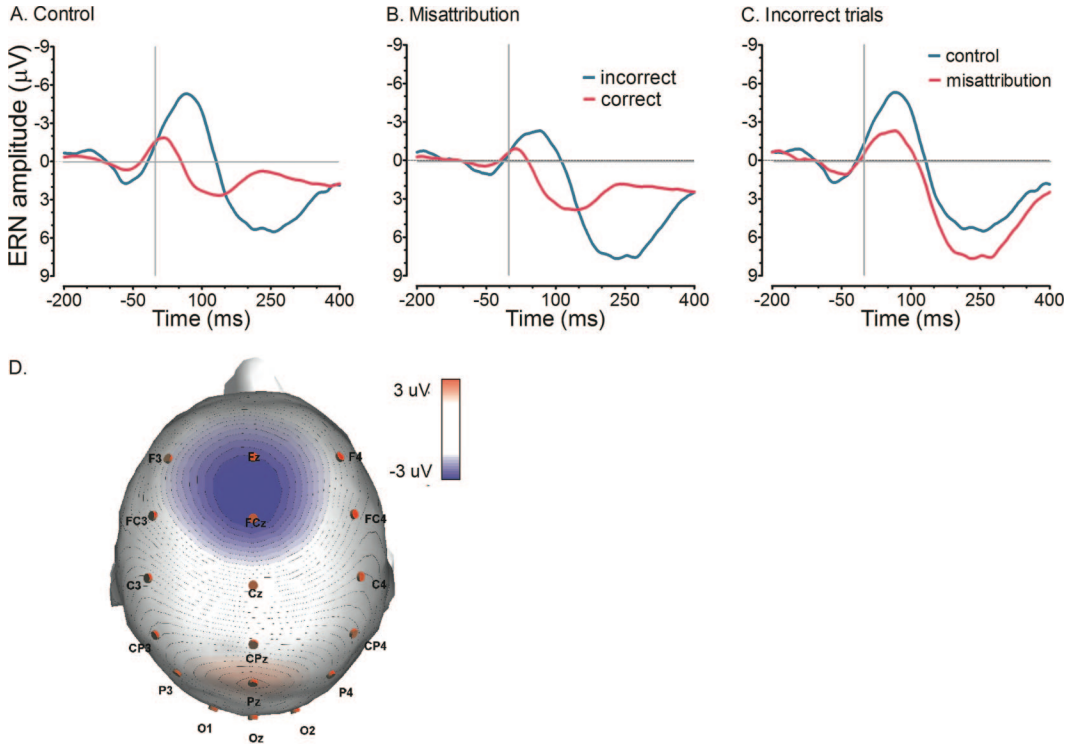

Figure 1. Response-locked waveform amplitude at FCz following correct and incorrect responses on the go/no-go task for participants in the (A) misattribution and (B) control conditions. (C) Comparison of ERN waveforms for the misattribution and control groups. (D) Headmap of voltage topographies across the scalp for error trials shows a clear frontocentral distribution.

CRN difference score[5] to specifically isolate error-related variance; we also log-transformed error rates to normalize the data but present raw scores for ease of interpretation. While the ERN was related to number of commission errors, $r(15) = .54$, $p < .02$, this was the case only for participants in the control group; the ERN was not related to the number of commission errors for participants in the misattribution group, $r(17) = .19$, $p = .44$ (see Figure 2). The ERN predicts performance, then, only when negative affect and arousal are attributable to the performance task; when this negative affect is misattributed, however, there is a dissociation between the ERN and cognitive control.

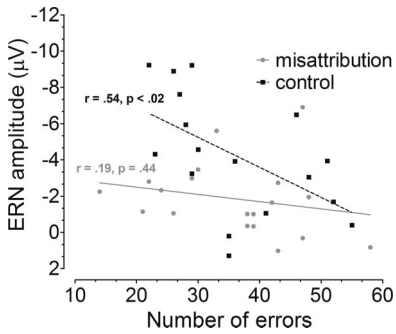

Figure 2. Correlations between ERN and number of commission errors for participants in the misattribution and control conditions. Greater ERN amplitude correlated with fewer commission errors in the control condition but not in the misattribution condition. The ERN is defined here as the ERN difference wave (i.e., ERN minus CRN).

General Discussion

The present study provides evidence that the ERN depends critically on the attribution of negative affect and perhaps on negative affect itself. In our study, when participants could misattribute their naturally occurring arousal to a benign external source, they exhibited reduced ERN amplitudes compared with control participants. Given past research indicating that misattributing arousal can reduce arousal states associated with fear, insomnia, and dissonance (Ross et al., 1969; Storms & Nisbett, 1970; Zanna & Cooper, 1974) and results of our pilot study indicating that it reduced states of anxiety produced by taking a test of cognitive control, our EEG results suggest that ERN amplitudes rely on negative affect. This article joins a growing

[5] According to Luck (2005), the absolute amplitudes of ERP components are meaningless in and of themselves because each component is affected by a large number of variables that may or may not reflect the putative psychological mechanism of interest. We accounted for this in the repeated measures analyses by examining the ERN relative to the CRN. For correlations, however, we take a difference wave approach (Luck, 2005) where we subtract the CRN from the ERN, an approach that cancels out processes common to both negative and positive responses and specifically isolates error-related variance.

number of others suggesting that the magnitude of the ERN is not exclusively determined by the computation of cognitive conflict (e.g., Bartholow et al., 2011; Grinband et al., 2011) but may instead be mediated in part by affect (Hajcak & Foti, 2008; Hajcak et al., 2005; Luu et al., 2000). Unlike past studies, most of which used correlational designs (e.g., Moser et al., 2012; Hajcak & Foti, 2008), the present study is unique because it provides evidence that manipulating the appraisal of negative affect leads to downstream changes in ERN amplitude.

Our results offer strong support for the affective interpretation of the ERN because we find that misattribution dampened the ERN and did so without corresponding changes in cognitive performance. Although past studies have made similar observations with the ERN and affect (e.g., Bartholow et al., 2011; Gehring et al., 2000), it is hard to know whether the results in these other studies were in fact due to changes in affect or to accompanying changes in task performance. In other words, to make strong claims about the ERN having arousal properties, one must first rule out the possibility that the observed effects reflect changes in the cognitive processes of error/conflict detection (Yeung, 2004). In the current study, we manipulated the attribution of anxiety and, according to our pilot study, anxiety itself, without changing cognitive performance, thus limiting the possibility that our effects are cognitive in nature. In other words, because we used a manipulation that selectively manipulated affect without manipulating cognitive performance, we can more strongly conclude that the ERN has emotion-like properties.

The current findings also suggest that negative affect may be an important determinant of whether the ERN predicts cognitive control. In accordance with past studies (e.g., Gehring et al., 1993; Inzlicht & Gutsell, 2007), greater ERN amplitude correlated with superior task performance. Our findings indicate, however, that this correlation holds up only when people are presumably able to match their negative affect to the task at hand. Conversely, when given the opportunity to "explain away" their anxiety, performance did not significantly relate to the ERN (see Figure 2). Interestingly, although widely predicted (e.g., Botvinick et al., 2001), the connection between the ERN and task performance is only sometimes found (Weinberg, Riesel, & Hajcak, in press). This suggests that something moderates the ERN-performance link. Given evidence that intrinsically motivating tasks not only increase the magnitude of the ERN but also produce robust associations between it and task performance (e.g., Bartholow et al., 2011; Legault & Inzlicht, 2011), we wonder if negative affect and the correct attribution of this negative affect might prove to be just such a moderator. Another possibility is that, just like the performance-arousal link (Yerkes & Dodson, 1908), the performance-ERN link might be represented by an inverted U-shaped curve. Future research will be needed to explore these possibilities.

Future research might also benefit from using a version of the misattribution paradigm that uses a within-subject design. If the appraisal of affect indeed modulates the ERN, as is indicated by the current study, then other kinds of appraisals might have similar influences. For example, we wonder, as has been found with other ERPs (e.g., Moser, Krompinger, Dietz, & Simons, 2009), whether instructions to upregulate or downregulate the negative emotions associated with errors might increase and decrease the amplitude of the ERN, respectively.

In a recent meta-analysis, Shackman and colleagues (2011) suggested that the dorsal ACC implements cognitive control by integrating negative affect to drive goal-directed behavior. The current results are consistent with this view and suggest that the ERN may be an electroencephalographic marker of this integration. Although the present results support an affective model of the ERN, it would be a mistake to conclude that the ERN is exclusively affective. Even when arousal was misattributed to the placebo beverage, participants nevertheless showed a pronounced ERN following errors, indicating that factors in addition to arousal modulate the amplitude of the ERN. The present findings are thus compatible with the computational models proposed by reinforcement learning (Holroyd & Coles, 2002) and conflict monitoring models (Botvinick et al., 2001) and suggest that the ERN is likely an affective signal that is based on the cognitive detection that things are worse than expected.

Conclusion

Despite mounting evidence that the ERN is influenced by affect and motivation, and other evidence that is difficult to reconcile with purely cognitive theories (e.g., Burle, Roger, Allain, Vidal, & Hasbroucq, 2008; Fellows & Farah, 2005), cognitive models of the ERN still very much prevail. For example, even with evidence that the ERN (and its putative ACC generator) is dissociated from cognitive control (e.g., Critchley et al., 2003; Fellows & Farah, 2005; Inzlicht & Tullett, 2010; Weinberg et al., in press), cognitive views remain the chief model used to explain the ERN. We hope the results of our study will play a part in changing this view. By using a manipulation that selectively manipulated state anxiety without manipulating cognitive performance, our study provides strong indication that the ERN is dissociable from cognitive performance but not negative affect.

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