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Neural Correlates of the Processing of Another's Mistakes: A Possible Underpinning for Social and Observational Learning

Matthew S. Shane¹, Michael Stevens², Carla L. Harenski¹, and Kent A. Kiehl^{1,3}

¹The MIND Institute, Albuquerque, NM

²Olin Neuropsychiatry Research Center, Hartford, CT

³Department of Psychology, University of New Mexico, Albuquerque, NM

Abstract

Recent work suggests that a generalized error-monitoring circuit, which shows heightened activation when one commits an error in goal-directed behavior, may exhibit synonymous activity when one watches another person commit a similar goal-directed error. In the present study, fMRI was utilized to compare and contrast those regions that show sensitivity to the performance, and to the observation, of committed errors. Participants performed a speeded go/nogo task, and also observed a video of another person performing the same task. Dorsal anterior cingulate, orbitofrontal cortex, and supplementary motor regions were commonly activated to both performed and observed errors, providing evidence for common neural circuitry underlying the processing of one's own, and another's mistakes. In addition, several regions, including inferior parietal cortex and anterorostral and ventral cinguli, did not show activation during performed errors, but were instead uniquely activated by the observation of another's mistakes. The unique nature of these 'observer-related' activations suggests that these regions, while of potential import towards recognition of another's errors, are not core to circuitry underlying error-monitoring. Rather, we suggest that these regions may represent components of a distributed network important for the representation and interpretation of complex social actions.

The ability to monitor and identify errors in goal-directed behavior exists as an integral component of effective behavioral regulation. Electrophysiological, neuroimaging and lesion research have converged in identifying the anterior cingulate (ACC) as a central component of a distributed neural network underlying these error monitoring processes (Falkenstein et al. 1991; Gehring et al. 1992; Gemba et al. 1986; Hogan et al. 2006; Kiehl et al. 2000; Swick and Turken 2002; Ullsperger and von Cramon 2006). Functional magnetic resonance imaging (fMRI) has demonstrated increased ACC activity after both errors (Mathalon et al. 2003; van Veen and Carter 2002) and negative feedback (Holroyd et al. 2004; Ruchow et al. 2002) concurrent with additional activity in medial prefrontal regions (Menon et al. 2001). Furthermore, the ACC is believed to serve as the primary generator of the error-related negativity (ERN/Ne), a negative-going event-related potential (ERP) that manifests after the commission of an error on a wide variety of laboratory tasks (Falkenstein et al. 1991; Gehring et al. 1995; Gehring et al. 1992), and also after the receipt of negative feedback indicating such

Please send all correspondences to: Dr. Matthew S. Shane, The MIND Institute, 1101 Yale Blvd. NE, Albuquerque, NM, 87106, Phone: (505) 272-4374, Fax: (505)272-8002, Email: mshane@themindinstitute.org.

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an error (Hajcak et al. 2005; Miltner et al. 1997; Muller et al. 2005). Dipole source analyses have confirmed the ERN's generator to be associated with activity within the ACC (Dehaene et al. 1994; Herrmann et al. 2004; Luu and Tucker 2001; Luu et al. 2003), with additional contributions from orbitofrontal and medial-frontal regions (Luu and Tucker 2001). Consistent with a role for ACC in executive control (Cunnington et al. 2006; Devinsky et al. 1995; Vogt et al. 1992), the activity of this neural network is believed integral for the top-down guidance and selection of contextually-appropriate action patterns (Cunnington et al. 2006; Luu and Tucker 2001; Luu et al. 2003). Indeed, recent work suggests that error-related ACC activity serves as a learning mechanism intended to adjust behavior to maximize avoidance of future goal-directed errors (Magno et al. 2006).

While much learning occurs through direct experience, humans and other organisms also have the capacity to learn through observation of others (Dawson and Foss 1965; Fiorito et al. 1990; Pollock and Lee 1992). Social etiquette, for instance, is largely learned through observation of other's behavior (Blakemore et al. 2004). Moreover, a substantial body of work has demonstrated improved performance on simple or complex motor tasks after watching skilled or unskilled models (Blandin and Proteau 2000; Carroll and Bandura 1982; Pollock and Lee 1992). Monkeys show an ability to learn to use a stick to reach food outside their cage if such behavior is initially demonstrated by a human model (Tomasello, Davis-Dasilva, Camak & Bard 1987). Similar capacities for observational and social learning have been demonstrated in dogs (Adler and Adler 1977), dolphins (Krutzen et al. 2005), octopi (Fiorito et al. 1990) and pigeons (Biederman et al. 1986). Such learning makes possible the acquisition of knowledge without direct personal experience, and allows for the prevention of common errors by observing the misfortune of others.

Recent electrophysiological (EEG) work has reported the presence of a negative deflection in the ERP that manifests when one watches another person make a mistake (referred to here within as the 'observer ERN' or 'oERN' (Bates et al. 2005; Miltner et al. 2004; van Schie et al. 2004). This ERP component appears to peak between 200–300 ms after the error is observed (Miltner et al. 2004), with initial dipole modeling suggesting a dorsal ACC-based generator (Miltner et al. 2004). This convergence of spatial and temporal characteristics has led to the suggestion that the oERN may exist as an ERN-like waveform, and may thus serve as a training mechanism (van Schie et al. 2004) that fosters the ability to identify, and learn from, the mistakes of others.

While EEG boasts precise temporal specificity, its relatively vague spatial specificity prevents firm conclusions regarding the brain regions responsible for the oERN's generation. Thus, while current evidence supports the notion that error-related activity presents following the observation of another's errors, techniques with greater spatial sensitivity, such as fMRI, will be required to confirm the brain regions underlying this response. Here we use fMRI to interrogate the activity associated with both the performance and the observation of errors, to gain a better understanding of the shared and unique circuitry underlying the processing of each error-type. Participants either performed, or observed another person performing, a speeded Go/No Go task designed to elicit errors. While performing the task, participants were required to press a button every time a 'Go' stimulus was presented, and to withhold their button-press for all 'No Go' stimuli. While observing the performance of another, the participant needed only to press one of two buttons to indicate if the actor successfully or unsuccessfully withheld their response to each 'No Go' stimulus. This design allowed us to investigate the neural circuitry underlying the monitoring of one's own, and another's errors, and to evaluate regions of overlap and distinction between these two processes.

We focused on the ACC, creating regions of interests (ROIs) to define regions that have shown error-related activity in previous work (Falkenstein et al. 1991; Gehring et al. 1992; Holroyd

and Coles 2002; Kiehl et al. 2000). Bush et al. (2000) have argued that error-related activity may shift from dorsal to rostroventral ACC as the context within which the error was made becomes increasingly affective (see also Bush et al. 2000; Polli et al. 2005; Whalen et al. 1998). One possibility is that the dorsal subdivision, with strong reciprocal connectivity to lateral prefrontal cortex (BA 46/9), parietal cortex (BA 7), and premotor and supplementary motor areas, is primarily responsible for cognitive aspects of error-monitoring, including the modulation of attention and the monitoring for response competition. The rostral subdivision, with stronger interconnectivity with limbic structures and medial frontal cortex, may, in turn, be more responsible for the emotional and motivational aspects of error-monitoring. Previous research from our laboratory has demonstrated both dorsal and rostral ACC activity in response to committed errors on the Go/No Go task implemented in the present research (Kiehl et al., 2000). We thus anticipated increased ACC activity within both dorsal and rostral subdivisions after participants committed their own mistakes.

Less understood is the error-related circuitry that activates upon observation of another person's errors. ERP studies suggest a similar dorsal ACC generator for the oERN. In addition, however, one may anticipate regions with involvement in the observation of action, and/or the processing of social information, to be uniquely active during the observation of another's errors. To this end, a fairly specific region of rostral ACC (anterior aspects of BA24, beginning approximately inline with the genu, and extending ventrally to include ventral ACC (BA25)) has recently been identified as existing within a widely-distributed "default on" network (Gusnard et al. 2001; Iacoboni et al. 2005; Raichle et al. 2001) involved in the processing of social and "other-referential" information, including the observation of hand and foot movements (Jackson et al., 2004), the observation of videos depicting persons (versus videos depicting objects; Mitchell et al., 2002), and during judgments of strangers (versus judgments about one's self; Ochsner et al., 2005). With this literature in mind, we anticipated observer-specific activations within this antero-rostral/ventral ACC region, not due to core involvement in error-monitoring, but rather due to a more general involvement in the processing of other-referential information.

Our hypotheses were thus two-fold. First, we hypothesized increased hemodynamic response within dorsal and/or rostral ACC during both the performance and the observation of errors. Second, we hypothesized that antero-rostral and ventral cingula would show a unique sensitivity to observed, but not to performed, errors. Together, these findings would serve as the first fMRI evidence for ACC involvement in the processing of observed errors, and would provide a gateway for future research investigating the underpinnings of social and observational learning.

Method

Participants

Participants were 21 healthy screened right-handed volunteers (15 men) ranging in age from 18 to 50 ($m = 28.48$; $sd = 9.07$). Recruitment was undertaken via advertisements and word of mouth at the Olin Neuropsychiatry Research Center (Hartford, CT). Participants provided written informed consent in protocols approved by Hartford Hospital's Institutional Review Board. All research procedures were conducted in adherence to ethical standards required for human subject protection.

Experimental Design

Each participant completed the performance condition, followed by the observation condition. While we recognize the inherent confound created by this consistent order, we established this

methodology to ease participants' comprehension of the observation condition by first having them perform the task themselves.

In the performance condition, participants were required to perform two runs of a challenging Go/No-go task designed to elicit errors. This task was identical to a paradigm described in previous reports (see Bates et al. 2004; Kiehl et al. 2000; Shane, Stevens, Harenski & Kiehl, under review), and involved the speeded, pseudo-random presentation of X's (the 'Go' stimulus, presented 412 times (~80% of trials)) and K's (the 'No-go' stimulus, presented 78 times, (~20% of trials)). Stimuli were presented on-screen for 1000 ms, with an inter-stimulus interval jittered between 750 ms, 1000 ms, and 1250 ms. Participants were instructed to go as quickly and as accurately as they could, and were reminded of this instruction between runs.

In the observation condition, participants watched two 7-minute videos portraying an actor (the first author) seated at a computer, performing the same go-nogo task described above (see Materials section for more details on the video). As in the performance condition, the actor was required to press the button on the response box with his index finger every time an 'X' was presented, and to withhold his button-press every time a 'K' was presented. Using a predetermined order, the actor successfully withheld his button-press for half of the 'K' presentations (30 trials), and committed a button-press error for the other half of the 'K' presentations (30 trials). While watching the video, the observer was instructed to press one of two buttons every time a 'K' was presented to the actor. If the actor successfully withheld his button-press response, the observer was required to press the button with the index finger on his/her right hand; if the actor made a button-press error, the observer was required to press the button with the middle finger on his/her right hand (finger mappings were counter-balanced across subjects). This button-press requirement was included in the study design quite purposely, to avoid characterizing the observation condition as a passive-viewing condition. While concerns related to passive-viewing paradigms have been discussed (Savoy, 2005), our primary motivation was concern for motor-related differences between the performance and observation conditions. Cingulate motor regions show robust response after a variety of motor actions (Devinsky, Morrell & Vogt, 1995; Shima & Tanji, 1998). By requiring that participants press a button to all observed errors, similarity was maximized across the observed error and performed error trials (which were necessarily characterized by a button-press response).

Materials—The videos were filmed with a Canon 2R40 digital video camera set up on a tripod behind the actor's right shoulder, and thus displayed an "over-the-shoulder" view of the computer, and of task performance. The actor was facing the computer monitor, providing the participant with a view of the back of the actor's head, his right shoulder and arm, and his right hand. The actor's right hand rested on a four-button response box with which all responses were made (see Figure 1 for a still-shot of the video). Effort was taken to ensure that the primary stimuli in the video – the presented Go/No-go stimuli, and the actor's button press responses – were centered as closely as possible within the video frame. To this end, feedback from piloting, and from participant performance, indicated that participants were fully capable of attending to both stimuli without decreasing identification of the actor's responses.

Imaging Parameters

A Siemens Allegra 3T system located at the Olin Neuropsychiatry Research Center was utilized for the scanning protocol. Each participant's head was firmly secured using a custom head holder, and head motion was restricted using a custom-built cushion inside the head coil. Localizer images were acquired to determine functional image volumes. The echo planar image (EPI) gradient-echo pulse sequence (TR/TE = 1500/28 ms; flip angle = 65°; FOV = 24 × 24 cm; 64 × 64 matrix; 3.4 × 3.4 mm in plane resolution; 5 mm effective slice thickness; 30 total slices) effectively covered the entire brain (150 mm) in 1.5 s. Each video lasted just over 7

minutes, or 281 scans. A 9-second rest period was included prior to the start of the task in each run to allow for T_1 effects to stabilize. The six initial images from these 9-seconds were not included in the reported analyses.

Image Processing

Functional images were reconstructed offline and reoriented to approximately the anterior commissure/posterior commissure (AC/PC) plane. Functional image runs were motion corrected using an algorithm unbiased by local signal changes (INRIAlign, Freire and Mangin 2001) as implemented in Statistical Parametric Mapping II (SPM2). No participants showed head movements in excess of 5mm, and were thus all retained for the analyses reported below.

A mean functional image volume was constructed for each run from the realigned image volumes. The mean EPI image was normalized to the EPI template. The spatial transformation into standard MNI space was determined using a tailored algorithm with both linear and nonlinear components (Friston et al. 1995). The normalization parameters determined for the mean functional volume were then applied to the corresponding functional image volumes for each participant. The normalized functional images were smoothed with a 9 mm full width at half-maximum (FWHM) Gaussian filter. Event-related responses were modeled using a synthetic hemodynamic response function composed of two gamma functions. The first gamma function modeled the hemodynamic response using a peak latency of 6 sec. A term proportional to the derivative of this gamma function was included to allow for small variations in peak latency. The second gamma function and associated derivative was used to model the small “overshoot” of the hemodynamic response on recovery. High-pass (cutoff period 116 s) and low-pass (cutoff period .23) filters were applied to remove any low- and high-frequency confounds, respectively. A latency variation amplitude-correction method was used to provide a more accurate estimate of hemodynamic response for each condition (Calhoun et al. 2004).

Data Analytic Strategies

Following preprocessing of the functional images, group analyses utilized a random effects model to determine voxel-wise t -statistics. At the first level, mean images corresponding to correct responses, incorrect responses, and successful inhibitions were computed and condition-specific images were contrasted using the general linear model. The study design afforded the opportunity to independently evaluate hemodynamic response to performed errors and to observed errors, by comparing error trials (performed or observed) to correct responses and to successful inhibitions. Differential activation in these condition-specific contrasts was assessed with second level t -statistics. In addition, hemodynamic response to performed and observed errors was contrasted directly, utilizing paired-sample t -statistics to evaluate activity unique to the observation of another’s errors. Finally, regions of common activation across both performed and observed errors were interrogated by creating a mask of all regions active in the ‘Performed error minus Performed correct’ contrast, filtering regions active in the ‘Observed error minus Observed correct’ contrast through this mask.

Our *a priori* hypotheses were constrained to specific regions within the ACC. Specifically, three regions of interest (ROI) were established, by implementing 10mm spheres of interest within each of the dACC (center coordinate: $x = 4, y = 22, z = 40$), rACC (center coordinate: $x = -8, y = 45, z = 15$), and vACC (center coordinate: $x = 3, y = 23, z = -8$). Central coordinates for the dACC and rACC ROIs were based on our own previously published findings reporting error-related activity localized to these voxels (Kiehl et al. 2001). Central coordinates for the vACC ROI were based on an averaging of coordinates across a relevant spectrum of previous studies (Gusnard et al. 2001; Jackson et al. 2006; Moran et al. 2006; Ochsner et al. 2005) that have reported observer-related, and/or other-referential activity localized to the vACC. Activations within these ROIs were subjected to small volume correction (SVC) within SPM2,

and were further bonferonni corrected for number of search regions. We also interrogated the data in a more exploratory whole-brain fashion, in order to evaluate regions activated outside of our ROIs. Unless otherwise noted, all findings were thresholded at $p = .001$, uncorrected and utilized an extent threshold of 5 contiguous voxels.

Results

First, we report hemodynamic activity associated with participant's own performance on the go/nogo task. Second, we report hemodynamic activity associated with participant's observation of another person performing the go/nogo task. Third, we directly compare these areas of activation, and report hemodynamic activity common and unique to the processing of both error types.

Performed Go/No Go task

Behavioral Data—Participants correctly responded to 94.83% of Go trials, and 66.33% of No go trials. Participants averaged 27.53 errors throughout the task (33%; range 13–49; $sd = 10.08$). Reaction times on false alarm error trials (344ms) tended to be slightly faster than reaction times on successful hit trials (373ms; $t = -5.62$, $p < .001$).

Imaging Data

Button Press Errors vs. Correct Button Presses: Consistent with the broad error monitoring literature, we hypothesized an increase in dorsal and/or rostral ACC activity after incorrect, compared to correct, responses. As displayed in Figure 2, ACC activity increased (bilaterally) after error responses compared to correct responses. This ACC activity appeared to generate on the border between dorsal and rostral ACC, and showed spreading activation within each subdivision.

Additional cortical regions showing error-related activity included right orbitofrontal and middle frontal cortices, as well as right orbitofrontal cortex and left supramarginal cortex. Subcortical regions of error-related activity included bilateral thalamus, left insula, and right hippocampus. Table 1 provides a complete list of activated regions. No regions showed significant deactivation to errors compared to correct responses ($p < .001$, uncorrected).

Button Press Errors vs. Successful Inhibitions: Button press errors could also be contrasted against successfully inhibited button presses (on nogo trials), which is a contrast that controls for the novelty inherent in the NoGo stimuli. This contrast also manifested bilateral ACC activity that originated near the dorsal/rostral border and spread into both ACC regions. Additional regions of activation included right supplementary motor area, left insula, and left superior temporal cortex (see Figure 3). A complete list of activated regions is listed in Table 2.

Observed Go/No Go task

Behavioral Data—With the exception of one individual, all participants identified the actor's correct and incorrect actions with a high degree of accuracy (82%). One individual performed at less than chance levels (only identified 46% of trials correctly), and was thus removed from further analyses. For all other participants ($n = 20$), all trials in which the actor's successful and unsuccessful responses were accurately identified were used to create individual image maps for each participant.

Imaging Data

Observed Errors vs. Observed Correct Responses: Consistent with recent ERP evidence, we anticipated increased dorsal ACC activity in response to the observation of another person's errors, compared to observation of successful responses. Indeed, we found broadly increased activity associated with observed errors in bilateral ACC (see Table 3 and Figure 4). Peak ACC activations were localized to a region close to the dorsal/rostral division, appeared to include both dorsal and rostral aspects, and extended into adjacent medial frontal regions. In addition, specific activity within hypothesized regions of ventral cingulate were identified, that did not manifest in the comparable contrast with performed errors.

Error-related activity was also observed in the supplementary motor region, likely representing motor activity associated with the participant's button press to identify observed errors. In addition, substantive left-sided activity within inferior parietal and orbitofrontal cortices also showed specific error-related activations.

Observed Button Press Errors vs. Observed Successful Inhibitions: Contrasting observed errors to observed successful inhibitions elicited increased activity within bilateral ACC and right medial-frontal cortex. The ACC activity appeared to include unique generators within both rostral and ventral ACC subdivisions, and corresponded with the rostral and ventral ACC regions of interest. This activity also extended down into medial prefrontal cortex (see Figure 5). Table 4 lists all regions showing increased activity related to observed errors. No regions showed significant deactivations.

Activity Common to the Performance and Observation of Errors

Activity common to the processing of performed and observed errors was obtained by creating a functional mask of the 'Performed errors minus Performed correct' contrasts, and using those masks as a filter for viewing regions active in the 'Observed errors minus Observed correct' contrasts. Areas of common activity are denoted with italics in Table 3. Consistent with hypotheses, both dorsal and rostral ACC showed common activity, as did regions within right orbitofrontal cortex, left supramarginal cortex, and bilateral thalamus.

Activity Unique to the Observation of Errors

In order to better understand that circuitry unique to the observation of goal-directed errors, we contrasted those regions that showed increased activity after the observations of other's errors than after the performance of one's own errors. It is important to recognize that errors exist as a constant within this contrast; thus, the contrast is specifically designed to identify circuitry central to differentiating between self- and other-referential errors. As can be seen in Figure 6, major regions of activation included regions of inferior and superior parietal cortices and medial temporal cortex. Table 5 reports all regions showing increased activity to observed errors.

Activity Unique to the Performance of Errors

Interestingly, only regions within primary and secondary motor cortices showed increased activity to performed errors compared to observed errors. Increased activity within these regions makes theoretical sense, as the button-press response in the course of performing the task was likely more "powerful" than was the response towards identification of the observed response. The lack of increased activity in other brain regions may be interpreted as further evidence for the similarity of processing one's own, and another's behaviors.

Discussion

Substantial EEG, fMRI and lesion evidence has implicated dorsal and rostroventral ACC, as well as diffuse medial frontal regions, in the monitoring of one's own errors (Kiehl et al. 2000; Holroyd & Coles 2002; Luu et al. 2003; Carter et al. 2004). The present work confirms recent EEG work (Bates et al. 2005; Miltner et al. 2004; van Schie et al. 2004) in demonstrating that these circuits are also engaged during the observation of another person making a mistake. Specifically, we report increased hemodynamic activity within both dorsal and rostral ACC after the performance of one's own errors, as well as after the observation of another's errors. These findings serve as the first hemodynamic evidence for ACC involvement in error observation, and provide initial support for the notion that the performance and observation of goal-directed errors activate similar underlying circuitry.

Previous research has demonstrated that post-error ACC activity shows substantial association with error-correction (Cohen et al. 2000), and with the ability to learn through trial-and-error (Holroyd and Coles 2002; Pratt et al. submitted). Recent theories thus posit error-related ACC activity as conferring guidance over contextually-appropriate action patterns (Cunnington et al. 2006; Luu and Tucker 2001; Luu et al. 2003), and as a training mechanism to promote post-error remediation of maladaptive behavior (Holroyd and Coles 2002; Magno et al. 2006). If this is true, then the recruitment of similar underlying circuitry for the processing of another's errors may serve an analogous role in the guidance of future action plans. Specifically, ACC activity related to error-observation may aid prevention of future errors by training the observer to avoid witnessed mistakes. In this manner, the avoidance of maladaptive actions need not wait until a goal-directed error is identified; rather, such action plans can be prevented even before they are committed by noting the success or failure of those who have tread before us.

The processing of success and failure can involve both cognitive and emotional components. Bush et al. (2000) have suggested that dorsal and rostral ACC are uniquely involved in the cognitive and emotional aspects of error-processing, respectively. Consistent with this notion, dorsal ACC shows strong reciprocal connectivity to areas involved in cognitive control including dorsolateral prefrontal cortex (Cohen et al. 2005), while rostral ACC shows stronger interconnectivity with limbic regions (Pandya and Yeterian 1996; Vogt and Pandya 1987). In the present study, we report activations that spread across both dorsal and rostral subdivisions during the performance and observation of goal-directed errors. This finding is in agreement with previously published research from our laboratory utilizing the same Go/No Go task (Kiehl et al. 2001), and suggests that there may be both cognitive and affective components to the processing of one's own, as well as another's, errors.

Activity common to the performance and observation of errors was also seen in additional regions, including thalamus and orbitofrontal cortex. Based on previous research, we would not presume direct involvement of these regions in the processing of error-related information. Rather, it is likely that activity in these regions represent supportive roles, perhaps towards the guidance of goal-directed behavior based on encountered outcome contingencies. Orbitofrontal cortex is well-established to track outcome desirability (Schultz & Dayan, 1997), and to show sensitivity on the basis of reward and punishment (Tremblay & Schultz, 1999). The thalamus, in turn, is believed to serve to relay and modulate cognitive and emotional information to further downstream targets (Sherman, 2001). Together with ACC, these regions may aid contingency-tracking and action-selection in the service of goal-directed behavior.

Recognizing and representing errors in another person's goal-directed behavior is not as simple as recognizing one's own errors. Accurate identification requires that the observer a) represent the actor's action patterns, b) understand the actor's goals and motivations, and c) code the success or failure of the actor's performance in lieu of these represented goals. It is unlikely

that the ACC performs each of these functions. Rather, we would suggest a more hierarchical organization, wherein ACC receives inputs from a variety of regions and utilizes this information to monitor and code the eventual outcome of the observer's response.

To this end, it is important to note several regions that have previously been identified as involved in the observation of action. Regions within inferior parietal cortex have shown robust activity upon presentation of another's hand or body movements (Grafton et al. 1996; Iacoboni et al. 1999; Rizzolatti et al. 1996) that have led researchers to posit involvement of these regions in the internal representation of basic motor movements (Decety and Chaminade 2003; Iacoboni et al. 1999; Jeannerod and Frak 1999). Similarly, regions within anterior and ventral ACC (r/vACC) as well as within surrounding ventromedial prefrontal cortex, have in turn shown activation during self-judgments (Ochsner et al. 2005), social cooperation (Rilling et al. 2006), observation of movie clips displaying human actors (Iacoboni et al. 2004), joint attention experience (Williams et al. 2005), the observation of hand and foot movements (Jackson et al. 2004), the observation of videos depicting persons versus videos depicting objects (Mitchell et al. 2002), and during judgments of strangers, versus judgments about one's self (Ochsner et al. 2005). In the present study, we found substantive activity within both inferior parietal and r/vACC that was unique to the observation of another's errors. In concert with the existing literature, we interpret this activity as underlying an important role for these regions in the observation and representation of action. Within this vein, we suggest that ACC may exist as a high-level component within a distributed network, and may utilize inputs from regions including inferior parietal and r/vACC toward determination of success and failure. Indeed, both functional (Awh and Jonides 2001) and anatomical (Pandya et al. 1981), linkages between dACC and inferior parietal cortex have been discussed, and all three regions are believed to reside within a "default-on" network underlying self-referential processing (Gusnard et al. 2001; Raichle et al. 2001).

It may be fundamental to note that r/vACC and IPC activity were not always revealed by identical contrasts. In particular, whereas both regions showed sensitivity when observed errors were compared to observed correct responses, only IPC activity also showed sensitivity when observed errors were directly compared to performed errors. While one could infer from this that the IPC activity may be more reliable or robust, we prefer a subtler interpretation that could highlight an important distinction between IPC and r/vACC contributions to observed action. Note that in the 'observed error minus performed error' contrast, the processing of errors was held constant, and only the perspective (self/other) was varied. Thus, IPC activity evidenced by this contrast may index a fundamental role for IPC in the distinction between self-initiated and other-initiated actions. In the 'observed error minus observed correct' contrast, however, the observation of action was held constant, while the success, or lack thereof, of that action was varied. This contrast thus interrogates regions involved in a more contextually-mediated processing of the observed behaviors, and we speculate that the IPC and v/rACC activity noted within these contrasts indicate their role in such contextually-mediated processing. One possibility, then, is that IPC is involved in the basic representation of observed action patterns, and that both IPC and v/rACC share responsibility for interpreting this behavior with reference to broader contextual details. If this is true, we may further speculate that processing performed by IPC and/or v/rACC, in the service of contextually-guided translation of observed action, may support subsequent processing within the distributed error-monitoring network that includes dorsal and rostral ACC and medial-frontal cortex

In conclusion, we report increased activity within dorsal, rostral and ventral ACC, as well as within additional regions with known involvement in the execution and observation of action. Dorsal and rostral ACC may underlie cognitive and emotional aspects of error processing, respectively, whereas ventral ACC may represent the translation of contextually-mediated information into terms relevant for the self. IPC activity, in turn, may play a role in both basic

and contextually-mediated processing of observed actions. Each of these regions may serve a supportive role in guiding error-related learning, and subsequent research may benefit from increased consideration of the manner in which they influence processing of observed action.

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Figure 1.
Still-shot of the video shown to participants while in the scanner.

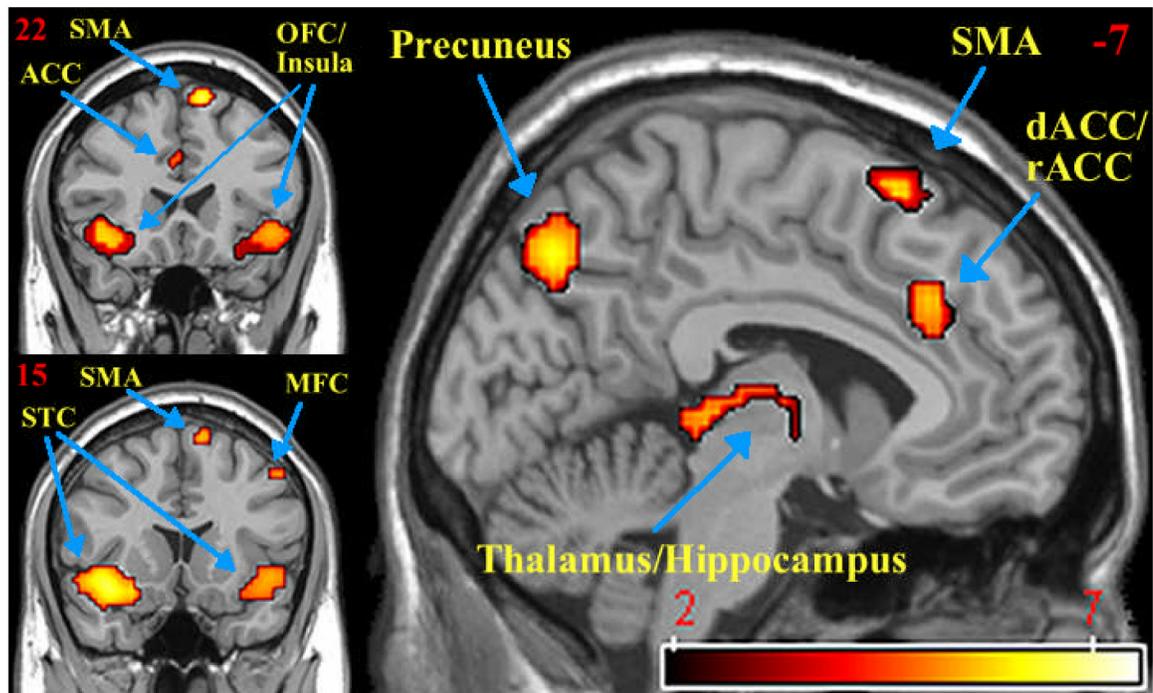


Figure 2.
Regions demonstrating increased activity to performed button press errors compared to performed correct button presses.
ACC = anterior cingulate; dACC = dorsal anterior cingulate ; rACC = rostral anterior cingulate ;
MFC = medialfrontal cortex; OFC = orbitofrontal cortex; SMA = supplementary motor area;
STC = superior temporal cortex. Image thresholded at $p < .0001$, uncorrected.

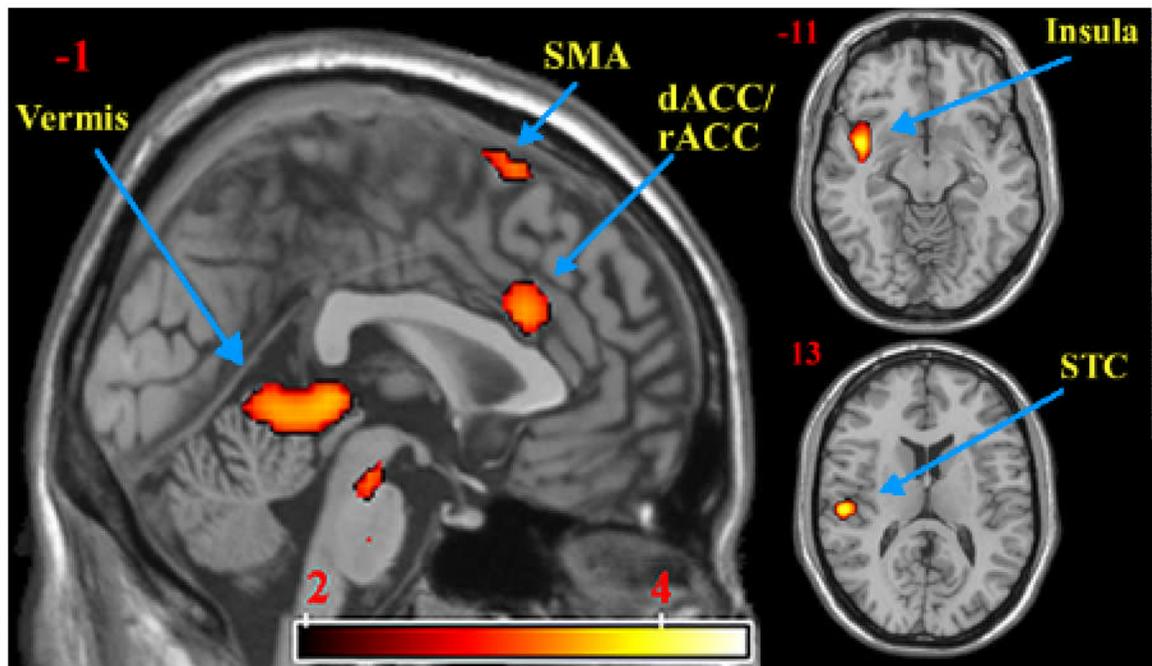


Figure 3. Regions demonstrating increased activity to performed button press errors compared to successfully performed inhibitions. dACC = dorsal anterior cingulate ; rACC = rostral anterior cingulate ; SMA = supplementary motor area; STC = superior temporal cortex. Image thresholded at $p < .0001$, uncorrected.

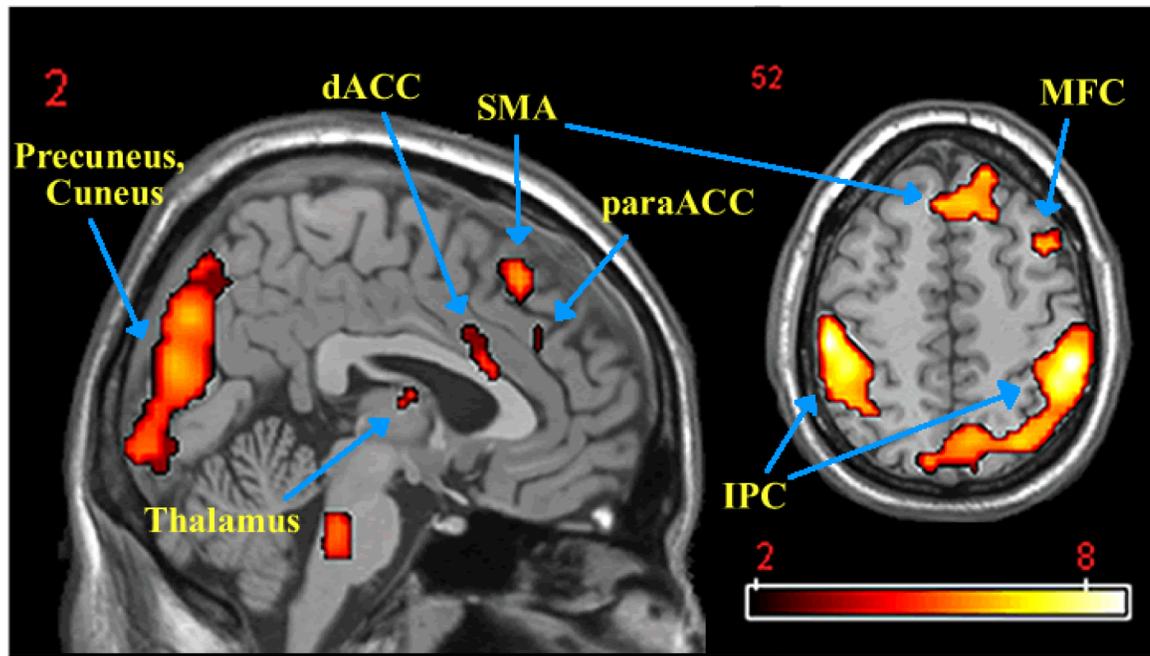


Figure 4. Regions demonstrating increased activity in response to observed button press errors compared to observed correct button presses. dACC = dorsal anterior cingulate ; IPC = inferior parietal cortex; paraACC = paracingulate cortex; MFC = medialfrontal cortex; SMA = supplementary motor area. Image thresholded at $p < .001$, uncorrected.

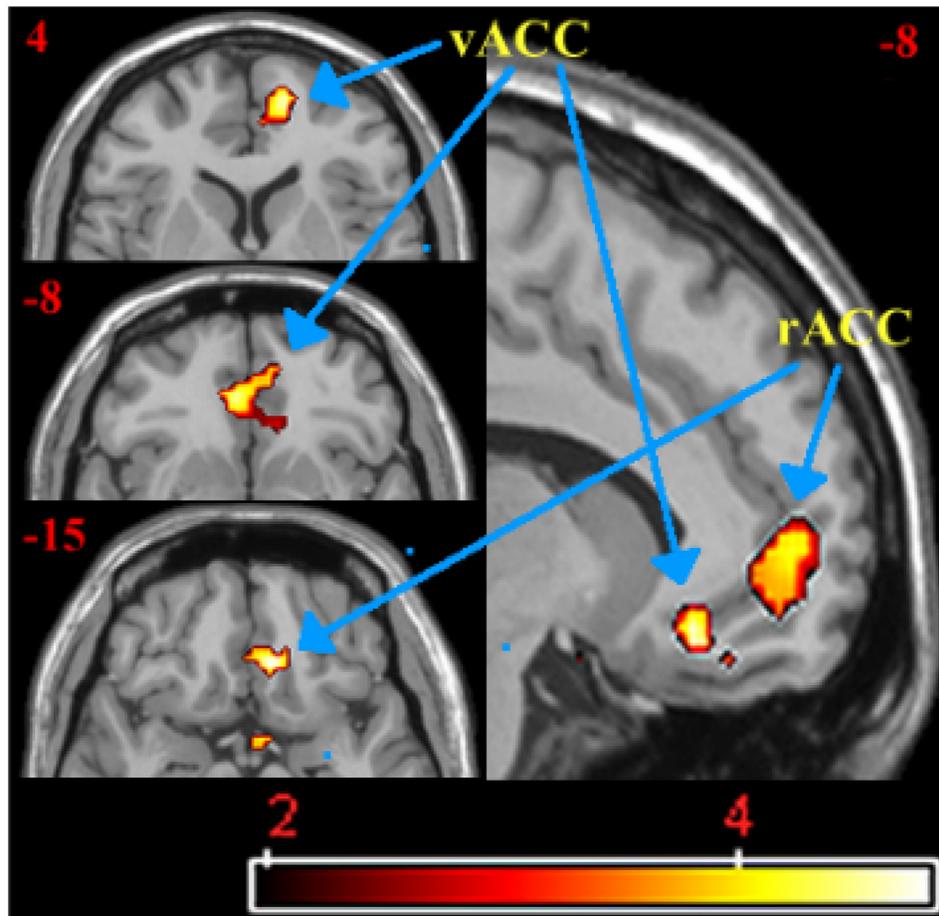


Figure 5. Regions demonstrating increased activity in response to observed button press errors compared to observed successful inhibitions. rACC = rostral anterior cingulate; vACC = ventral anterior cingulate. Image thresholded at $p < .001$, uncorrected.

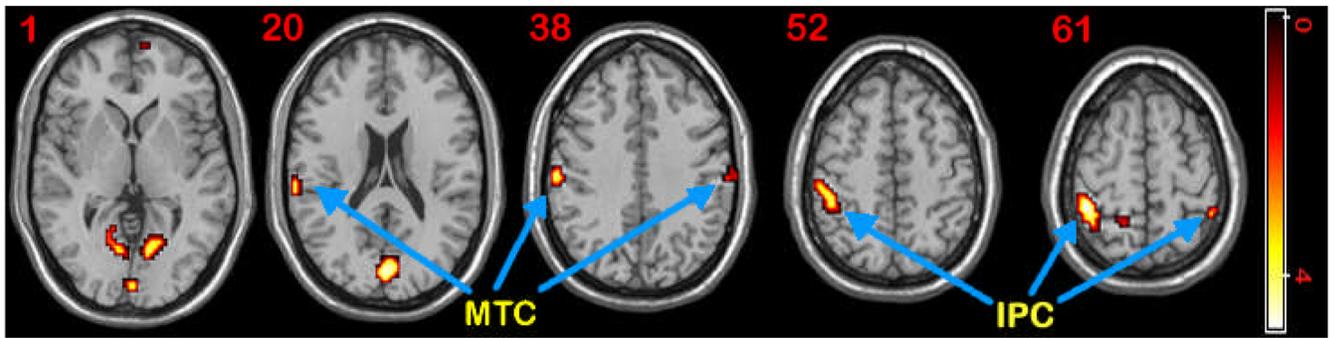


Figure 6. Regions demonstrating increased activity in response to observed button press errors compared to performed button press errors. MTC = superior temporal cortex; IPC = inferior parietal cortex. Image thresholded at $p < .001$, uncorrected.

Table 1

Regions showing increased activity to performed errors compared to correctly performed response.

Region	L/R	Estimated Brodmann Area	Peak Voxel Coordinates	t-scores
Insula	L	13	-39,15, -15	6.54*
Supramarginal Cortex	L	40	-63, -45, 36	6.03
SMA	R	6	9, 21, 63	5.91
Precuneus	R	7	3, -72, 45	5.72
Cerebellum	L	37	-39, -51, -27	5.69
Dorsal ACC	R	32	6, 30, 33	5.59
Thalamus	-	-	0, -24, 6	5.54
Orbitofrontal Cortex	R	47	45, 21, -9	5.51
Middle Frontal Cortex	R	8	48, 12, 45	5.44
Hippocampus	R	-	18, -33, -3	4.98
Thalamus	R	-	9, -3, 6	4.79
Brainstem	R	-	3, -21, -33	4.70
Rostral ACC	L	32	-9, 39, 12	4.38

Bolded regions FWE-corrected for 10mm sphere small-volume search and bonferonni corrected for multiple comparisons. Dorsal ACC coordinates: x = 4, y = 22, z = 40; Rostral ACC coordinates: x = -8, y = 45, z = 15).

* p < .05 FWE-corrected for whole-brain volume.

All other activations $p < .0001$, uncorrected for whole-brain volume.

Table 2

Regions showing increased activity to performed errors compared to correctly performed inhibitions.

Region	L/R	Estimated Brodmann Area	Peak Voxel Coordinates	t-scores
Superior Temporal Cortex	L	38	-45,3,-12	3.79
Cuneus	L		3, -93, 30	3.44
Lingual	R	-	3, -36, 0	3.37
Brainstem	L	-	-15, -24, -39	3.31
Anterior Transverse Temporal Cortex	L	41	-57, -21, 15	3.30
Dorsal ACC	L	24	-3, 24, 27	3.19
SMA	R	6	9, 21, 66	3.18
Dorsal ACC	-	32	0, 24, 30	2.99

Bolded regions corrected for 10mm sphere small-volume search and bonferonni corrected for multiple comparisons around the following coordinates: Dorsal ACC coordinates: x = 4, y = 22, z = 40).

All other activations $p < .005$, uncorrected for whole-brain volume.

Table 3

Regions showing increased activity to observed errors compared to observed correct responses.

Region	L/R	Estimated Brodmann Area	Peak Voxel Coordinates	t-scores
Inferior Parietal Cortex	R	40	51, -36, 48	9.63*
Postcentral Cortex	L	2	-54, -27, 51	8.21*
Inferior Parietal Cortex	L	40	-39, -54, 60	7.48
Middle Occipital Cortex	L	7/19	-45, -81, 0	7.56
Cerebellum	R	-	39, -42, -36	7.38
<i>Cerebellum</i>	L	-	-39, -57, -36	7.30
Middle Frontal Cortex	R	46	45, 39, 30	7.20
<i>Orbitofrontal Cortex</i>	R	11	21, 42, -21	7.18
Middle Temporal Cortex	R	39	54, -63, 3	7.07
<i>Thalamus</i>	L	-	-12, -6, 12	6.92
Inferior Frontal Tri Cortex	R	45	57, 18, 24	6.91
<i>Supramarginal Cortex</i>	L	40	-63, -24, 18	6.89
<i>Thalamus</i>	R	-	27, -24, 3	6.81
Calcarine	R	-	12, -69, 6	6.88
Middle Frontal Cortex/SMA	R	6/46	39, 6, 60	6.73
<i>Dorsal ACC</i>	R	33	3, 27, 45	5.49
<i>Dorsal ACC</i>	-	32	0, 15, 33	4.76
<i>Rostral ACC</i>	L	24	-9, 36, 18	4.20
<i>Ventral ACC</i>	R	25	12, 21, -9	4.05

Bolded regions FWE-corrected for 10mm sphere small-volume search and bonferonni corrected for multiple comparisons around the following coordinates: Dorsal ACC coordinates: x = 4, y = 22, z = 40; Rostral ACC coordinates: x = -8, y = 45, z = 15; Ventral ACC coordinates: x = 2, y = 23, z = -7).

* $p < .01$, FWE-corrected for whole-brain volume.

All other activations $p < .05$, FWE-corrected for whole-brain volume. Italicized regions indicate those regions that also demonstrated increased activity in the performance condition.

Table 4

Regions showing increased activity to observed errors compared to observed correct inhibitions.

Region	L/R	Estimated Brodmann Area	Peak Voxel Coordinates	t-scores
Ventral ACC	R	32	6, 30, -12	4.80*
Medial Frontal Cortex	R	10	12, 57, 6	4.68*
Rostral ACC	L	32	-6, 42, -6	4.59*
Caudate	L	-	3, 3, -15	4.12

Bolded regions FWE-corrected for 10mm sphere small-volume search and bonferonni corrected for multiple comparisons around the following coordinates: Dorsal ACC coordinates: x = 4, y = 22, z = 40; Rostral ACC coordinates: x = -8, y = 45, z = 15; Ventral ACC coordinates: x = 3, y = 23, z = -8).

* $p = .0001$, uncorrected for whole-brain volume.

All other activations $p < .001$, uncorrected for whole-brain volume.

Table 5

Regions showing differential activity to observed errors compared to performed errors.

Region	L/R	Estimated Brodmann Area	Peak Voxel Coordinates	t-scores
<i>Observed > Performed</i>				
Inferior Parietal Cortex	L	40	-45, -39, 60	4.76*
Cerebellum	L	-	-33, -45, -51	4.68*
Cuneus	-	-	0, -81, 21	4.67*
Postcentral Cortex		3	-66, -24, 18	4.59
Posterior Cingulate	R	30	12, -66, 9	4.55
Inferior Parietal Cortex	L	40	-60, -21, 42	4.34
Superior Parietal Cortex	L	7	-18, -48, 63	4.02
Inferior Parietal Cortex	L	40	-54, -27, 51	3.99
Middle Temporal Cortex	L	39	-54, -72, 12	3.88
Precuneus	L	19	-15, -51, -3	3.84
Superior Medial Frontal Cortex	R	10	6, 66, 3	3.75
Postcentral Cortex	R	3	63, -18, 36	3.71
Inferior Parietal Cortex	R	40	45, -45, 60	3.66
<i>Performed > Observed</i>				
Primary Motor Cortex/SMA	L	4/6	-9, 0, 60	4.87*

* $p < .0001$, uncorrected for whole-brain volume.All other activations $p < .005$, uncorrected for whole-brain volume.