

# Avoiding another mistake: Error and posterror neural activity associated with adaptive posterror behavior change

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The magnitude of posterior medial frontal cortex (pmFC) activity during commission of an error has been shown to relate to adaptive posterror changes in response behavior on the trial immediately following. In the present article, we examined neural activity during and after error commission to identify its relationship to sustained posterror behavior changes that led to performance improvements several trials into the future. The standard task required participants to inhibit a prepotent motor response during infrequent *lure trials*, which were randomly interspersed among numerous *go trials*. Posterror behavior was manipulated by introducing a dynamic condition, in which an error on a lure trial ensured that the next lure would appear within two to seven go trials. Behavioral data indicated significantly higher levels of posterror slowing and accuracy during the dynamic condition, as well as fewer consecutive lure errors. Bilateral prefrontal cortex (PFC) and pmFC activity during the posterror period, but not during commission of the error itself, was associated with increased posterror slowing. Activity within two of these regions (right PFC and pmFC) also predicted success on the next lure trial. The findings support a relationship between pmFC/PFC activity and adaptive posterror behavior change, and the discrepancy between these findings and those of previous studies—in the present study, this relationship was detected during the posterror period rather than during commission of the error itself—may have resulted from the requirements of the present task. Implications of this discrepancy for the flexibility of cognitive control are discussed.

The neural mechanisms underlying error processing have been the subject of much research (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004); however, how error-related neural activity influences posterror changes in behavior remains relatively unclear. In particular, it is unclear which error-related neural processes are associated with sustained adaptive changes in behavior that help us avoid repeating a mistake. The ability to adjust our behavioral responses following an error is critical to everyday life and provides the foundation for learning many complex skills. Healthy adults learn from their mistakes remarkably

quickly, often making only one mistake before adapting their behavior (Noble, 1957). Conversely, difficulty in adjusting behavior following an error, or making the same error repeatedly, is characteristic of a number of clinical conditions (Barbarotto, Capitani, Jori, Laiacona, & Molinari, 1998; Kopp & Rist, 1994; Russell & Jarrold, 1998).

A range of evidence implicates the posterior medial frontal cortex (pmFC) in error processing (Gehring, Goss, Coles, Meyer, & Donchin, 1993; Ridderinkhof et al., 2004; Ullsperger & von Cramon, 2003) and includes the finding that many clinical conditions are as-

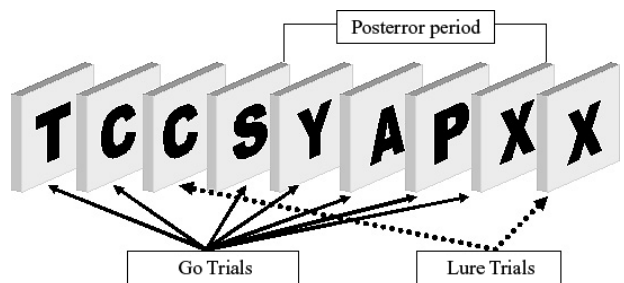
sociated with dysfunctional pMFC responses to errors (Carter, MacDonald, Ross, & Stenger, 2001; Gehring & Knight, 2000; Kaufman, Ross, Stein, & Garavan, 2003). The magnitude of neural activity in the pMFC has also been related to adaptive posterror changes in response behavior, such as improvement in response speed following an error, which suggests increases in cognitive control (Kerns et al., 2004), or generalized slowing of responding, which, it is argued, reflects more cautious posterror behavior (Debener et al., 2005; Garavan, Ross, Murphy, Roche, & Stein, 2002; Gehring et al., 1993; Hajcak, McDonald, & Simons, 2003). These studies have also suggested that adaptive posterror changes in behavior appear to stem from a relationship between error-related activity in the pMFC and posterror activity in the dorsolateral prefrontal cortices (dlPFC) (Garavan et al., 2002; Kerns et al., 2004). For example, Kerns and colleagues found that higher levels of error-related pMFC activity during a Stroop task were associated with greater right dlPFC activity on the next trial, which in turn was associated with slowed response speeds during that trial (or with the magnitude of posterror slowing). Such research has focused primarily on immediate posterror changes in executive control behavior, in which the error and behavior change are in close temporal proximity (typically, in consecutive trials). The present study sought to answer two questions: First, does error-related neural activity influence adaptive changes in behavior beyond the trial immediately following an error? Second, is error-related neural activity or posterror neural activity predictive of performance (i.e., not repeating an erroneous response) several trials in the future?

A two-alternative forced choice task was employed while fMRI data was acquired. The task presented a series of random letters that for 90% of trials (*go trials*) required the same rapid buttonpress response (thus creating a prepotency for this response). When the same letter was presented on two consecutive trials (a *lure trial*), participants were required to provide the alternative response. The difficulty that participants typically experience in inhibiting a highly prepotent response was utilized in the present task to induce a large number of errors for the purpose of analysis. Two conditions of the task were administered. In the dynamic condition, participants were instructed that if they made an error in a lure trial, then the next lure trial would appear in the next 2–7 trials (see Figure 1). A successful response to a lure trial, on the other hand, ensured that the next lure trial would not appear for at least 10 trials (appearing instead between the 11th and 15th trials). In the standard condition, participants were instructed that their performance did not influence the trial sequence, and lure trials were pseudorandomly placed every 2–15 trials.

The aim of this design was to encourage adaptive posterror behavior during the dynamic condition—that is, to slow responses to go trials following a commission error. Past research indicates that commission errors are associated with faster than average response speed and that successful inhibitions are associated with slower than average response speed (Bellgrove, Hester, & Garavan, 2004). Although posterror slowing typically occurs without a specific manipulation's inducing it, it is not routinely

identified across studies, nor is it consistent across all participants within a study. We expected that by making a performance failure predictive of future trial presentation and thereby ensuring that another test of cognitive control of the type just failed would appear quickly (within the next seven trials), the salience of an error would be increased. Previous studies have found that increasing the salience of an error via financial punishment (Taylor et al., 2006; Yeung, Holroyd, & Cohen, 2005) or emphasizing accuracy over speed (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring et al., 1993) results in increased pMFC activity and greater posterror slowing. We therefore predicted that participants would show significantly more posterror slowing during the dynamic condition than during the standard condition. Commission errors were expected to elicit significant blood oxygen level dependent (BOLD) signals from the pMFC. Given the previous evidence of a relationship between the magnitude of posterror slowing and pMFC activity, we predicted that pMFC activity would be greater for errors in the dynamic condition than in the standard condition.

Presenting the next lure trial at least two and no more than seven trials after a commission error provided a variable posterror block of trials that could be used to examine posterror neural activity. In addition to inducing posterror slowing, the imminent presentation of the next lure was also expected to induce significant BOLD activity in the dlPFC region during the posterror period. Given previous findings of a relationship between posterror slowing and pMFC activity, we expected that the dynamic condition—in which a greater number of trials showing posterror slowing was predicted—would show a higher level of posterror period pMFC activity than would the standard condition. On the basis of previous findings of a relationship between speed of responding and lure trial performance, we also predicted an association between posterror slowing, dlPFC activity during the posterror block, and performance on the next lure trial. Although a number of studies have demonstrated a relationship between error-related pMFC activity and posterror performance, this relationship has so far been found only in the trial that immediately follows commission of an error. We examined this relationship during the dynamic condition, comparing neural activity associated with *adaptive errors*—errors followed by successful



**Figure 1.** The two-alternative forced choice response task. Participants were required to press the “1” button for each go trial in the sequence and the “2” button whenever the same letter was presented on consecutive trials (lure trials).

performance on the next lure trial—with that associated with *maladaptive errors*—errors followed by failed performance on the next lure trial.

## METHOD

### Participants

Fifteen healthy volunteers (8 females and 7 males; mean age = 23.4 years, range = 21–29) participated in the experiment. All of the participants provided written informed consent, which was approved by ethics committees at the University of Melbourne and St. Vincent's Hospital, Melbourne.

### Experimental Protocols

A two-alternative forced choice response task was administered in which random letters of the alphabet were presented serially at 1 Hz. The participants were instructed to press a button (the “1” button) at the appearance of each letter in the sequence, except when the same letter was presented consecutively (see Figure 1), in which case they were to press the “2” button. Stimulus duration was 700 msec and was followed by a 300-msec blank interstimulus interval screen. Stimuli were presented in two different conditions. In the standard condition, lure trials were pseudorandomly interspersed among 960 go trials. The number of go trials separating lure trials ranged between 1 and 15, with the gap between lure trials randomly selected from within this range (although this gap always consisted of an odd number of go trials, to ensure that lure trials always coincided with the beginning of an image acquisition).

The dynamic condition repeated the stimulus presentation and response parameters from the standard condition, except that the participants were instructed that their performance on lure trials would influence the task trial sequence. Incorrect responses to lure trials—that is, making the prepotent go response (pressing the “1” button again) rather than the alternate lure response (pressing the “2” button)—ensured that the next lure would be presented in 2 to 7 trials (at least 1 go trial separated all lure trials so that lures were never presented on consecutive trials). Conversely, correct responses to lure trials ensured that the next lure would not be presented for at least 10 trials (the 11th at the earliest, the 15th at the latest). The aim of this design was to encourage more cautious posterror responding, which would be reflected in slower response times (RTs) for go trials in the posterror blocks that separated lure trials. Slower response speeds in go trials that precede lure trials in inhibition tasks have been associated with improved performance (Bellgrove et al., 2004; Ridderinkhof, Nieuwenhuis, & Bashore, 2003), and this relationship suggests that the additional time provides more opportunity for the alternate response to overcome the prepotent go response in the response selection race (Logan, Cowan, & Davis, 1984). The larger gap between lure trials following correct lure responses was required to ensure sufficient separation of critical events for an event-related fMRI analysis.

Before entering the MRI scanner, the participants practiced two novel blocks of each condition to ensure that they understood the task instructions and how their performances would influence trial sequence in the dynamic condition. Four blocks of 260 trials each were administered for each condition during MRI data collection, with each block separated by a short break. All four blocks of a condition were administered consecutively, with the order of conditions counterbalanced across participants. The participants were informed which condition was being administered prior to each block of trials. During the scanner session, on average, 88 lure trials were administered in the standard condition (ranging from 85 to 90), and 89 were administered in the dynamic condition (ranging from 77 to 109). The average delay between error events was also not significantly different between the conditions (the standard condition averaged a 10.1-trial delay; the dynamic condition, a 10.7-trial delay).

All aspects of stimulus delivery and response recording were controlled by E-Prime software Version 1.1 (Psychology Software Tools, Pittsburgh, PA), running on a laptop PC (Celeron 2-GHz, 128-MB

Nvidia Video Card) that was interfaced with the MR scanner during acquisition of fMRI data. Stimuli were back projected onto a screen at the foot of the scanner bed, with a headcoil-mounted mirror enabling participants to view stimuli. The participants responded to each stimulus using the right hand, entering their responses on a two-button MR-compatible response box (Fibre-Optic Response Pads, Current Designs, Philadelphia, PA).

### Image Acquisition

Functional MR images were acquired at St. Vincent's Hospital, Melbourne, using a whole-body 1.5-tesla Siemens Vision scanner with a gradient-echo echoplanar imaging (EPI) sequence. The scanner was equipped with a standard radio frequency birdcage headcoil for signal transmission and reception. Lateral head stabilizers were used to minimize head movement. EPI images were acquired using a gradient-echo pulse sequence and sequential slice acquisition ( $T_R = 2,000$  msec,  $T_E = 48$  msec, flip angle =  $78^\circ$ ; 18 contiguous slices of 6-mm thickness, 10% gap, in-plane resolution of  $1.8 \times 1.8$  pixels in a FOV of 240 mm). Each functional run began with two volume acquisitions that were later discarded, to allow for steady-state tissue magnetization. A total of 188 EPI volumes were collected for each functional run, and a total of eight functional runs were performed for each participant. Activation data were registered to high-resolution T1-weighted isotropic (1-mm<sup>3</sup>) structural MPRAGE images to localize the pattern of physiological changes associated with the task.

### Data Analysis

All analyses were conducted using AFNI software Version 2.56 (afni.nimh.nih.gov/afni/). Following image reconstruction, the time series data were time shifted (using Fourier interpolation) to remove differences in slice acquisition times and then motion corrected using 3-D volume registration (least-squares alignment of three translational and three rotational parameters). Activation outside the brain was also removed using edge detection techniques.

Separate hemodynamic response functions at 2-sec temporal resolution were calculated using deconvolution techniques for successful lure responses (*stops*) and errors of commission (*errors*) in both the standard and the dynamic conditions. Dynamic condition errors were further classified according to whether they had been preceded by correct or erroneous lure performance. Only those preceded by correct performance were considered to be dynamic condition error events, because of the potential differences created by the posterror manipulation in the dynamic condition.

Block activation functions for the posterror block of go trials were identified as the image acquisitions between lure trials; these activation functions always coincided with the beginning of the 2-sec TR cycle. Because all trials were presented at 1 Hz, the posterror block spanned from the second go trial after a lure error to the final go trial preceding the next lure trial. For the purposes of analysis, posterror blocks of only seven trials were used in both the dynamic and the standard conditions, despite larger posterror blocks in the standard condition. To provide an equivalent period for comparison, the length and location of posterror periods in the dynamic condition were transposed to the posterror periods in the standard condition. Due to the temporal spacing of lures, the creation of blocks following standard errors did not overlap with other events of interest.

A mixed regression analysis was used whereby posterror blocks were calculated as a percentage of change relative to baseline. The baseline in this design was an implicit one and was indicative of ongoing and remaining task-related processing after the variance related to the other types of events had been removed. Separate impulse response functions (IRFs) were calculated for the four event-related conditions: standard stops and errors and dynamic stops and errors. A nonlinear regression program determined the best-fitting gamma variate function for these IRFs (Cohen, 1997) as previously described (Garavan, Ross, & Stein, 1999). The area under the curve of the gamma variate function was expressed as a percentage of the area under the baseline. The percentage area (event-related activation) and percentage change map (block activation) voxels were resampled at

1-mm<sup>3</sup> resolution, then warped into standard Montreal Neurological Institute (MNI) space, and then spatially blurred with a 3-mm isotropic RMS Gaussian kernel.

To ensure that activation produced during the posterror period did not overlap with activation produced by the events, an additional analysis that accommodated the hemodynamic lag of the posterror period was performed. Blocks corresponding to the posterror periods were convolved with a standard hemodynamic response and all analyses were repeated. This analysis produced results identical to those of the initial analysis, so only the results from the first analysis will be reported.

Group activation maps for each event type were determined with one-sample *t* tests against the null hypothesis of zero event-related activation changes (i.e., no change relative to tonic task-related activity). Significant voxels passed a voxelwise statistical threshold ( $t = 4.14, p \leq .001$ ) and were required to be part of a larger 142- $\mu$ l cluster of contiguous significant voxels. Thresholding was determined through Monte Carlo simulations and resulted in a 5% probability of a cluster's surviving due to chance.

The activation maps were then combined within categories but across conditions, deriving three Or maps: standard and dynamic stops, errors, and posterror periods after errors. For example, the errors Or map includes the voxels of activation indicated as significant according to either the standard errors map or the dynamic errors map. The mean activation for clusters in the combined maps was calculated for each condition for the purposes of an ROI analysis, and these data were used for a series of pairwise comparisons between conditions (standard vs. dynamic), corrected using a modified Bonferroni procedure for multiple comparisons (Keppel, 1991).

**Secondary analyses.** Due to the interest in the relationship between error-related pMFC activity and future performance, an additional analysis was performed. Errors in the dynamic condition were divided into two categories: adaptive or maladaptive, depending on whether performance on the next lure trial after an error was successful (adaptive), or incorrect (maladaptive). The posterror blocks were reclassified in the same way, to enable examination of the posterror neural activity that preceded success or failure on the next lure trial. The analysis focused on the dynamic condition because of the manipulation of posterror slowing behavior, which directly linked posterror slowing with success on the next lure trial. The analyses followed the same steps as outlined above, with the addition of a new multiple regression analysis to estimate percentage of event-related activation change on the basis of the newly categorized adaptive and maladaptive errors (determined by the participant's performance) for the events of interest. All analyses used the same measure of baseline activity (tonic go trial activity). Because the dynamic condition, which has the capacity to confound the spatial extent of activity in whole-brain analyses (Murphy & Garavan, 2005), had a lower number of events, we chose to perform an ROI analysis that compared the relevant events' activity using the clusters of activity from the original event-related analysis. The mean activation for the ROIs was then calculated using the voxel-level percentage change estimates for each event type, deriving mean activation scores for adaptive and maladaptive errors and for posterror blocks. The estimates were compared using repeated measures *t* tests, corrected via the modified Bonferroni procedure for multiple comparisons (Keppel, 1991).

## RESULTS

### Behavioral Results

Participants responded correctly to a higher proportion of lures in the dynamic condition than in the standard condition (see Table 1): 69% correct responses versus 54%. Similarly, participants avoided making two consecutive errors more often during the dynamic condition than during the standard condition: 77% avoidance versus 56%. Go trial RTs did not differ between the conditions, nor did the RTs for error responses; however, correct lure responses in the standard

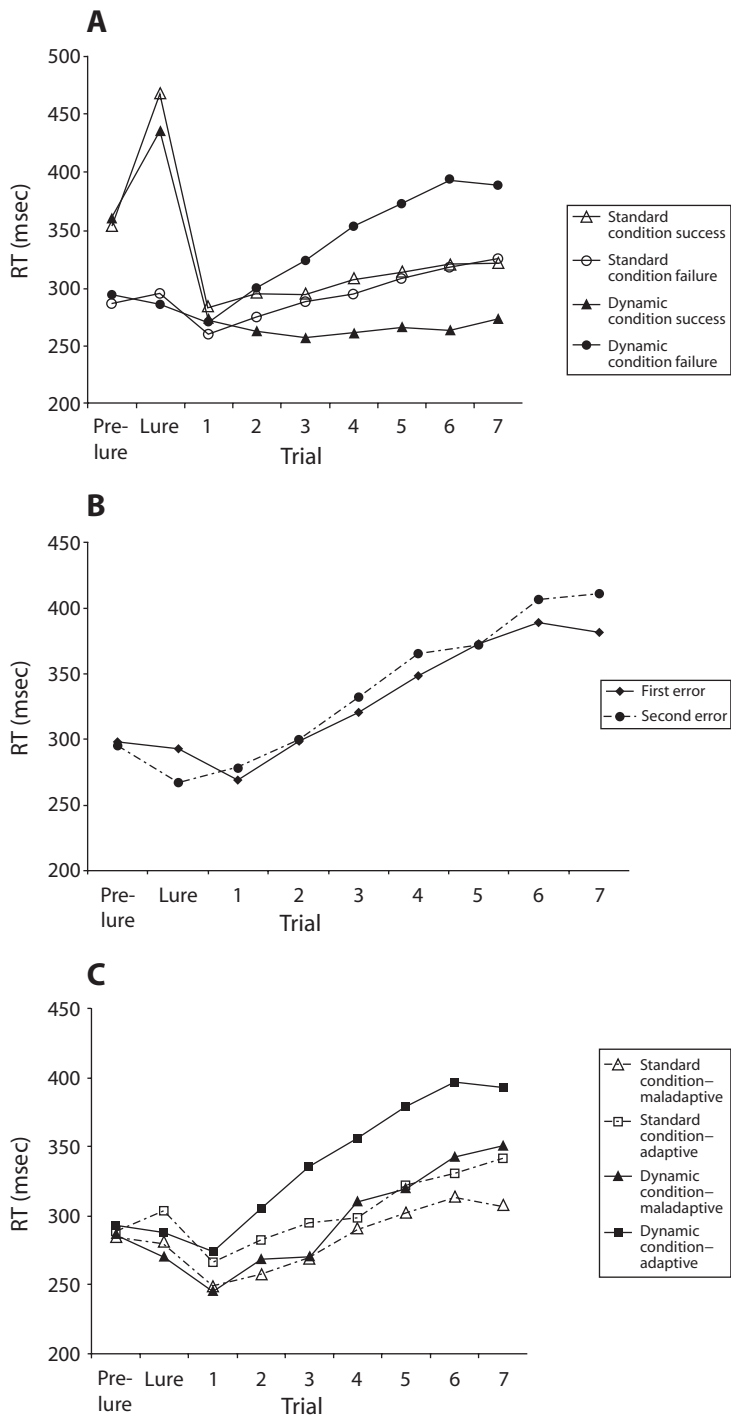
**Table 1**  
**Mean Response Times (RTs, in Milliseconds) With Standard Deviations and Percentages of Accuracy and Adaptation for Performance on the Two-Alternative Forced Choice Tasks**

Measure	Condition			
	Standard		Dynamic	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Go trial RTs	310	61	289	58
Correct lure RTs*	467	55	435	52
Error RTs	294	58	285	41
Lure accuracy*	54%	19%	69%	14%
Error adaptation* (avoiding consecutive lure errors)	56%	21%	77%	16%

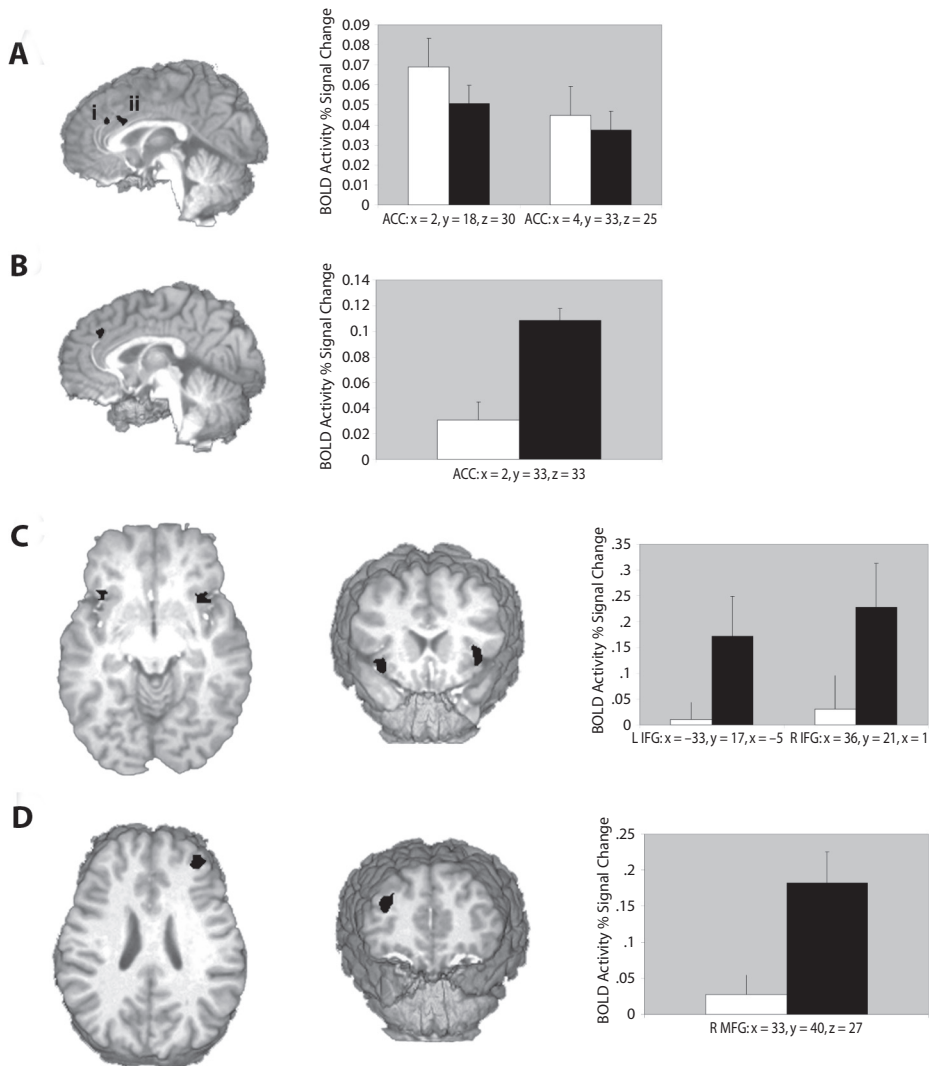
Note—Asterisks represent a significant difference between the standard and the dynamic conditions.

condition were approximately 32 msec slower than those in the dynamic condition. Accuracy performance for go trials during the standard and dynamic conditions was 99% and 98%, respectively. RTs for lure errors in the standard condition were significantly faster than the equivalent go trial RTs, whereas error and go trial RTs were not significantly different in the dynamic condition. The latter effect may have been influenced by the relatively fast go trial RTs in the dynamic condition. The large interlure gap following a successful inhibition in this condition appeared to increase the average speed of go trial responses, presumably because participants were aware that the next 10 trials would be go stimuli and that they therefore did not need to process the stimulus before making a response. For example, the mean go RT for these 10 trials was 268 msec for the dynamic condition and 312 msec for the standard condition.

Posterror slowing was assessed by creating difference scores, which were calculated by subtracting the go trial RT for each of the seven trials following a lure error from the RT for the go trial prior to the lure (see Figure 2). This difference score approach was taken to ensure that the effect of an error on phasic response behavior was captured, thereby minimizing the effect of other task manipulations on go trial RTs and consequently on posterror slowing (e.g., the effect mentioned in the paragraph above). A 2 (condition: standard vs. dynamic)  $\times$  7 (number of trials) repeated measures ANOVA was used to compare posterror slowing across conditions; it indicated that the dynamic condition showed significantly more posterror slowing than did the standard condition: 49 msec (average of the seven posterror trials) versus 9 msec [ $F(1,14) = 31.4, p < .01$ ]. The interaction between condition and trial was also significant [ $F(6,84) = 9.8, p < .01$ ], with post hoc tests (corrected for multiple comparisons) indicating significantly more posterror slowing for the dynamic condition during Trials 1–7. A within-condition, repeated measures ANOVA revealed that in the standard condition, Posterror Trials 5, 6, and 7 were executed significantly more slowly than they were in the prelure baseline condition ( $p < .05$ , corrected), whereas Posterror Trials 3–7 in the dynamic condition were executed significantly more slowly than they were in the prelure condition. Unexpectedly, in both conditions, the go trial RT for the trial immediately following an error was faster than the RTs for both the trial



**Figure 2.** Mean response times (RTs) for the go trial preceding an error (the prelure trial), for lure errors, and for the seven posterror go trials in the following conditions. Panel A displays successful and fail performances in the standard and dynamic conditions. Panel B displays performances in the dynamic condition, classified according to whether an error response was preceded by correct performance on the previous lure (first) or an error (second). Panel C displays levels of posterror slowing in both conditions classified according to performance on the following lure trial, with adaptive errors defined as accurate performance on the next lure trial, and maladaptive errors as failed performance (i.e., another error) on the next lure trial.



**Figure 3. Event-related brain activity.** In the bar graphs, white bars represent activity in the standard condition and black bars, activity in the dynamic condition. (A) Sagittal view ( $x$  slice = 4) of significant clusters of event-related anterior cingulate cortex (ACC) activity in the *i* and the *ii* clusters (Montreal Neurological Institute [MNI] coordinates:  $x = 2, y = 18, z = 30$  and  $x = 4, y = 33, z = 25$ ). Activity in these clusters was the same both for errors committed in the standard condition and for those committed in the dynamic condition. (B) Sagittal view ( $x = 2$ ) of the ACC activity cluster during the posterror period (MNI coordinates:  $x = 2, y = 33, z = 33$ ). This cluster showed significantly more activity in the dynamic than in the standard condition. (C) Axial and coronal views of the bilateral inferior frontal/insula gyrus (IFG) ( $z = -5, y = 21$ ) clusters of activity from the posterror period. (D) Axial and coronal views of the right middle frontal gyrus (MFG) ( $z = 27, y = 40$ ). All of these clusters showed significantly more activity during the dynamic condition than during the standard condition, and activity in the right middle and inferior frontal clusters during the dynamic condition was also predictive of future performance improvements.

that preceded the error and the trial on which the error itself was made (see Figure 2A). We repeated this analysis for the dynamic condition to check the potential for lure position (i.e., whether a lure had been preceded by an error or a correct lure response) to influence posterror behavior. Figure 2B demonstrates that lure position did not influence posterror behavior.

Given the interest in the influence of posterror behavior changes on future performance, the analysis described above was repeated after each posterror period was categorized

according to performance on the subsequent lure trial (see Figure 2C). The analysis revealed significantly more posterror slowing when performance on the next lure trial was successful (adaptive posterror slowing) than when it failed (maladaptive): 53 versus 7 msec [ $F(1,12) = 13.2, p < .01$ ].

### Imaging Results

**Errors.** Errors were associated with two clusters of significant activity in the posterior medial frontal cortex (see Figure 3A). The centers of mass for both clusters of

**Table 2**  
**Regions of Event-Related Neural Activity Differentiating Errors Made in the Standard and Dynamic Conditions**

Brain Region	Volume (μl)	MNI Coordinates		
		x	y	z
R middle frontal gyrus	383	30	45	32
R inferior frontal gyrus	190	44	9	14
R caudate	158	18	23	17

activity were located in the right anterior cingulate cortex (MNI coordinates:  $x = 4, y = 33, z = 25$ , and  $x = 2, y = 18, z = 30$ ), which falls within the rostral cingulate zone highlighted by Ridderinkhof et al.'s (2004) review of performance monitoring. Within these functionally defined ROIs, no significant difference was seen between BOLD activity for the standard condition errors and that for the dynamic condition errors. Unexpectedly, the trend of activity suggested greater activity for the standard condition, where there was significantly less posterror slowing. Activity in three other regions differentiated errors in the standard and dynamic conditions (see Table 2). Right middle frontal, inferior frontal, and caudate regions all showed significantly greater activity for standard condition errors, whereas no region of activity showed significantly higher levels during the dynamic condition. The right middle frontal cluster ( $x = 30, y = 45, z = 32$ , Brodmann area 9) is located proximal to the region identified by Kerns et al. (2004) ( $x = 30, y = 34, z = 37$ ) as associated with behavioral adjustments in response speed following an error.

**Posterror neural activity.** Activity during the standard and dynamic conditions was also compared for the variable block of activity following errors. Two patterns emerged. The first consisted of significantly greater activity in the right ACC (MNI coordinates:  $x = 2, y = 33, z = 33$ ), right middle frontal, and bilateral inferior frontal/insula (the center of mass is located in the inferior frontal gyrus, specifically in the pars orbitalis, but the extent of the activity does include the insula), in comparison with the pattern of activity in the standard condition (see Table 3 and Figure 3). The second pattern showed deactivation of the left midorbital and posterior cingulate during both the standard and the dynamic conditions, with the level of deactivation significantly greater in the dynamic condition.

**Adaptive versus maladaptive errors.** A secondary analysis of the dynamic condition was performed to compare adaptive and maladaptive errors. Adaptive errors were succeeded by correct performance on the following lure trial, whereas maladaptive errors were succeeded by another lure error. The ROIs from the initial error-related and posterror block period analyses were used to maximize the power of the analysis (Hester, Fassbender, & Garavan, 2004; Murphy & Garavan, 2005), with both event-related activity during errors and posterror block activity compared across the two categories (adaptive vs. maladaptive). A comparison of error-related activity did not reveal any significant differences between adaptive and maladaptive errors. However, posterror activity did appear

to be associated with subsequent lure performance. Three regions—the right middle frontal, right inferior frontal/insula, and right anterior cingulate cortex—showed significantly greater activity during adaptive posterror slowing than during maladaptive posterror slowing.

**DISCUSSION**

Error-related neural activity in the pmFC has been associated with immediate adaptive changes in response behavior. The results of the present study suggest that deliberately slowing responses after an error, in an effort to avoid making the same mistake on an upcoming trial, was associated with the magnitude of posterror-related pmFC activity but not with activity during commission of the error itself. Posterror slowing was observed in both experimental conditions, although to a significantly greater extent in the dynamic condition. The latter condition trained participants to expect another critical (lure) trial to quickly follow an error, providing an incentive for more cautious response behavior and hence greater posterror slowing. This behavior change clearly benefited performance, with increases in the magnitude of posterror slowing associated with increased accuracy on the subsequent lure trial. Despite these behavioral findings, error-related pmFC activity was not statistically different for the standard and the dynamic conditions. Only activity in the occipital cortex and thalamus (medial dorsal nucleus) was greater for the dynamic condition.

More surprisingly, activity in the right prefrontal cortex ( $x = 30, y = 45, z = 32$ ; Brodmann area 9), previously associated with posterror conflict adaptation effects (Kerns et al., 2004), was greater for standard condition errors that preceded smaller amounts of posterror slowing. Kerns et al. found that activity in the right middle frontal gyrus ( $x = 30, y = 34, z = 37$ ; Brodmann area 9) during the trial following an error was associated with increased posterror conflict adaptation and greater ACC activity during the error trial that had preceded it. We acknowledge that our results may have been influenced by unexpected differences in baseline performances during the two conditions. For example, RTs for errors (relative to RTs for go trials)

**Table 3**  
**Regions of Neural Activity Differentiating Posterror Periods in the Standard and Dynamic Conditions**

Brain Region	Volume (μl)	MNI Coordinates		
		x	y	z
Dynamic > Standard				
<b>R middle frontal gyrus</b>	<b>987</b>	<b>33</b>	<b>40</b>	<b>27</b>
L inferior frontal/insula	357	-33	17	-5
<b>R inferior frontal/insula</b>	<b>238</b>	<b>36</b>	<b>21</b>	<b>1</b>
<b>R anterior cingulate cortex</b>	<b>196</b>	<b>2</b>	<b>33</b>	<b>33</b>
Standard > Dynamic				
L mid orbital gyrus	850	-7	42	-2
L posterior cingulate cortex	299	-6	-51	32

Note—Regions showing significantly greater activity during adaptive posterror periods, in comparison with maladaptive posterror periods, are highlighted in bold text.

were significantly faster in the standard condition than in the dynamic condition. This effect has previously been associated with both higher levels of inattentiveness and compensatory increases in neural activity for inhibition-related cortical regions (Bellgrove et al., 2004). Future studies might resolve the origin of this effect by parametrically manipulating the incentive for posterror slowing within a single task condition—by, for example, providing feedback during errors that indicates approximately when the next lure will appear (e.g., within the next 5, 10, or 15 trials).

Although error-related activity did not show the expected relationship to adaptive posterror behavior, amounts of neural activity in the posterror period (the block of go trials that preceded the next lure trial) did correlate with the magnitude of posterror slowing and accuracy. The dynamic condition, which had greater amounts of posterror slowing and fewer consecutive lure errors than did the standard condition, elicited significantly greater activity than did the standard condition in the right pMFC, right middle frontal, bilateral inferior frontal, and left cerebellum. Furthermore, posterror activity in three of these regions (right pMFC, right middle frontal, and right inferior frontal) was predictive of future success. When posterror periods in the dynamic condition were divided by posterror lure performance, those periods that preceded successful adaptation of performance (i.e., a correct response on the next lure trial), as compared with periods that preceded another lure error, had significantly greater activity in the right prefrontal and pMFC regions. RTs for the posterror periods in this comparison revealed significantly greater posterror slowing during the adaptive posterror periods than during the maladaptive periods.

The association between posterror period activity in the right PFC and improved performance on the next lure trial may be consistent with two other convergent findings. Whereas a reciprocal relationship between pMFC activity and left task set prefrontal regions has been highlighted by previous studies (MacDonald, Cohen, Stenger, & Carter, 2000), inhibitory control over motor responses (which is required for lure trials) has consistently been associated with activity in right prefrontal regions, particularly the right inferior frontal cortex (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003; Aron, Robbins, & Poldrack, 2004; Chambers et al., 2006; De Zubicaray, Andrew, Zelaya, Williams, & Dumanoir, 2000; Garavan et al., 1999). We have also previously shown that when participants are cued to expect an upcoming inhibition lure, activity during the preparatory period (between the cue and lure) increases in those regions that are active during the inhibition trial itself (Hester, Murphy, et al., 2004). Posterror activity in the right PFC may therefore have represented preparation for the upcoming lure trial. Other studies have shown similar relationships between cognitive functions and preparatory activity (Brass & von Cramon, 2004; Lawrence, Ross, Hoffmann, Garavan, & Stein, 2003; Pochon et al., 2001; Sakai & Passingham, 2003), with one study (Polli et al., 2005) finding that deactivation of default mode network regions such as the orbitofrontal cortex, the posterior cingulate, the superior temporal cortex, and the angular

gyrus was associated with accurate performance. It is argued that the purpose of such task-induced deactivation in the default mode network is to optimize task performance through the allocation of resources to task-related regions and away from task-irrelevant ones (Drevets & Raichle, 1998). Although we found greater levels of posterror period deactivation for default mode network regions (the midorbital gyri and the posterior cingulate) during the dynamic condition—the condition with greater levels of success and posterror improvement of performance—activity levels in these regions did not predict future success during the dynamic condition.

Our results appear to support the critical relationship that the pMFC and prefrontal regions have to posterror changes. In addition to demonstrating greater activity in these regions during the periods of peak behavioral change, the results suggest that such activity was adaptive, insofar as it predicted changes in behavior that led to future success. The results do, however, present an inconsistency with the extant literature. Previous studies have indicated that increases in error-related activity, coinciding with the incorrect behavioral response, were related to posterror slowing (Debener et al., 2005; Garavan et al., 2002) or to speeding of responses (conflict adaptation effects) (Kerns, 2006; Kerns et al., 2004) in the trial immediately following the error. Similarly, pMFC activity response locked to the error has been related to increased prefrontal activity during the subsequent posterror trial (Kerns et al., 2004). Our results identify a similar neuroanatomical relationship, but place it in the period after an error has occurred rather than during the same period as the error. How might the inconsistency with past results be reconciled?

Although the timing of activity in the present study appears to be inconsistent with previous results, it is worthwhile to consider the relationship between task requirements, brain activity, and behavior. Previous studies have shown that heightened pMFC activity coincides with an error that immediately precedes behavior change in the form of response speed changes on the very next trial. Typically, these studies have administered tasks whose every trial is critical, in that a random distribution of trials ensures that the trial following an error could require immediate changes in behavior. In contrast, the present task never presented two lure trials in succession (in order to enable fMRI analysis), meaning that the participants were aware that the trial immediately following an error would never be a critical trial. The behavioral data indicate that the RT for a trial immediately following an error was faster than the RT for the error trial or for the trial preceding the error. Posterror slowing did not occur until the second trial after an error, after which the magnitude of slowing appeared to increase linearly. The fact that pMFC and dlPFC activity appeared to be delayed until the block period after an error had been made, rather than occurring during the error event, appears to coincide with the delay in implementing adaptive posterror behavior.

Interpretation of the inconsistency between the past studies and the present one is made more complex by the latter's use of block-related analysis, which averages neural activity over a series of posterror trials. Typically, pre-



vious research examined only event-related neural activity, whereas the present study included both event-related and block-related activity. For example, the increase in dynamic posterror block activity may have been due to a phasic increase within the block of trials or a sustained increase across them, potentially the results of different behavioral mechanisms at work. The temporal insensitivity of block-related analysis limits our ability to further examine these questions with the available data, but it does pose an interesting question for further studies.

Another consideration is the influence that trial sequence information had on the relationship between error and posterror behavior. In many of the paradigms typically described in the literature, errors implicitly inform participants that their response styles have become inconsistent with the task requirements and that they need to increase their level of control to successfully complete future trials of similar difficulty. In the present task, this relationship was made explicit to participants, in that an error would always induce the presentation of another high-control trial among the ensuing seven trials. It is unclear how the explicitness of this relationship, which potentially increases the task relevancy of errors not seen in other tasks examining these processes, may have influenced the brain–behavior relationships. The inconsistency between the present study's findings and those of previous studies may, therefore, stem from the differences in the paradigms used—in particular, the delay between critical trials in the present study.

The absence of greater error-related activity during the dynamic condition, despite greater posterror slowing, may not necessarily be inconsistent with the conflict theory, insofar as this theory posits activity to be related to the level of conflict between competing responses during a trial. Consequently, the theory predicts greater pMFC activity during error trials due to the high level of conflict between the prepotent go response and the infrequent no-go response. The dynamic condition does not appear on face value to present greater levels of conflict during error trials, and the lower, nonsignificant levels of conflict we observed during this condition might be consistent with previous findings of reduced pMFC activity when participants were in a high-control state (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Carter et al., 2000; Kerns et al., 2004). Similarly, the theory also predicts that under conditions such as the dynamic condition's posterror periods—in which the potential for another lure trial increases the competition between the two competing responses—response uncertainty is increased, and therefore, conflict is increased as well. It is also worth considering whether the higher posterror activity during the dynamic condition was related to error likelihood/risk (Brown & Braver, 2005) or to task set activity (Dosenbach et al., 2006). As mentioned above, committing an error appears to have acted as a cue, prompting the participant to be cautious of an ensuing lure trial that, implicitly or explicitly, resulted in slower, more cautious responses. The idea that participants enacted an alternate task set following an error also appears parsimonious with the linear increase in posterror slowing observed during the

dynamic condition. RTs increased for each go trial following an error, as though the nonappearance of the expected posterror lure trial increased expectancy, and therefore cautiousness, with each ensuing go trial.

Our results, when combined with others derived from examinations of the relationship between cortical activity and posterror modulations of cognitive control, appear to speak to the flexibility of cognitive control, particularly as implemented by the pMFC. Although the present study suggests that increased cognitive control was implemented in association with increased pMFC activity across multiple trials, it would appear that the timing of pMFC interventions may have been a consequence of task demands. Studies to date have shown that increased cognitive control is associated with activity increases in the pMFC several trials before the critical trial (Hester, Murphy, et al., 2004; Ridderinkhof et al., 2003), within a trial (Magno, Foxe, Molholm, Robertson, & Garavan, 2006), or across consecutive trials (Hajcak et al., 2003; Kerns et al., 2004; MacDonald et al., 2000; Polli et al., 2005). Although our findings further support evidence of the relationship between pMFC/PFC activity and subsequent changes in cognitive control, they leave open the question of what properties of an error initiate this dynamic relationship.

#### AUTHOR NOTE

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