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Anterior cingulate cortex and conflict detection: An update of theory and data

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The dorsal anterior cingulate cortex (ACC) and associated regions of the medial frontal wall have often been hypothesized to play an important role in cognitive control. We have proposed that the ACC's specific role in cognitive control is to detect conflict between simultaneously active, competing representations and to engage the dorsolateral prefrontal cortex (DLPFC) to resolve such conflict. Here we review some of the evidence supporting this theory, from event-related potential (ERP) and fMRI studies. We focus on data obtained from interference tasks, such as the Stroop task, and review the evidence that trial-to-trial changes in control engagement can be understood as driven by conflict detection; the data suggest that levels of activation of the ACC and the DLPFC in such tasks do indeed reflect conflict and control, respectively. We also discuss some discrepant results in the literature that highlight the need for future research.

Cognitive control usually refers to the ability to guide information processing and behavior in the service of a goal; such control is a central aspect of many higher-level cognitive functions, including attention, working memory, and planning (E. K. Miller & Cohen, 2001). It has been noted that any theory of cognitive control would have to specify how the cognitive system is able to determine how much control is needed (Botvinick, Braver, Barch, Carter, & Cohen, 2001). Accordingly, we and others have suggested that one monitoring function capable of regulating the extent to which control is engaged is the detection of conflict occurring between competing, concurrently active, mutually incompatible representations (Botvinick et al., 2001; Botvinick, Cohen, & Carter, 2004; Gruber & Goschke, 2004; van Veen & Carter, 2002a, 2006). Following the detection of a conflict, control processes are thought to be engaged to resolve the conflict and to prevent future performance decrements. In particular, the monitoring system appears to have a distinct neural substrate—namely, the anterior cingulate cortex (ACC). Since top-down control appears to be mostly implemented in the dorsolateral prefrontal cortex (DLPFC; E. K. Miller & Cohen, 2001), it is suggested that following the detection of conflict by the ACC, these lateral areas are engaged in order to reduce conflict.

For illustration, consider the parallel distributed processing model of the Stroop task displayed in Figure 1 (adapted from Botvinick et al., 2001; E. K. Miller & Cohen, 2001). This model features two input layers (one for colors, one for words), a response layer, a control layer, and a conflict monitor. In the Stroop task, participants are presented with color names printed in a particular color, and they must name the color while ignoring the word. When a stimulus is incongruent (i.e., color and word are not identical), the color and word dimensions activate the associated responses, resulting in conflict between the activated responses and an increased likelihood of errors. This conflict is proposed to activate a conflict monitor localized in the ACC, which in turn engages the control functions of the DLPFC (in the model, specifically it would engage the color naming unit in Figure 1). This increased engagement of the DLPFC increases attention to the color on subsequent trials, resulting in improved performance.

In this review, we will first discuss some of the evidence linking activation of the ACC to conflict as it occurs in speeded response interference tasks, during both correct trials and errors. In the next section, we will discuss evidence linking the trial-by-trial modulation of control, as implemented in the DLPFC, to the conflict-monitoring functions of the ACC, suggesting that these mechanisms are linked to form a conflict–control loop, as illustrated in the model depicted in Figure 1. In the subsequent sections, we will discuss some areas of research that have yielded results that are not necessarily consistent with this theory, for which further theoretical and empirical work is needed if these results are to be reconciled with the theory. Finally, we will briefly discuss alternative theories that

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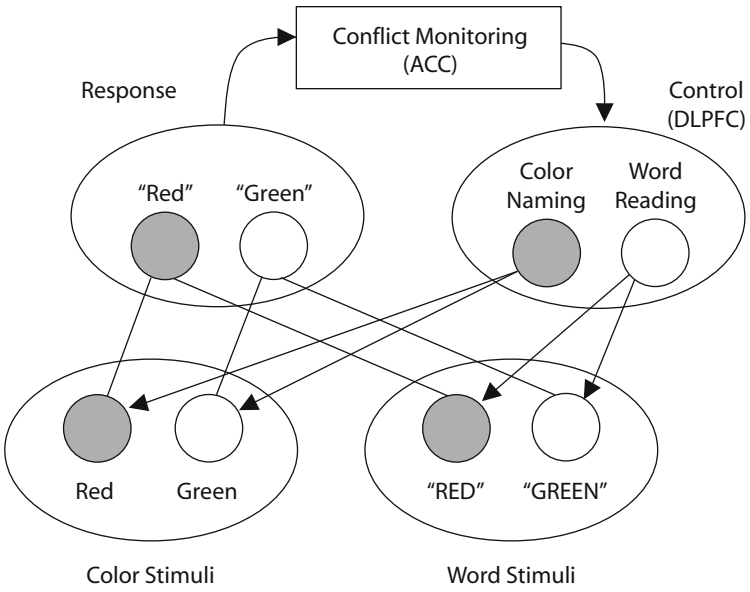

Figure 1. Example of a computational model of an interference task. When relevant (color) and irrelevant (word) stimulus dimensions are incongruent and activate conflicting responses, the anterior cingulate cortex (ACC) detects this conflict and engages attentional control mechanisms in the dorsolateral prefrontal cortex (DLPFC).

compete with our view that the ACC functions as a conflict monitor.

CONFLICT DURING CORRECT AND ERROR TRIALS

Conflict During Correct Trials

The conflict–control loop (CCL) theory of ACC functioning was originally based on data obtained from speeded response tasks. Not only are the notions of conflict and control relatively well understood in this type of task, but these tasks have also provided most of the empirical dissociations of conflict and control, as well as empirical support for linking these component processes to a known neural circuitry understood to contribute to cognitive control. In this section, the concept of conflict in speeded response tasks will be elaborated, and we will discuss evidence for the relationship between cognitive conflict and activation of the ACC and other frontal cortical regions.

Conflict in cognitive tasks can be caused or modulated by several experimental parameters. The most often studied experimental manipulation of conflict in speeded response tasks is the coactivation of different, incompatible response channels induced by conflicting information present in a stimulus. For example, this occurs in the case of the Stroop task (MacLeod, 1991, 1992; MacLeod & MacDonald, 2000; Stroop, 1935), the Eriksen flanker task (B. A. Eriksen & Eriksen, 1974; C. W. Eriksen & Schultz, 1979), and the Simon task (Ridderinkhof, 2002; Simon, 1969; Simon & Berbaum, 1990). In such tasks, the participant is to respond as quickly and accurately as possible to one (relevant) stimulus dimension while trying to ignore another (irrelevant) stimulus dimension; conflict is caused when the irrelevant stimulus dimension is mapped onto an incorrect response. Congruent stimulus–response (S–R) mappings exist when both stimulus dimensions are mapped onto the same response, leading to fast and accurate responses; when the irrelevant stimulus dimension is mapped onto a different motor response, responses are characterized by increased reaction times (RTs) and error rates. So-called "neutral" conditions involve an irrelevant stimulus that is not mapped onto any response in the response set. In this type of task, processing of the irrelevant stimulus dimension is thought to occur more quickly and automatically than processing of the relevant one, resulting in a fast activation of the response associated with the irrelevant stimulus. Conversely, processing of the relevant stimulus dimension in such tasks is usually slower and more controlled, leading to a relatively slow activation of the associated response (see, e.g., Cohen, Dunbar, & McClelland, 1990; Gratton, Coles, & Donchin, 1992; Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988).

The level of conflict depends on the extent to which competing responses are activated—either as a result of strategy or of automatic processes. The relative frequencies at which the stimuli and the associated responses occur are another way in which conflict can be manipulated. Participants have been found to respond more quickly to frequent S–R mappings than to infrequent ones; they are thought to optimize performance by priming the frequent response to each trial, at a cost of decreased performance in infrequent trials (Jones, Cho, Nystrom, Cohen, & Braver, 2003; J. Miller, 1998). For instance, in the "go/no-go" task (see, e.g., Karlin, Martz, & Mordkoff, 1970), participants have to respond to frequent stimuli (go trials) and withhold responses to certain infrequent stimuli (no-go trials). Be-

cause go trials are frequent, participants tend to prepare a response to each stimulus and activate that response during each trial, including no-go trials. Thus, during no-go trials, the tendency to withhold (the no-go response) has to compete with the primed go response, resulting in increased false alarms to no-go trials (Jones et al., 2003). Conversely, when participants have to withhold responding on a majority of trials, as occurs in target detection tasks, they tend to adopt a strategy in which the infrequent go response is activated relatively slowly and less automatically because of competition with the tendency to withhold responding, resulting in greater RTs and reduced accuracy to the infrequent target stimulus (Jones et al., 2003).

Evidence indicating that the "automatic" response activation in these tasks occurs relatively quickly, and that activation of the relatively more controlled S–R mapping occurs much more slowly, comes from many converging sources. These include studies of response force, electromyographical data, and lateralized readiness potentials, as well as distributional analyses of RT and accuracy (Coles, Gratton, Bashore, Eriksen, & Donchin, 1985; de Jong, Liang, & Lauber, 1994; de Jong, Wierda, Mulder, & Mulder, 1988; Dehaene et al., 1998; Eimer, 1995; Eimer, Hommel, & Prinz, 1995; Gehring, Gratton, Coles, & Donchin, 1992; Gratton et al., 1992; Gratton et al., 1988; Kopp, Mattler, Goertz, & Rist, 1996; Kopp, Rist, & Mattler, 1996; Kornblum, Hasbroucq, & Osman, 1990; J. Miller, 1998; J. Miller & Hackley, 1992; Praamstra & Oostenveld, 2003; Ridderinkhof, 2002; Ridderinkhof & van der Molen, 1995; Spencer & Coles, 1999). Furthermore, neuroimaging studies have almost invariably found dorsal ACC and prefrontal cortex activity when comparing high- and low-conflict trial types in these and related tasks (e.g., Braver, Barch, Gray, Molfese, & Snyder, 2001; Durston, Thomas, Worden, Yang, & Casey, 2002; Kerns et al., 2004; van Veen & Carter, 2005; van Veen, Cohen, Botvinick, Stenger, & Carter, 2001).

Conflict might, of course, not be limited to conflict between possible responses; in theory, it could occur anywhere within the information processing system. We and others initially found that the ACC appears to be engaged only during conflict between response representations (Milham et al., 2001; Nelson, Reuter-Lorenz, Sylvester, Jonides, & Smith, 2003; van Veen et al., 2001), suggesting that the ACC is activated selectively with such conflicts. However, subsequent studies have suggested that in some situations, the ACC can also be engaged with conflicts between other types of representations, such as semantic or conceptual representations (Badre & Wagner, 2004; van Veen & Carter, 2005; Weissman, Giesbrecht, Song, Mangun, & Woldorff, 2003).

Conflict During Error Trials

Trials in which errors are made have also been associated with ACC activation. People are very efficient and fast at correcting their own slips of action; in speeded response tasks, the time between the erroneous and the corrective responses is typically less than 200 msec, making error correction one of the fastest cognitive processes known (Cooke & Diggles, 1984; Rodríguez-Fornells, Kurzbuch, & Münte, 2002). Event-related potential (ERP) research has shown that a slip of action is immediately followed by a large-amplitude, sharp, negative waveform that peaks 50–100 msec following buttonpress (e.g., Dehaene, Posner, & Tucker, 1994; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Luu & Tucker, 2001; Ridderinkhof et al., 2002; Rodríguez-Fornells et al., 2002; van Veen & Carter, 2002b) or 100–150 msec after electromyograph onset (e.g., Gehring & Fencsik, 2001; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Kopp, Rist, & Mattler, 1996; Scheffers & Coles, 2000; Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996). This is the error-related negativity (ERN), and dipole-modeling studies have consistently shown that this component might be generated by the ACC (Dehaene et al., 1994; Holroyd, Dien, & Coles, 1998; Miltner et al., 2003; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; van Veen & Carter, 2002b; Yeung, Botvinick, & Cohen, 2004). Converging evidence from fMRI studies has confirmed this result by showing that action slips are accompanied by ACC activation (Braver et al., 2001; Carter et al., 1998; Garavan, Ross, Kaufman, & Stein, 2003; Mathalon, Whitfield, & Ford, 2003; Rubia, Smith, Brammer, & Taylor, 2003; Ullsperger & von Cramon, 2001).

According to the first interpretations of the ERN, this component reflected a process that detected errors by comparing a representation of the actual response to a representation of the intended response; the ERN would be generated by a mismatch between these two representations (Falkenstein et al., 1991; Falkenstein et al., 2000; Gehring et al., 1993; Scheffers et al., 1996). One attractive and parsimonious feature of this theory is that it regards the ERN as comparable to other types of medial frontal negativities thought to be the results of a mismatch process, including the "mismatch negativity" and the N400 (Falkenstein et al., 1991).

The conflict theory of ACC functioning grew out of this "comparator" error detection theory of the ERN, and these two theories remain closely related. However, we view the conflict theory as more general than the comparator error detection theory, and therefore more parsimonious. Furthermore, in speeded forced choice response tasks, errors are typically fast, impulsive responses. Analyses of the conditional accuracy functions of interference tasks such as the Eriksen task have shown that the accuracy of extremely fast responses to incongruent trials is frequently below chance, suggesting that these responses are for the most part based on the irrelevant stimulus dimension (see, e.g., Gratton et al., 1992, Experiment 1). The error detection theory assumes that such fast erroneous responses are nevertheless intended to be correct; we find this assumption questionable, and know of no evidence supporting it. Nevertheless, the conflict and error detection theories are perhaps more similar than they are different; indeed, there is much overlap between the notions of a mismatch between two representations and of conflict between the representations.

Conflict theory treats errors as another type of conflict trial and assumes that errors are detected by the same pro-

cess responsible for detecting conflict. In speeded forced choice response tasks, there are substantial similarities between error trials, on the one hand, and correct trials involving conflict, on the other, as well as in the patterns of neural activity associated with these two trial types. Psychophysiological evidence has suggested that the timing of the activation of the correct and incorrect responses appears to be similar during errors and correct conflict trials (e.g., Kopp, Rist, & Mattler, 1996). As discussed earlier, correct conflict trials are often characterized by a fast, impulsive, small-amplitude activation of the response associated with the irrelevant stimulus dimension. Error trials, in contrast, are usually fast, impulsive responses that are immediately followed by the tendency to correct the error. Ongoing processing of the stimulus is thought to continue during and after the commission of the error, leading to activation of the correct response and to the frequent occurrence of a fast correction of the erroneous response (Kopp, Rist, & Mattler, 1996; Rabbitt & Rodgers, 1977; Rabbitt & Vyas, 1981). Hence, during both errors and correct trials, activation of the incorrect response appears to precede activation of the correct response; the difference between these two trial types appears to be that the fast activation of the incorrect response during correct conflict trials does not manage to reach response threshold, whereas it does during errors trials (van Veen & Carter, 2002b). Kopp, Rist, and Mattler, using an Eriksen task, showed that the ERN and the frontal N2 have the same scalp topography and are both related in the same way to the amplitude of the initial incorrect response activation (as measured by lateralized readiness potentials [LRPs] and squeeze force). We reasoned that the maximum response conflict and ACC activity should precede the response during correct conflict trials; conversely, response conflict and ACC activity should occur immediately following the erroneous response during error trials, since these slips are usually immediately corrected. Indeed, we found that the frontocentral N2 during correct conflict trials and the ERN could both be modeled by a dipole in the same area of the ACC (van Veen & Carter, 2002b). Thus, ACC activity appears to be reflected in the frontocentral N2 component during correct conflict trials, reflecting conflict prior to the moment of the response, and in the ERN during error trials, reflecting conflict immediately following the response. This finding has subsequently been replicated elsewhere (Nieuwenhuis et al., 2003; Yeung et al., 2004).[1] Consistent with these dipole models, Mathalon et al. (2003) showed that on a between-subjects basis, both N2 and ERN amplitude are correlated with ACC activity as measured with fMRI. Thus, ACC activation appears to occur prior to the response during correct conflict trials and immediately following the response during error trials, consistent with predictions made by the conflict theory.

An important detail to note is that, according to the conflict theory, the detection of postresponse conflict does not trigger the corrective response itself. Rather, this corrective response is considered to be triggered by ongoing processing of the stimulus (Rabbitt & Vyas, 1981; Yeung et al., 2004). Indeed, very fast corrective responses have been observed, as little as 20 msec after the initial erroneous response (Rabbitt, 1967, 2002). The much slower ERN latency would not be consistent with these findings if the ACC itself were proposed to trigger the corrective response.

The view that the ERN reflects the conflict between a fast erroneous response and a subsequent corrective response predicts that it is related to the amplitudes of both response signals. The results of several empirical and modeling studies confirm this prediction. Yeung et al. (2004) implemented in a series of computational models the hypothesized mechanism of an ERN that is related to conflict between a fast erroneous response and a subsequent corrective response. Much as we had previously predicted on the basis of psychophysiological data (van Veen & Carter, 2002b), Yeung et al. showed that a model of conflict during this task does indeed predict that conflict would reach maximum prior to the response during correct conflict trials but immediately following the response on erroneous trials. Yeung et al. furthermore showed that this simple model can account for several key findings concerning the relationship of ERN amplitude to various experimental manipulations, which we will now discuss in turn (see Yeung et al., 2004, for details).

Scheffers and Coles (2000) observed that the ERN is greater for errors on congruent than on incongruent trials. Yeung et al. (2004) found that their model readily accounted for these data; postresponse conflict between the erroneous response and the subsequent corrective response appears to be greater because the activity of the correct-response unit builds up faster in the case of congruent errors, since congruent trials present greater evidence for the correct than for the incorrect response. Furthermore, the ERN appears to be greater for corrected than for uncorrected trials (Scheffers & Coles, 2000). When separating the model's output responses on the basis of whether the corrective response reached the response threshold, Yeung et al. found that postresponse conflict was indeed greater for corrected than for uncorrected responses, showing that the theory can account for these data. Consistently, other studies have also found that the ERN is greater for corrected than for uncorrected errors (Gehring et al., 1993; Rodríguez-Fornells et al., 2002). Rodríguez-Fornells et al. compared ERNs in conditions in which self-correction of action slips was either encouraged or forbidden, and they showed that ERN amplitude was greatly enhanced when participants corrected their errors. Moreover, in the condition in which correction was encouraged, the amplitude of the ERN was positively related to correction speed. Both of these effects were also clearly present in the LRP; the correction condition elicited a very small posterror LRP deflection, and faster correction elicited a posterror LRP deflection with an earlier peak.

Second, the ERN has been observed to have an increasing amplitude the more participants emphasize accuracy over speed in their performance (Falkenstein et al., 2000; Gehring et al., 1993). Consistently, error-related ACC activation as measured by fMRI is greater under accuracy emphasis than under speed emphasis (Ullsperger & von Cramon, 2004; van Veen, 2006). Yeung et al. (2004)

implemented accuracy emphasis in their model by means of a stricter response criterion and increased attention to the relevant stimulus dimension. These manipulations caused the model to display greater corrective response force under accuracy emphasis, as well as greater conflict following erroneous responses, again consistent with the observed data.

A third important finding is the observation that the ERN is greater when flanker stimuli are frequent than when they are infrequent (Holroyd & Coles, 2002). Yeung et al. (2004) accounted for these data by biasing the frequent stimulus and response in the model, facilitating the buildup of evidence of the correct response unit when this corresponded to the frequent correct response. This manipulation resulted in greater conflict between the erroneous and corrective responses for frequent stimuli.

Finally, the models by Yeung et al. (2004) can account for the seemingly inconsistent results of Gehring et al. (1993), who found a greater ERN paired with smaller amplitude of the erroneous response, as measured by response force, and Scheffers et al. (1996), who found a greater ERN with increasing response force of the erroneous response. Yeung et al. noticed that the participants in the Scheffers et al. study were more likely to correct their errors than were the participants in the Gehring et al. study, and Yeung et al. showed that their models could account for the proportions of corrective responses and for the different relationships of the amplitude of erroneous responses to the amplitude of the ERN in both experiments.

Thus, these computational modeling studies show that conflict theory can account remarkably well for a variety of ERN data, including results that seem conflicting or paradoxical. In sum, the available research suggests that the ERN is a psychophysiological correlate of conflict detection by the ACC between two simultaneously active, incompatible response channels: a fast, impulsive erroneous response, and a subsequent corrective response (van Veen & Carter, 2002b). It is, however, important to note that although the Yeung et al. (2004) models can successfully simulate much of the available data on the ERN from within conflict theory, Holroyd, Yeung, Coles, and Cohen (2005) have also provided an alternative computational account that explains a great deal of the same data on the basis of an explicit comparator/mismatch mechanism.

CONTROL ENGAGEMENT FOLLOWING CONFLICT

Trial-to-trial adjustments of control (referred to by Ridderinkhof, 2002, as *micro-adjustments*, or the effect of a previous trial on performance on the present trial) have been shown in a number of studies to be related to the amount of conflict occurring on the previous trial. We and others have focused on two types of micro-adjustments that might reflect the recruitment of control mechanisms by conflict detection: the conflict adaptation effect and posterror slowing.

The *conflict adaptation effect* refers to the finding that the difference in performance between incongruent and congruent trials is dependent on the nature of the previous trial (see, e.g., Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Burle, Possamaï, Vidal, Bonnet, & Hasbroucq, 2002; Egner & Hirsch, 2005a, 2005b; Gratton et al., 1992; Kerns et al., 2004; Kunde, 2003; Ridderinkhof, 2002; Stürmer & Leuthold, 2003; Stürmer, Leuthold, Soetens, Schröter, & Sommer, 2002; Ullsperger, Bylsma, & Botvinick, 2005; Wühr & Ansorge, 2005). Specifically, following correct incongruent trials, performance is less influenced by the distracting irrelevant stimulus dimension than following correct congruent trials, when performance is relatively more influenced by the distracting stimulus dimension. When an incongruent trial is preceded by another incongruent trial (an iI trial), RTs are shorter and accuracy is higher than when the current trial is preceded by a congruent one (cI); likewise, when one congruent trial is preceded by another (cC), RTs are shorter than when the current trial is preceded by an incongruent one (iC). This phenomenon has been interpreted as the dynamic adjustment of control depending on conflict (Botvinick et al., 2001). Following a nonconflict trial, control is relaxed, and participants allow their responses to be more influenced by the distracting irrelevant stimulus dimension. Following a conflict trial, control is more highly engaged, resulting in less susceptibility to the irrelevant stimulus dimension. RTs to cC trials are very fast; the relevant and irrelevant stimulus dimensions both activate the same (correct) response, resulting in a faster buildup of activity leading to a faster response. RTs to iC trials are therefore relatively slow; the facilitating effect of the irrelevant stimulus dimension is reduced, and activation of the correct response is slower. Conversely, iI trials elicit relatively fast and accurate responses because the incongruent distracting stimulus is filtered out to a larger degree, resulting in a smaller activation of the incorrect response. On cI trials, the incongruent irrelevant stimulus dimension has a larger effect on the response, creating more response conflict, and thus resulting in a longer RT and reduced accuracy. Analyses of RT distributions have shown that the reduced accuracy to cI trials relative to iI trials is specifically due to fast, impulsive slips rather than to slow errors (Gratton et al., 1992; Stürmer et al., 2002). Similarly, LRP data have shown that the initial incorrect response activation typical of incongruent trials is greater for cI than for iI trials (Gratton et al., 1992; Stürmer & Leuthold, 2003; Stürmer et al., 2002). This pattern supports the notion that slips in these types of tasks are caused by the initial activation of the incorrect response by the irrelevant stimulus, and that control serves to attenuate this initial activation.

Consistently, neuroimaging data have shown that ACC activation is greatest to cI trials in the Eriksen task (Botvinick et al., 1999), the Stroop task (Egner & Hirsch, 2005b; Kerns et al., 2004), and the Simon task (Kerns, 2006; van Veen, 2006, Experiment 1). Furthermore, the reduction of conflict on the subsequent trial has been related to activation of the DLPFC (Egner & Hirsch, 2005a, 2005b; Kerns, 2006; Kerns et al., 2004; van Veen, 2006). Moreover, ACC activation during conflict trials has been found to predict both the DLPFC activation during the

subsequent high-control trials and the behavioral interference effect (Kerns, 2006; Kerns et al., 2004; van Veen, 2006).

Other data suggest that control does indeed operate to reduce interference by biasing stimulus processing toward the relevant stimulus dimension. Scerif, Worden, Davidson, Seiger, and Casey (2006) had participants perform an Eriksen task in which flankers without a central target were occasionally presented as foils. They showed that not only was the early P1 component of the ERP reduced for iI trials, suggesting that processing of the irrelevant stimulus features had been reduced due to increased control, but that this component was also reduced on foil trials preceded by incongruent rather than by congruent trials, suggesting that control was engaged by the preceding incongruent trial. Egner and Hirsch (2005a) used a Stroop task analogue in which participants were presented with the names and faces of famous politicians or actors and were asked to respond to either the name or the face, the other stimulus being the distractor. The researchers observed modulation of the fusiform face area by the conflict adaptation effect when faces were targets, but not when they were distractors, and also observed a tight functional coupling between the DLPFC and this area, suggesting that the role of the DLPFC in the conflict adaptation effect is to increase the processing of task-relevant stimuli, rather than to inhibit task-irrelevant stimuli, consistent with a biased-competition view of attentional selection.

Posterror slowing is another form of micro-trade-off that might reflect engagement of the conflict–control loop. It has long been known that people respond more slowly and accurately following error trials (Kleiter & Schwarzenbacher, 1989; Laming, 1979; Rabbitt, 1966; Rabbitt & Rodgers, 1977). Posterror slowing is frequently thought to be dependent on a control mechanism, and it appears to be associated with increased DLPFC activation on the trial following the error (Kerns et al., 2004; van Veen, 2006).

There also appears to be a relationship between error-related ACC activity and posterror slowing within participants: the greater the activity, the greater the extent to which participants tend to slow down during the subsequent trial (Debener et al., 2005; Garavan, Ross, Murphy, Roche, & Stein, 2002; Gehring et al., 1993). In an interesting recent study, Debener et al. simultaneously recorded both ERPs and fMRI and observed a within-subjects, trial-to-trial coupling between the ERN amplitude, error-related ACC activation as measured by fMRI, and posterror slowing. They suggested that the occasional failure to find a between-subjects relationship between ERN and posterror slowing (see, e.g., Hajcak, McDonald, & Simons, 2003) might be due to other between-subjects factors, such as individual differences, morphological variation of the ACC, or skull thickness.

Botvinick et al. (2001) implemented in several connectionist models the notion that the conflict adaptation effect and posterror slowing are conflict-driven, and they showed that the behavior of these models on these tasks provided a good fit to the empirically observed behavioral and neuroimaging data. In their model, Botvinick et al. (2001) assumed that high conflict during errors reduced activation of the response units on the subsequent trial, which caused the model to respond more slowly on trials following errors because more buildup of activation was needed for the response units to reach the decision threshold. Furthermore, since it had been found that posterror slowing tends to linger past the immediate posterror trial, Botvinick et al. (2001) assumed that the ACC integrates conflict over a series of several trials.

It should be emphasized here that the control mechanism underlying posterror slowing that they proposed is somewhat different from the control mechanism underlying the conflict adaptation effect. That is, in the models of Botvinick et al. (2001), control in the conflict adaptation effect was implemented as greater perceptual attention toward the relevant stimulus dimension; in posterror slowing, however, control was implemented as a change in the strategic priming of the response units. Nevertheless, Kerns et al. (2004) found that the same area of the DLPFC was implicated in both the conflict adaptation effect and posterror slowing. How or why the DLPFC's control mechanisms respond differently to the two instances of conflict is a question that remains to be addressed in empirical work and modeling studies. Interestingly, Brown, Reynolds, and Braver (2007) have recently described a computational model implementing two separate conflict–control loops. In their model, the detection of conflict between *simultaneously* active tasks and responses shifts control toward the processing of task-relevant information (and toward exploitation rather than exploration), whereas the detection of conflict across *successively* active tasks and responses shifts control toward accuracy on the speed–accuracy trade-off continuum (and toward exploration rather than exploitation). Their model is able to account for a large amount of task-switching data (including the finding that task switching takes longer following high-conflict trials; see Goschke, 2000; Gruber & Goschke, 2004), and it could very well explain why control engagement following error trials and control engagement following correct conflict trials are expressed somewhat differently in performance.

Interestingly, disturbances in conflict monitoring have been found in several psychiatric illnesses previously associated with control deficits, including schizophrenia, suggesting that at least part of the symptomatology and of the impaired executive control in these mental illnesses might be understood as arising from disturbances in action monitoring. Schizophrenia patients appear to show reduced conflict-related ACC activation (Kerns et al., 2005; Kopp & Rist, 1999) and a reduced conflict adaptation effect (Kerns et al., 2005); likewise, they show reduced error-related ACC activation (Alain, McNeely, He, Christensen, & West, 2002; Kerns et al., 2005; Kopp & Rist, 1999; Mathalon et al., 2002) and reduced posterror slowing (Kerns et al., 2005). Similarly, patients with attention deficit/hyperactivity disorder show a reduced ERN (Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005) and reduced posterror slowing (Schachar et al., 2004).

Despite the similarities between ACC activation to errors and to conflict trials, dissociations between ACC

activation to conflict and error trials can be predicted on the basis of this theory. Whereas at first glance such dissociations might appear to be incompatible with the conflict theory, they can actually be predicted from, and accounted for by, computational models. Specifically, Yeung and Cohen (2006) noted several observed dissociations in the literature and extended their computational models discussed earlier (Yeung et al., 2004) to account for these dissociations. First, it had been observed that alcohol intake reduces the ERN but leaves N2 intact (Ridderinkhof et al., 2002). In order to account for these data, Yeung and Cohen assumed that alcohol impairs stimulus processing, rather than directly affecting the functions of the ACC, and modeled this result by reducing the quality of the stimulus representations and reducing the model's attention to the relevant stimulus dimension. Conflict to incongruent stimuli did not differ between the "sober" and "intoxicated" models, because stimulus degradation and reduced attention had opposite effects on response unit activity; in contrast, conflict following errors was reduced in the simulated alcohol condition, because the corrective response activity was reduced. Thus, the model could account for the observed data by Ridderinkhof et al. A second observed dissociation between N2 and ERN involved a patient with a small localized lesion to the ACC who displayed an increased N2 but reduced ERN (Swick & Turken, 2002). In their model, Yeung and Cohen assumed that this lesion disrupted the control engagement following conflict detection rather than impacting conflict detection per se. Because of this reduced control, the model's performance during incongruent trials was impaired, which increased conflict preceding the response on correct trials; in contrast, conflict following errors in the model was reduced. Thus, again, the model proved a good fit for the observed data, showing that the theory can account for observed dissociations between ACC activation during correct conflict trials and during error trials.

We recently have noted that modulation by speed–accuracy trade-off provides another predicted double dissociation between conflict and errors (van Veen, 2006). We reasoned that conflict during correct trials should be greater when participants are emphasizing speed in their performance, but during error trials conflict should be greater when participants are emphasizing accuracy. Indeed, it is well known that error-related ACC activation is greater when participants are asked to emphasize accuracy (Falkenstein et al., 2000; Gehring et al., 1993; Ullsperger & von Cramon, 2004; van Veen, 2006), and neural network models of conflict can readily account for this (Yeung et al., 2004). Consistently, posterror slowing is greater when participants emphasize accuracy over speed (Jentzsch & Leuthold, 2006; Ullsperger & Szymanowski, 2004; van Veen, 2006), as is DLPFC activation on the trial following the error (van Veen, 2006). Conflict during correct trials, on the other hand, should be greater when participants emphasize speed, and indeed, the conflict N2 appears to be greater when participants emphasize speed over accuracy (Band, Ridderinkhof, & van der Molen, 2003; Jodo & Kayama, 1992). As predicted, and in contrast to how performance and network activation during and following errors are modulated by a speed–accuracy trade-off, we found the conflict adaptation effect and underlying neural activation to be greater under speed emphasis. Thus, the conflict adaptation effect, conflict-related ACC activation, and the subsequent control-related DLPFC activation are all greater under speed emphasis.

If conflict detection by the ACC leads to the engagement of control, lesions to the ACC should lead to impairment in the micro-adjustments discussed in the preceding paragraphs. Indeed, a recent study compared the performance of 8 patients with damage to the rostral ACC with the performance of control participants, and it found that ACC lesions reduced both posterror slowing and the conflict adaptation effect in a Simon task (di Pellegrino, Ciaramelli, & Làdavas, 2007). Thus, these results suggest that the ACC is indeed necessary for both types of micro-adjustments.

DISCREPANCIES

Despite the good fit between theory and data in many areas of research, it should be noted that some discrepancies exist. As explained, conflict theory predicts that greater conflict, with a greater ACC signal, should lead to greater engagement of control, and thus to greater subsequent performance adjustments. This relationship has not always been observed. One area of research that has led to inconsistent results involves the effect of pharmacological manipulations on performance and brain activity. For instance, it has been consistently observed that the administration of benzodiazepines reduces the ERN but does not impact posterror slowing (de Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004; Riba, Rodríguez-Fornells, Münte, & Barbanoj, 2005). Likewise, administration of catecholamine agonists and antagonists affects the ERN but has inconsistent effects on posterror performance. For instance, haloperidol (a dopamine antagonist) reduces the ERN (Zirnheld et al., 2004), whereas yohimbine (an epinephrine antagonist), D-amphetamine (a dopamine agonist), and caffeine increase it (de Bruijn et al., 2004; Riba, Rodríguez-Fornells, Morte, Münte, & Barbanoj, 2005; Tieges, Ridderinkhof, Snel, & Kok, 2004); however, none of these drugs have been found to affect posterror slowing. Thus, these data contradict the notion that increased ACC activation should result in subsequent performance adjustments, as predicted by the conflict theory.

Another source of discrepancies regarding conflict theory comes from studies of clinical populations. As mentioned earlier, some evidence supporting the conflict theory comes from data from patients suffering from schizophrenia; that is, schizophrenia patients, as compared with controls, show reduced ACC activation on correct conflict trials and on error trials, and also show reduced engagement of control, as evidenced by a reduced conflict adaptation effect and reduced posterror slowing (see, e.g., Kerns et al., 2005). However, in other psychiatric populations, this relationship does not appear to be as clear cut. Specifically, obsessive–compulsive disorder (OCD) is characterized by increased ACC activation on correct conflict trials and on error trials (Gehring, Himle,

& Nisenson, 2000; Hajcak & Simons, 2002; Johannes et al., 2003; Santesso, Segalowitz, & Schmidt, 2006; Ursu, Jones, Shear, Stenger, & Carter, 2003), but OCD patients do not appear to display increased posterror slowing (Hajcak & Simons, 2002). Similarly, conflict-related and error-related ACC activation are reduced in older adults (West, 2004; West & Moore, 2005), but little difference in the conflict adaptation effect and posterror slowing is observed between older and younger adults; if anything, these effects tend to be increased in older adults (West & Moore, 2005).

Finally, inconsistent results have been obtained from individuals with lesions to the ACC. Fellows and Farah (2005) compared the performance of 4 patients with ACC lesions with that of a control group, and in the patient group they found intact Stroop interference and posterror slowing. Similarly, Critchley et al. (2003) observed that 3 patients with ACC lesions were largely unimpaired on a variety of attentional tests, displaying only reduced autonomic responsiveness. On the other hand, Ochsner et al. (2001) discussed a patient who, following a cingulotomy, displayed impairment on a variety of tasks requiring cognitive control, including the Stroop task. In addition, as mentioned earlier, a recent study of 8 patients with ACC lesions showed that both the conflict adaptation effect and posterror slowing were reduced in the patient group relative to controls (di Pellegrino et al., 2007). Thus, evidence from patients with ACC lesions is mixed. Perhaps one reason for such discrepancies is that some of these studies have lacked statistical power. Regardless, future research needs to address the findings that appear inconsistent with the theory. Perhaps modeling can be used to account for these discrepant findings, much as Yeung and Cohen (2006) used a model to account for the findings of Swick and Turken (2002).

ALTERNATIVE THEORIES

In addition to the theory that the ERN reflects error detection by a comparator process, as mentioned earlier, there have been other theoretical attempts to explain the evidence discussed so far. We will now discuss several of these alternative theories and their data and contrast them with conflict theory.

It has been claimed that stimulus priming effects can account for the behavioral conflict adaptation effect in the Eriksen task (Mayr, Awh, & Laurey, 2003). Mayr et al. pointed out that half of both cC and iI trials consist of exact repetitions and showed that, by removing these stimulus repetitions, the conflict adaptation effect was removed. This finding suggested that the effect might exist independent of control. However, controlling for stimulus repetitions has never been shown to remove the conflict adaptation effect in the Simon task (Stürmer et al., 2002; Wühr & Ansorge, 2005) or in the Stroop task (Egner & Hirsch, 2005a, 2005b; Kerns et al., 2004); even in the Eriksen task, the Mayr et al. null effect has not always been replicated (Ullsperger et al., 2005). Ullsperger et al. suggested that the null results found by Mayr et al. might have been due to negative priming or task switching. However, Nieuwenhuis et al. (2006) failed to find significant conflict adaptation effects after removing repetition effects in the Eriksen task, and they suggested that this task might differ from the Stroop, Simon, and related tasks in such important ways that it might simply not be suited to the study of control engagement following conflict. How or why these different findings occur remains to be seen, since conflict-related control engagement appears to be robust in the Stroop and Simon tasks (Egner & Hirsch, 2005a, 2005b; Kerns, 2006; Kerns et al., 2004; Stürmer et al., 2002).

Holroyd and Coles (2002) have proposed a different interpretation of conflict-related and error-related ACC activation, in a model based on reinforcement learning theory. They proposed that behavior is monitored by an "adaptive critic," localized in the basal ganglia. This adaptive critic determines whether events are better or worse than expected, signaling this distinction with a phasic increase or decrease, respectively, in dopaminergic activity in the ACC. According to this elegant and biologically appealing theory, different cognitive processes compete for access to the motor system, and the function of the ACC is to select between these different cognitive processes on the basis of how it has been trained by the dopamine signal from the basal ganglia. Holroyd and Coles assume that error-related ACC activation is generated by a phasic reduction in dopamine influx; the inhibitory influence of the dopaminergic innervation in the ACC is briefly disrupted, fine-tuning the ACC to do a more appropriate selection job on future trials. Holroyd and Coles based their proposal in large part on findings involving an ERP component that is elicited by error feedback stimuli and that somewhat resembles the ERN (or N2), often referred to as the *feedback-related negativity*. This component has also been modeled as having an ACC generator (Miltner, Braun, & Coles, 1997). However, fMRI studies of the Miltner et al. (1997) paradigm have generally failed to show significant ACC activation to error feedback stimuli, thus casting doubt on the assumption that this component is necessarily generated by the ACC (Nieuwenhuis, Slagter, Alting von Geusau, Heslenfeld, & Holroyd, 2005; van Veen, Holroyd, Cohen, Stenger, & Carter, 2004).

Another compelling theory has been put forward by Brown and Braver (2005), who proposed that rather than detecting conflict, the ACC detects situations in which errors are likely. They used a change-signal task, in which participants had to make a forced choice response to a go stimulus unless a second stimulus, presented shortly afterward, indicated that the opposite response was required (a "change" stimulus). Easy and hard conditions were obtained by varying the stimulus onset asynchrony between the go and change stimuli. Prior to the go and change stimuli, a cue instructed participants whether the upcoming stimulus set was going to be difficult or easy. Brown and Braver found that ACC activation was greatest to difficult change stimuli and smallest to easy go stimuli, as predicted by their model. According to them, a model in which the ACC responded to the conflict between the two responses would have predicted that the ACC should respond equally strongly to change stimuli under hard

and easy conditions, because of the equivalent levels of conflict in their model. However, other experiments have failed to find effects on ACC activation from cues that signal error likelihood (Nieuwenhuis, Schweizer, Mars, Botvinick, & Hajcak, 2007). Thus, we believe that more evidence is needed in order to properly evaluate both Holroyd and Coles's (2002) reinforcement learning theory and Brown and Braver's error likelihood theory. Nevertheless, an attractive feature of both theories is that they can account well for learning effects, something that conflict-based models have not yet attempted to do.

Another interesting theory has been put forward by Critchley and colleagues (Critchley, 2005; Critchley et al., 2003; Critchley, Tang, Glaser, Butterworth, & Dolan, 2005). They have observed that ACC activation closely predicts various measures of autonomic arousal, and furthermore, that task-induced autonomic arousal is considerably reduced in patients with lesions to the ACC. They have suggested that, rather than a strictly cognitive function, the ACC appears to primarily regulate autonomic arousal in order to accommodate task demands. Critchley (2005) has proposed that the generation of autonomic arousal is detected by areas including the insular and orbitofrontal cortices, and that this contributes to the motivational state. We believe that this evidence linking ACC activation to autonomic arousal is entirely consistent with the view of the ACC as contributing indirectly to cognitive control and could be considered complementary to the notion that this region has a fundamental role in regulating control by detecting conflict.

Another idea, based mostly on research involving nonhuman animals, is that the ACC relates actions to their perceived consequences (see, e.g., Rushworth, Walton, Kennerley, & Bannerman, 2004). That is, the ACC is proposed to play a role in encoding the relationship between an action and the reinforcement value of its outcome, even when the outcome is a reward and not an error. Although this work is interesting and provocative, the relationship between this theory and the notion of conflict monitoring is unclear. There are, in fact, some parallels between the two theories, since according to the modeling work of Botvinick et al. (2001), conflict signals are integrated across several trials in much the same way as action–outcome associations are in the Rushworth model. Future theoretical and modeling work might clarify the degree to which these theories are complementary or inconsistent with each other.

CONCLUSIONS

In this review, we have argued that one role of the ACC in the cognitive architecture is to detect conflicts between simultaneously active, competing representations and to engage context representations in the DLPFC, resulting in increased cognitive control. We have made this argument on the basis of data obtained from a limited set of tasks (speeded response interference tasks). However, the theory has also been applied to other areas of cognitive research, including underdetermined responding (Barch, Braver, Sabb, & Noll, 2000), moral judgment (Greene, Nystrom, Engell, Darley, & Cohen, 2004), causal reasoning (Fugelsang & Dunbar, 2005), emotion regulation in anxiety (Bishop, Duncan, Brett, & Lawrence, 2004), the tip-of-the-tongue phenomenon (Maril, Wagner, & Schacter, 2001), thought suppression (Anderson et al., 2004), and mnemonic competition (Kuhl, Dudukovic, Kahn, & Wagner, 2007), among others. We find this broadness of application particularly interesting; if this conflict–control loop is such an important part of cognitive architecture, its engagement should be evident in other phenomena above and beyond speeded forced choice response tasks, and we are interested in seeing whether the theory also generalizes to other cognitive phenomena.

Several other issues will also need to be addressed, apart from those already mentioned in the preceding paragraphs. Future studies might, for instance, focus on how the ACC engages the DLPFC; this could be done via direct connections, or potentially by increasing activation of the locus coeruleus, thus increasing norepinephrine influx into the DLPFC (Aston-Jones & Cohen, 2005; Cohen, Botvinick, & Carter, 2000). Other lines of research might investigate the effects of cingulate lesions on conflict monitoring; to date, relatively few studies have done this, and they have achieved mixed results (di Pellegrino et al., 2007; Fellows & Farah, 2005; Ochsner et al., 2001). Another potentially interesting topic of future investigation is the relationship among awareness of conflict, ACC activation (Mayr, 2004), and trial-to-trial adjustments.

In addition, one area that needs further empirical investigation is data recorded from single cells in nonhuman primates. Much interesting work has suggested that differences might exist not only between monkey and human task performance (see Ito, Stuphorn, Brown, & Schall, 2003), as displayed in differences between ACC activation as recorded by fMRI from humans and as recorded from single cells in nonhuman primates (see, e.g., Ito et al., 2003; Nakamura, Roesch, & Olson, 2005), but also between the cytoarchitecture (Nimchinsky et al., 1999) and sulcal patterns (Fornito et al., 2004; Paus et al., 1996) of the different species. Future fMRI studies of behaving primates and different kinds of electrophysiology (e.g., recording field potentials) may shed light on this interesting and important issue.

In conclusion, the conflict–control loop theory provides an integrative and parsimonious account for a large body of empirical work concerning how humans adjust control during task performance in response to changing task demands. We are excited by the large amount of interest this theory has received and the empirical research it has stimulated over the past decade. However, much more empirical and theoretical work remains in order to understand the neural basis of cognitive control, in both healthy and mentally ill brains.

AUTHOR NOTE

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REFERENCES

ALAIN, C., MCNEELY, H. E., HE, Y., CHRISTENSEN, B. K., & WEST, R. (2002). Neurophysiological evidence of error-monitoring deficits in patients with schizophrenia. *Cerebral Cortex*, **12**, 840-846.

ANDERSON, M. C., OCHSNER, K. N., KUHL, B., COOPER, J., ROBERTSON, E., GABRIELI, S. W., ET AL. (2004). Neural systems underlying the suppression of unwanted memories. *Science*, **303**, 232-235.

ASTON-JONES, G., & COHEN, J. D. (2005). An integrative theory of locus coeruleus–norepinephrine function: Adaptive gain and optimal performance. *Annual Review of Neuroscience*, **28**, 403-450.

BADRE, D., & WAGNER, A. D. (2004). Selection, integration, and conflict monitoring: Assessing the nature and generality of prefrontal cognitive control mechanisms. *Neuron*, **41**, 473-487.

BAND, G. P. H., RIDDERINKHOF, K. R., & VAN DER MOLEN, M. W. (2003). Speed–accuracy modulation in case of conflict: The roles of activation and inhibition. *Psychological Research*, **67**, 266-279.

BARCH, D. M., BRAVER, T. S., SABB, F. W., & NOLL, D. C. (2000). Anterior cingulate and the monitoring of response conflict: Evidence from an fMRI study of overt word generation. *Journal of Cognitive Neuroscience*, **12**, 298-309.

BISHOP, S., DUNCAN, J., BRETT, M., & LAWRENCE, A. D. (2004). Prefrontal cortical function and anxiety: Controlling attention to threat-related stimuli. *Nature Neuroscience*, **7**, 184-188.

BOTVINICK, M. M., BRAVER, T. S., BARCH, D. M., CARTER, C. S., & COHEN, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, **108**, 624-652.

BOTVINICK, M. M., COHEN, J. D., & CARTER, C. S. (2004). Conflict monitoring and anterior cingulate cortex: An update. *Trends in Cognitive Sciences*, **8**, 539-546.

BOTVINICK, M. M., NYSTROM, L. E., FISSELL, K., CARTER, C. S., & COHEN, J. D. (1999). Conflict monitoring versus selection-for-action in anterior cingulate cortex. *Nature*, **402**, 179-181.

BRAVER, T. S., BARCH, D. M., GRAY, J. R., MOLFESE, D. L., & SNYDER, A. (2001). Anterior cingulate cortex and response conflict: Effects of frequency, inhibition and errors. *Cerebral Cortex*, **11**, 825-836.

BROWN, J. W., & BRAVER, T. S. (2005). Learned predictions of error likelihood in the anterior cingulate cortex. *Science*, **307**, 1118-1121.

BROWN, J. W., REYNOLDS, J. R., & BRAVER, T. S. (2007). A computational model of fractionated conflict-control mechanisms in task-switching. *Cognitive Psychology*, **55**, 37-85.

BURLE, B., POSSAMAÏ, C.-A., VIDAL, F., BONNET, M., & HASBROUCQ, T. (2002). Executive control in the Simon effect: An electromyographic and distributional analysis. *Psychological Research*, **66**, 324-336.

CARTER, C. S., BRAVER, T. S., BARCH, D. M., BOTVINICK, M. M., NOLL, D., & COHEN, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, **280**, 747-749.

COHEN, J. D., BOTVINICK, M. M., & CARTER, C. S. (2000). Anterior cingulate and prefrontal cortex: Who's in control? *Nature Neuroscience*, **3**, 421-423.

COHEN, J. D., DUNBAR, K., & MCCLELLAND, J. L. (1990). On the control of automatic processes: A parallel distributed processing account of the Stroop effect. *Psychological Review*, **97**, 332-361.

COLES, M. G. H., GRATTON, G., BASHORE, T. R., ERIKSEN, C. W., & DONCHIN, E. (1985). A psychophysiological investigation of the continuous flow model of human information processing. *Journal of Experimental Psychology: Human Perception & Performance*, **11**, 529-553.

COOKE, J. D., & DIGGLES, V. A. (1984). Rapid error correction during human arm movements: Evidence for central monitoring. *Journal of Motor Behavior*, **16**, 348-363.

CRITCHLEY, H. D. (2005). Neural mechanisms of autonomic, affective, and cognitive integration. *Journal of Comparative Neurology*, **493**, 154-166.

CRITCHLEY, H. D., MATHIAS, C. J., JOSEPHS, O., O'DOHERTY, J., ZANINI, S., DEWAR, B.-K., ET AL. (2003). Human cingulate cortex and autonomic control: Converging neuroimaging and clinical evidence. *Brain*, **126**, 2139-2152.

CRITCHLEY, H. D., TANG, J., GLASER, D., BUTTERWORTH, B., & DOLAN, R. J. (2005). Anterior cingulate activity during error and autonomic response. *NeuroImage*, **27**, 885-895.

DE BRUIJN, E. R. A., HULSTIJN, W., VERKES, R. J., RUIGT, G. S. F., & SABBE, B. G. C. (2004). Drug-induced stimulation and suppression of action monitoring in healthy volunteers. *Psychopharmacology*, **177**, 151-160.

DE JONG, R., LIANG, C.-C., & LAUBER, E. J. (1994). Conditional and unconditional automaticity: A dual-process model of effects of spatial stimulus–response correspondence. *Journal of Experimental Psychology: Human Perception & Performance*, **20**, 731-750.

DE JONG, R., WIERDA, M., MULDER, G., & MULDER, L. J. (1988). Use of partial stimulus information in response processing. *Journal of Experimental Psychology: Human Perception & Performance*, **14**, 682-692.

DEBENER, S., ULLSPERGER, M., SIEGEL, M., FIEHLER, K., VON CRAMON, D. Y., & ENGEL, A. K. (2005). Trial-by-trial coupling of concurrent electroencephalogram and functional magnetic resonance imaging identifies the dynamics of performance monitoring. *Journal of Neuroscience*, **25**, 11730-11737.

DEHAENE, S., NACCACHE, L., LE CLEC'H, G., KOECHLIN, E., MUELLER, M., DEHAENE-LAMBERTZ, G., ET AL. (1998). Imaging unconscious semantic priming. *Nature*, **395**, 597-600.

DEHAENE, S., POSNER, M. I., & TUCKER, D. M. (1994). Localization of a neural system for error detection and compensation. *Psychological Science*, **5**, 303-305.

DI PELLEGRINO, G., CIARAMELLI, E., & LÀDAVAS, E. (2007). The regulation of cognitive control following rostral anterior cingulate cortex lesion in humans. *Journal of Cognitive Neuroscience*, **19**, 275-286.

DURSTON, S., THOMAS, K. M., WORDEN, M. S., YANG, Y., & CASEY, B. J. (2002). The effect of preceding context on inhibition: An event-related fMRI study. *NeuroImage*, **16**, 449-453.

EGNER, T., & HIRSCH, J. (2005a). Cognitive control mechanisms resolve conflict through cortical amplification of task-relevant information. *Nature Neuroscience*, **8**, 1784-1790.

EGNER, T., & HIRSCH, J. (2005b). The neural correlates and functional integration of cognitive control in a Stroop task. *NeuroImage*, **24**, 539-547.

EIMER, M. (1995). Stimulus–response compatibility and automatic response activation: Evidence from psychophysiological studies. *Journal of Experimental Psychology: Human Perception & Performance*, **21**, 837-854.

EIMER, M., HOMMEL, B., & PRINZ, W. (1995). S–R compatibility and response selection. *Acta Psychologica*, **90**, 301-313.

ERIKSEN, B. A., & ERIKSEN, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics*, **16**, 143-149.

ERIKSEN, C. W., & SCHULTZ, D. W. (1979). Information processing in visual search: A continuous flow conception and experimental results. *Perception & Psychophysics*, **25**, 249-263.

FALKENSTEIN, M., HOHNSBEIN, J., HOORMANN, J., & BLANKE, L. (1991). Effects of crossmodal divided attention on late ERP components: II. Error processing in choice reaction tasks. *Electroencephalography & Clinical Neurophysiology*, **78**, 447-455.

FALKENSTEIN, M., HOORMANN, J., CHRIST, S., & HOHNSBEIN, J. (2000). ERP components on reaction errors and their functional significance: A tutorial. *Biological Psychology*, **51**, 87-107.

FELLOWS, L. K., & FARAH, M. J. (2005). Is anterior cingulate cortex necessary for cognitive control? *Brain*, **128**, 788-796.

FORNITO, A., YÜCEL, M., WOOD, S., STUART, G. W., BUCHANAN, J.-A., PROFFITT, T., ET AL. (2004). Individual differences in anterior cingulate/paracingulate morphology are related to executive functions in healthy males. *Cerebral Cortex*, **14**, 424-431.

FUGELSANG, J. A., & DUNBAR, K. N. (2005). Brain-based mechanisms underlying complex causal thinking. *Neuropsychologia*, **43**, 1204-1213.

GARAVAN, H., ROSS, T. J., KAUFMAN, J., & STEIN, E. A. (2003). A midline dissociation between error-processing and response-conflict monitoring. *NeuroImage*, **20**, 1132-1139.

GARAVAN, H., ROSS, T. J., MURPHY, K., ROCHE, R. A. P., & STEIN, E. A. (2002). Dissociable executive functions in the dynamic control of behavior: Inhibition, error detection, and correction. *NeuroImage*, **17**, 1820-1829.

GEHRING, W. J., & FENCSIK, D. E. (2001). Functions of the medial frontal cortex in the processing of conflict and errors. *Journal of Neuroscience*, **21**, 9430-9437.

GEHRING, W. J., GOSS, B., COLES, M. G. H., MEYER, D. E., & DON-

CHIN, E. (1993). A neural system for error detection and compensation. *Psychological Science*, **4**, 385-390.

GEHRING, W. J., GRATTON, G., COLES, M. G. H., & DONCHIN, E. (1992). Probability effects on stimulus evaluation and response processes. *Journal of Experimental Psychology: Human Perception & Performance*, **18**, 198-216.

GEHRING, W. J., HIMLE, J., & NISENSON, L. G. (2000). Action-monitoring dysfunction in obsessive-compulsive disorder. *Psychological Science*, **11**, 1-6.

GOSCHKE, T. (2000). Intentional reconfiguration and involuntary persistence in task set switching. In S. Monsell & J. Driver (Eds.), *Control of cognitive processes: Attention and performance XVIII* (pp. 331-355). Cambridge, MA: MIT Press.

GRATTON, G., COLES, M. G. H., & DONCHIN, E. (1992). Optimizing the use of information: Strategic control of activation of responses. *Journal of Experimental Psychology: General*, **121**, 480-506.

GRATTON, G., COLES, M. G. H., SIREVAAG, E. J., ERIKSEN, C. W., & DONCHIN, E. (1988). Pre- and poststimulus activation of response channels: A psychophysiological analysis. *Journal of Experimental Psychology: Human Perception & Performance*, **14**, 331-344.

GREENE, J. D., NYSTROM, L. E., ENGELL, A. D., DARLEY, J. M., & COHEN, J. D. (2004). The neural bases of cognitive conflict and control in moral judgment. *Neuron*, **44**, 389-400.

GRUBER, O., & GOSCHKE, T. (2004). Executive control emerging from dynamic interactions between brain systems mediating language, working memory and attentional processes. *Acta Psychologica*, **115**, 105-121.

HAJCAK, G., MCDONALD, N., & SIMONS, R. F. (2003). To err is autonomic: Error-related brain potentials, ANS activity, and post-error compensatory behavior. *Psychophysiology*, **40**, 895-903.

HAJCAK, G., & SIMONS, R. F. (2002). Error-related brain activity in obsessive-compulsive undergraduates. *Psychiatry Research*, **110**, 63-72.

HOLROYD, C. B., & COLES, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, **109**, 679-709.

HOLROYD, C. B., DIEN, J., & COLES, M. G. H. (1998). Error-related scalp potentials elicited by hand and foot movements: Evidence for an output-independent error-processing system in humans. *Neuroscience Letters*, **242**, 65-68.

HOLROYD, C. B., YEUNG, N., COLES, M. G. H., & COHEN, J. D. (2005). A mechanism for error detection in speeded response time tasks. *Journal of Experimental Psychology: General*, **134**, 163-191.

ITO, S., STUPHORN, V., BROWN, J. W., & SCHALL, J. D. (2003). Performance monitoring by the anterior cingulate cortex during saccade countermanding. *Science*, **302**, 120-122.

JENTZSCH, I., & LEUTHOLD, H. (2006). Control over speeded actions: A common processing locus for micro- and macro-trade-offs? *Quarterly Journal of Experimental Psychology*, **59**, 1329-1337.

JODO, E., & KAYAMA, Y. (1992). Relation of a negative ERP component to response inhibition in a go/no-go task. *Electroencephalography & Clinical Neurophysiology*, **82**, 477-482.

JOHANNES, S., WIERINGA, B. M., NAGER, W., RADA, D., MÜLLER-VAHL, K. R., EMRICH, H. M., ET AL. (2003). Tourette Syndrome and obsessive-compulsive disorder: Event-related brain potentials show similar mechanisms of frontal inhibition but dissimilar target evaluation processes. *Behavioural Neurology*, **14**, 9-17.

JONES, A. D., CHO, R. Y., NYSTROM, L. E., COHEN, J. D., & BRAVER, T. S. (2002). A computational model of anterior cingulate function in speeded response tasks: Effects of frequency, sequence, and conflict. *Cognitive, Affective, & Behavioral Neuroscience*, **2**, 300-317.

KARLIN, L., MARTZ, M. J., & MORDKOFF, A. M. (1970). Motor performance and sensory-evoked potentials. *Electroencephalography & Clinical Neurophysiology*, **28**, 307-313.

KERNS, J. G. (2006). Anterior cingulate and prefrontal cortex activity in an fMRI study of trial-to-trial adjustments on the Simon task. *NeuroImage*, **33**, 399-405.

KERNS, J. G., COHEN, J. D., MACDONALD, A. W., III, CHO, R. Y., STENGER, V. A., & CARTER, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science*, **303**, 1023-1026.

KERNS, J. G., COHEN, J. D., MACDONALD, A. W., III, JOHNSON, M. K., STENGER, V. A., AIZENSTEIN, H., & CARTER, C. S. (2005). Decreased conflict- and error-related activity in the anterior cingulate cortex in subjects with schizophrenia. *American Journal of Psychiatry*, **162**, 1833-1839.

KLEITER, G. D., & SCHWARZENBACHER, K. (1989). Beyond the answer: Post-error processes. *Cognition*, **32**, 255-277.

KOPP, B., MATTLER, U., GOERTZ, R., & RIST, F. (1996). N2, P3 and the lateralized readiness potential in a nogo task involving selective response priming. *Electroencephalography & Clinical Neurophysiology*, **99**, 19-27.

KOPP, B., & RIST, F. (1999). An event-related brain potential substrate of disturbed response monitoring in paranoid schizophrenia patients. *Journal of Abnormal Psychology*, **108**, 337-346.

KOPP, B., RIST, F., & MATTLER, U. (1996). N200 in the flanker task as a neurobehavioral tool for investigating executive control. *Psychophysiology*, **33**, 282-294.

KORNBLUM, S., HASBROUCQ, T., & OSMAN, A. (1990). Dimensional overlap: Cognitive basis for stimulus–response compatibility: A model and taxonomy. *Psychological Review*, **97**, 253-270.

KUHL, B. A., DUDUKOVIC, N. M., KAHN, I., & WAGNER, A. D. (2007). Decreased demands on cognitive control reveal the neural processing benefits of forgetting. *Nature Neuroscience*, **10**, 908-914.

KUNDE, W. (2003). Sequential modulations of stimulus–response correspondence effects depend on awareness of response conflict. *Psychonomic Bulletin & Review*, **10**, 198-205.

LAMING, D. (1979). Choice reaction performance following an error. *Acta Psychologica*, **43**, 199-224.

LIOTTI, M., PLISZKA, S. R., PEREZ, R., KOTHMANN, D., & WOLDORFF, M. G. (2005). Abnormal brain activity related to performance monitoring and error detection in children with ADHD. *Cortex*, **41**, 377-388.

LUU, P., & TUCKER, D. M. (2001). Regulating action: Alternating activation of midline frontal and motor cortical networks. *Clinical Neurophysiology*, **112**, 1295-1306.

MACLEOD, C. M. (1991). Half a century of research on the Stroop effect: An integrative review. *Psychological Bulletin*, **109**, 163-203.

MACLEOD, C. M. (1992). The Stroop task: The "gold standard" of attentional measures. *Journal of Experimental Psychology: General*, **121**, 12-14.

MACLEOD, C. M., & MACDONALD, P. A. (2000). Interdimensional interference in the Stroop effect: Uncovering the cognitive and neural anatomy of attention. *Trends in Cognitive Sciences*, **4**, 383-391.

MARIL, A., WAGNER, A. D., & SCHACTER, D. L. (2001). On the tip of the tongue: An event-related fMRI study of semantic retrieval failure and cognitive conflict. *Neuron*, **31**, 653-660.

MATHALON, D. H., FEDOR, M., FAUSTMAN, W. O., GRAY, M., ASKARI, N., & FORD, J. M. (2002). Response-monitoring dysfunction in schizophrenia: An event-related brain potential study. *Journal of Abnormal Psychology*, **111**, 22-41.

MATHALON, D. H., WHITFIELD, S. L., & FORD, J. M. (2003). Anatomy of an error: ERP and fMRI. *Biological Psychology*, **64**, 119-141.

MAYR, U. (2004). Conflict, consciousness, and control. *Trends in Cognitive Sciences*, **8**, 145-148.

MAYR, U., AWH, E., & LAUREY, P. (2003). Conflict adaptation effects in the absence of executive control. *Nature Neuroscience*, **6**, 450-452.

MILHAM, M. P., BANICH, M. T., WEBB, A., BARAD, V., COHEN, N. J., WSZALEK, T., & KRAMER, A. F. (2001). The relative involvement of anterior cingulate and prefrontal cortex in attentional control depends on nature of conflict. *Cognitive Brain Research*, **12**, 467-473.

MILLER, E. K., & COHEN, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, **24**, 167-202.

MILLER, J. (1998). Effects of stimulus–response probability on choice reaction time: Evidence from the lateralized readiness potential. *Journal of Experimental Psychology: Human Perception & Performance*, **24**, 1521-1534.

MILLER, J., & HACKLEY, S. A. (1992). Electrophysiological evidence for temporal overlap among contingent mental processes. *Journal of Experimental Psychology: General*, **121**, 195-209.

MILTNER, W. H. R., BRAUN, C. H., & COLES, M. G. H. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a "generic" neural system for error detection. *Journal of Cognitive Neuroscience*, **9**, 788-798.

MILTNER, W. H. R., LEMKE, U., WEISS, T., HOLROYD, C., SCHEFFERS, M. K., & COLES, M. G. H. (2003). Implementation of error-processing in the human anterior cingulate cortex: A source analysis

of the magnetic equivalent of the error-related negativity. *Biological Psychology*, **64**, 157-166.

NAKAMURA, K., ROESCH, M. R., & OLSON, C. R. (2005). Neuronal activity in macaque SEF and ACC during performance of tasks involving conflict. *Journal of Neurophysiology*, **93**, 884-908.

NELSON, J. K., REUTER-LORENZ, P. A., SYLVESTER, C.-Y. C., JONIDES, J., & SMITH, E. E. (2003). Dissociable neural mechanisms underlying response-based and familiarity-based conflict in working memory. *Proceedings of the National Academy of Sciences*, **100**, 11171-11175.

NIEUWENHUIS, S., SCHWEIZER, T. S., MARS, R. B., BOTVINICK, M. M., & HAJCAK, G. (2007). Error-likelihood prediction in the medial frontal cortex: A critical evaluation. *Cerebral Cortex*, **17**, 1570-1581.

NIEUWENHUIS, S., SLAGTER, H. A., ALTING VON GEUSAU, N. J., HESLENFELD, D. J., & HOLROYD, C. B. (2005). Knowing good from bad: Differential activation of human cortical areas by positive and negative outcomes. *European Journal of Neuroscience*, **21**, 3161-3168.

NIEUWENHUIS, S., STINS, J. F., POSTHUMA, D., POLDERMAN, T. J. C., BOOMSMA, D. I., & DE GEUS, E. J. (2006). Accounting for sequential trial effects in the flanker task: Conflict adaptation or associative priming? *Memory & Cognition*, **34**, 1260-1272.

NIEUWENHUIS, S., YEUNG, N., VAN DEN WILDENBERG, W., & RIDDERINKHOF, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: Effects of response conflict and trial type frequency. *Cognitive, Affective, & Behavioral Neuroscience*, **3**, 17-26.

NIMCHINSKY, E. A., GILISSEN, E., ALLMAN, J. M., PERL, D. P., ERWIN, J. M., & HOF, P. R. (1999). A neuronal morphologic type unique to humans and great apes. *Proceedings of the National Academy of Sciences*, **96**, 5268-5273.

OCHSNER, K. N., KOSSLYN, S. M., COSGROVE, G. R., CASSEM, E. H., PRICE, B. H., NIERENBERG, A. A., & RAUCH, S. L. (2001). Deficits in visual cognition and attention following bilateral anterior cingulotomy. *Neuropsychologia*, **39**, 219-230.

PAUS, T., TOMAIUOLO, F., OTAKY, N., MACDONALD, D., PETRIDES, M., ATLAS, J., ET AL. (1996). Human cingulate and paracingulate sulci: Pattern, variability, asymmetry, and probabilistic map. *Cerebral Cortex*, **6**, 207-214.

PRAAMSTRA, P., & OOSTENVELD, R. (2003). Attention and movement-related motor cortex activation: A high-density EEG study of spatial stimulus–response compatibility. *Cognitive Brain Research*, **16**, 309-322.

PRITCHARD, W. S., SHAPPELL, S. A., & BRANDT, M. E. (1991). Psychophysiology of N200/N400: A review and classification scheme. In J. R. Jennings, P. K. Ackles, & M. G. H. Coles (Eds.), *Advances in psychophysiology* (Vol. 4, pp. 43-106). Greenwich, CT: JAI Press.

RABBITT, P. M. A. (1966). Errors and error-correction in choice-response tasks. *Journal of Experimental Psychology*, **71**, 264-272.

RABBITT, P. M. A. (1967). Time to detect errors as a function of factors affecting choice-response time. *Acta Psychologica*, **27**, 131-142.

RABBITT, P. M. A. (2002). Consciousness is slower than you think. *Quarterly Journal of Experimental Psychology*, **55A**, 1081-1092.

RABBITT, P. M. A., & RODGERS, B. (1977). What does a man do after he makes an error? An analysis of response programming. *Quarterly Journal of Experimental Psychology*, **29**, 727-743.

RABBITT, P. M. A., & VYAS, S. (1981). Processing a display even after you make a response to it: How perceptual errors can be corrected. *Quarterly Journal of Experimental Psychology*, **33A**, 223-239.

RIBA, J., RODRÍGUEZ-FORNELLS, A., MORTE, A., MÜNTE, T. F., & BARBANOJ, M. J. (2005). Noradrenergic stimulation enhances human action monitoring. *Journal of Neuroscience*, **25**, 4370-4374.

RIBA, J., RODRÍGUEZ-FORNELLS, A., MÜNTE, T. F., & BARBANOJ, M. J. (2005). A neurophysiological study of the detrimental effects of alprazolam on human action monitoring. *Cognitive Brain Research*, **25**, 554-565.

RIDDERINKHOF, K. R. (2002). Micro- and macro-adjustments of task set: Activation and suppression in conflict tasks. *Psychological Research*, **66**, 312-323.

RIDDERINKHOF, [K.] R., DE VLUGT, Y., BRAMLAGE, A., SPAAN, M., ELTON, M., SNEL, J., & BAND, G. P. H. (2002). Alcohol consumption impairs detection of performance errors in mediofrontal cortex. *Science*, **298**, 2209-2211.

RIDDERINKHOF, K. R., & VAN DER MOLEN, M. W. (1995). When global information and local information collide: A brain potential analysis of the locus of interference effects. *Biological Psychology*, **41**, 29-53.

RODRÍGUEZ-FORNELLS, A., KURZBUCH, A. R., & MÜNTE, T. F. (2002). Time course of error detection and correction in humans: Neurophysiological evidence. *Journal of Neuroscience*, **22**, 9990-9996.

RUBIA, K., SMITH, A. B., BRAMMER, M. J., & TAYLOR, E. (2003). Right inferior prefrontal cortex mediates response inhibition while mesial prefrontal cortex is responsible for error detection. *NeuroImage*, **20**, 351-358.

RUSHWORTH, M. F. S., WALTON, M. E., KENNERLEY, S. W., & BANNERMAN, D. M. (2004). Action set and decisions in the medial frontal cortex. *Trends in Cognitive Sciences*, **8**, 410-417.

SANTESSO, D. L., SEGALOWITZ, S. J., & SCHMIDT, L. A. (2006). Error-related electrocortical responses are enhanced in children with obsessive-compulsive behaviors. *Developmental Neuropsychology*, **29**, 431-445.

SCERIF, G., WORDEN, M. S., DAVIDSON, M., SEIGER, L., & CASEY, B. J. (2006). Context modulates early stimulus processing when resolving stimulus–response conflict. *Journal of Cognitive Neuroscience*, **18**, 781-792.

SCHACHAR, R. J., CHEN, S., LOGAN, G. D., ORNSTEIN, T. J., CROSBIE, J., ICKOWICZ, A., & PAKULAK, A. (2004). Evidence for an error monitoring deficit in attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*, **32**, 285-293.

SCHEFFERS, M. K., & COLES, M. G. H. (2000). Performance monitoring in a confusing world: Error-related brain activity, judgments of response accuracy, and types of errors. *Journal of Experimental Psychology: Human Perception & Performance*, **26**, 141-151.

SCHEFFERS, M. K., COLES, M. G. H., BERNSTEIN, P., GEHRING, W. J., & DONCHIN, E. (1996). Event-related brain potentials and error-related processing: An analysis of incorrect responses to go and no-go stimuli. *Psychophysiology*, **33**, 42-53.

SIMON, J. R. (1969). Reactions toward the source of stimulation. *Journal of Experimental Psychology*, **81**, 174-176.

SIMON, J. R., & BERBAUM, K. (1990). Effect of conflicting cues on information processing: The "Stroop effect" vs. the "Simon effect." *Acta Psychologica*, **73**, 159-170.

SPENCER, K. M., & COLES, M. G. H. (1999). The lateralized readiness potential: Relationship between human data and response activation in a connectionist model. *Psychophysiology*, **36**, 364-370.

STROOP, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, **18**, 643-662.

STÜRMER, B., & LEUTHOLD, H. (2003). Control over response priming in visuomotor processing: A lateralized readiness potential study. *Experimental Brain Research*, **153**, 35-44.

STÜRMER, B., LEUTHOLD, H., SOETENS, E., SCHRÖTER, H., & SOMMER, W. (2002). Control over location-based response activation in the Simon task: Behavioral and electrophysiological evidence. *Journal of Experimental Psychology: Human Perception & Performance*, **28**, 1345-1363.

SWICK, D., & TURKEN, A. U. (2002). Dissociation between conflict detection and error monitoring in the human anterior cingulate cortex. *Proceedings of the National Academy of Sciences*, **99**, 16354-16359.

TIEGES, Z., RIDDERINKHOF, K. R., SNEL, J., & KOK, A. (2004). Caffeine strengthens action monitoring: Evidence from the error-related negativity. *Cognitive Brain Research*, **21**, 87-93.

ULLSPERGER, M., BYLSMA, L. M., & BOTVINICK, M. M. (2005). The conflict adaptation effect: It's not just priming. *Cognitive, Affective, & Behavioral Neuroscience*, **5**, 467-472.

ULLSPERGER, M., & SZYMANOWSKI, F. (2004). ERP correlates of error relevance. In M. Ullsperger & M. Falkenstein (Eds.), *Errors, conflict and the brain: Current opinions on performance monitoring* (pp. 171-177). Leipzig: Max Planck Institute for Human Cognitive and Brain Science.

ULLSPERGER, M., & VON CRAMON, D. Y. (2001). Subprocesses of performance monitoring: A dissociation of error processing and response competition revealed by event-related fMRI and ERPs. *NeuroImage*, **14**, 1387-1401.

ULLSPERGER, M., & VON CRAMON, D. Y. (2004). Neuroimaging of performance monitoring: Error detection and beyond. *Cortex*, **40**, 593-604.

URSU, S., JONES, M., SHEAR, M. K., STENGER, V. A., & CARTER, C. S.

(2003). Overactive action monitoring in obsessive-compulsive disorder: Evidence from functional MRI. *Psychological Science*, **14**, 291-388.

VAN VEEN, V. (2006). *A neuroimaging approach to the relationship between attention and speed–accuracy tradeoff.* Unpublished doctoral dissertation, University of Pittsburgh, Pittsburgh, PA.

VAN VEEN, V., & CARTER, C. S. (2002a). The anterior cingulate as a conflict monitor: fMRI and ERP studies. *Physiology & Behavior*, **77**, 477-482.

VAN VEEN, V., & CARTER, C. S. (2002b). The timing of action-monitoring processes in the anterior cingulate cortex. *Journal of Cognitive Neuroscience*, **14**, 593-602.

VAN VEEN, V., & CARTER, C. S. (2005). Separating semantic conflict and response conflict in the Stroop task: A functional MRI study. *NeuroImage*, **27**, 497-504.

VAN VEEN, V., & CARTER, C. S. (2006). Conflict and cognitive control in the brain. *Current Directions in Psychological Science*, **15**, 237-240.

VAN VEEN, V., COHEN, J. D., BOTVINICK, M. M., STENGER, V. A., & CARTER, C. S. (2001). Anterior cingulate cortex, conflict monitoring, and levels of processing. *NeuroImage*, **14**, 1302-1308.

VAN VEEN, V., HOLROYD, C. B., COHEN, J. D., STENGER, V. A., & CARTER, C. S. (2004). Errors without conflict: Implications for performance monitoring theories of anterior cingulate cortex. *Brain & Cognition*, **56**, 267-276.

WEISSMAN, D. H., GIESBRECHT, B., SONG, A. W., MANGUN, G. R., & WOLDORFF, M. G. (2003). Conflict monitoring in the human anterior cingulate cortex during selective attention to global and local object features. *NeuroImage*, **19**, 1361-1368.

WEST, R. (2003). Neural correlates of cognitive control and conflict detection in the Stroop and digit-location tasks. *Neuropsychologia*, **41**, 1122-1135.

WEST, R. (2004). The effects of aging on controlled attention and conflict processing in the Stroop task. *Journal of Cognitive Neuroscience*, **16**, 103-113.

WEST, R., BOWRY, R., & MCCONVILLE, C. (2004). Sensitivity of medial frontal cortex to response and nonresponse conflict. *Psychophysiology*, **41**, 739-748.

WEST, R., & MOORE, K. (2005). Adjustments of cognitive control in younger and older adults. *Cortex*, **41**, 447-616.

WÜHR, P., & ANSORGE, U. (2005). Exploring trial-by-trial modulations of the Simon effect. *Quarterly Journal of Experimental Psychology*, **58A**, 705-731.

YEUNG, N., BOTVINICK, M. M., & COHEN, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review*, **111**, 931-959.

YEUNG, N., & COHEN, J. D. (2006). The impact of cognitive deficits on conflict monitoring: Predictable dissociations between the error-related negativity and N2. *Psychological Science*, **17**, 164-171.

ZIRNHELD, P. J., CARROLL, C. A., KIEFFABER, P. D., O'DONNELL, B. F., SHEKHAR, A., & HETRICK, W. P. (2004). Haloperidol impairs learning and error-related negativity in humans. *Journal of Cognitive Neuroscience*, **16**, 1098-1112.

NOTE

1. In the Stroop task, ACC activation appears to be reflected in a component referred to as the *N450*, which also peaks prior to response and has a scalp topography similar to that of the frontocentral N2 (West, 2003; West, Bowry, & McConville, 2004). We assume that the frontocentral N2 and the N450 in the different types of tasks are functionally similar components, reflecting the detection of conflict by the ACC.

Note that the frontocentral N2 and the N450 mentioned in the present article should not be confused with other instances of N2 and N4, which might reflect different psychological processes and the activation of different brain regions (see Pritchard, Shappell, & Brandt, 1991).

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