

Research Article

The Impact of Cognitive Deficits on Conflict Monitoring

Predictable Dissociations Between the Error-Related Negativity and N2

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ABSTRACT—Monitoring of ongoing processing plays a critical role in regulating cognitive function. Two event-related potential components, the error-related negativity (ERN) and N2, have been proposed to reflect this monitoring function. Specifically, it has been suggested that both components reflect the role of anterior cingulate cortex (ACC) in monitoring for the occurrence of response conflict. This view appears to be challenged by findings that alcohol consumption and lesions in ACC have dissociable effects on the ERN and N2. Using a computational model, the present research demonstrates that the conflict-monitoring theory can account for these dissociations in terms of the dissociable effects of alcohol and ACC lesions on processing of relevant stimulus information (which determines ERN amplitude) and processing of irrelevant, distracting information (which determines N2 amplitude). The simulation results suggest new interpretations of the cognitive deficits caused by alcohol consumption (impaired stimulus processing) and ACC lesions (impaired attentional control).

The ability to monitor ongoing behavior, and make appropriate adjustments when current goals are not being met, is critical to normal cognitive functioning. It has long been recognized that detection of overt errors may provide vital information in this regulative process (Rabbitt, 1966b; Reason, 1990). More recently, it has been proposed that detecting cross talk, or *conflict*, during processing may provide important additional information

(Botvinick, Braver, Carter, Barch, & Cohen, 2001; Carter et al., 1998). Specifically, detecting conflict during response selection (i.e., the simultaneous activation of incompatible actions) may provide early warning of conditions in which errors are likely and hence in which increased attention is required.

Event-related brain potential (ERP) findings suggest that anterior cingulate cortex (ACC) plays a critical role in these monitoring functions. First, ACC is the likely neural source of the error-related negativity (ERN), a medial-frontal potential that peaks within 100 ms of error commission in simple decision tasks (Falkenstein, Hohnsbein, Hoorman, & Blanke, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993). This finding suggests the involvement of ACC in error processing. Second, ACC is also the likely generator of the N2, a component observed in stimulus-locked averages on trials with correct responses. N2 amplitude is increased on high-conflict trials, for example, when incongruent target and distractor stimuli activate conflicting responses in the Stroop task and Eriksen flanker task (Kopp, Rist, & Mattler, 1996; Liotti, Woldorff, Perez, & Mayberg, 2000). These findings suggest the involvement of ACC in conflict monitoring (van Veen & Carter, 2002). Together, study of the ERN and study of the N2 promise to provide insight into the cognitive and neural mechanisms of performance monitoring.

Two recent studies have reported dissociations between the N2 and ERN. First, Ridderinkhof et al. (2002) found that alcohol consumption leads to a substantial reduction in ERN amplitude, but does not affect the N2. Second, Swick and Turken (2002) reported that a patient with a left-ACC lesion exhibited a substantially reduced ERN, but a greatly increased N2. These results have been interpreted as evidence that alcohol consumption and ACC lesions cause specific cognitive deficits, in which error monitoring (indexed by the ERN) is impaired but conflict monitoring (indexed by the N2) is spared. These findings

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therefore suggest that conflict and error monitoring may be at least partially separable functions, a conclusion that challenges our recent proposal that conflict monitoring can provide a reliable mechanism for error detection, and that the ERN and N2 reflect the operation of a common conflict-monitoring mechanism (Yeung, Botvinick, & Cohen, 2004). Instead, these results suggest that the ERN and N2 have separate neural generators (cf. Mathalon, Whitfield, & Ford, 2003).

In previous research, we have used a connectionist model of conflict monitoring to account for detailed properties of the ERN and N2 (Yeung et al., 2004). In the present research, we developed this model to address the challenging findings of dissociations between these components. Our aim was to investigate whether the conflict-monitoring theory of ACC function can provide principled explanations of these dissociations. In what follows, we outline our model of the ERN and N2 (for full implementation details, see Botvinick et al., 2001; Cohen, Servan-Schreiber, & McClelland, 1992; Yeung et al., 2004). We then show how it can account for the findings reported by Ridderinkhof et al. (2002) and Swick and Turken (2002) and, in so doing, provide a new account of the underlying cognitive deficits caused by alcohol consumption and ACC lesions.

CONFLICT-MONITORING THEORY OF THE ERN AND N2

Our model (Fig. 1a) simulates a version of the Eriksen flanker task (Eriksen & Eriksen, 1974) in which subjects are instructed to respond to a target stimulus—press a left button if the letter *H* is presented and a right button if the letter *S* is presented—and to ignore irrelevant distractors that on some trials cue the same response as the target (congruent trials, e.g., *HHH*) and on others cue the opposite response (incongruent trials, e.g., *SHS*). In the model, stimuli are represented as patterns of activity across the stimulus layer. Activity flows from this layer through its connections to the response layer. Input from the attention layer boosts processing of target stimuli relative to the flankers, ensuring that the model usually produces the correct response, even on incongruent trials. However, noise in processing means that errors sometimes occur.

The conflict-monitoring mechanism is sensitive to conflict in the response layer. Conflict is formalized as the product of the activation levels of the competing response units scaled by the strength of the inhibitory connection between them. This product measure captures the concept of conflict in a simple way. For example, on incongruent trials, when target and flanking stimuli activate different responses, the product of response activation levels is large, and conflict is high. In contrast, on congruent trials, when target and flanking stimuli activate the same (correct) response and incorrect response activity is low or zero, the product of activation levels is low or zero, and there is little or no conflict.

The model simulates the ERN as a difference in conflict between correct and error trials that peaks shortly after the

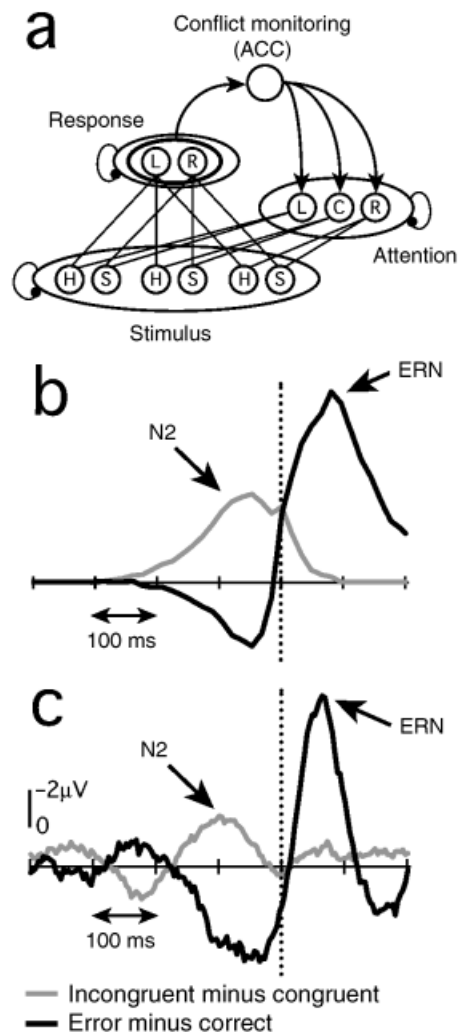


Fig. 1. Schematic illustration of the model of conflict monitoring in the flanker task (a), the model's predictions for the relative timing of the error-related negativity (ERN) and N2 (b), and empirically observed timing of the ERN and N2 at electrode location FCz (c; Yeung, Botvinick, & Cohen, 2004). In (a), the input layer has a unit for each possible stimulus: the letters *H* and *S* in the left (L), center (C), and right (R) locations. The response layer has a unit for each possible response. The attention layer has a unit for each of the three locations. The conflict-monitoring unit, corresponding to anterior cingulate cortex (ACC), is sensitive to coactivation of response units. The data in (b) and (c) are shown time-locked to the response, the latency of which is indicated with a dotted line.

response (Fig. 1b, black line). The conflict account of the ERN is based on the observation that subjects tend to “correct” their mistakes—by making the response they should have made—shortly after the initial error (Rabbitt, 1966a, 1966b). Error corrections reflect continued processing of the stimulus after the incorrect response has been produced (Rabbitt & Vyas, 1981). In the model, error-correcting activity from continued stimulus processing leads to postresponse conflict between the initial error (generated by noise coupled with early processing of the flankers) and the correcting response. In contrast, there is little conflict following correct responses because continued stimulus

processing simply reinforces the (correct) decision. Thus, greater conflict follows errors than correct responses, and the timing of this posterror conflict closely replicates the timing of the ERN.

Figure 1b also plots the simulated N2 (gray line), which is the difference in response conflict between congruent and incongruent trials with correct responses. On these trials, the correct response begins to dominate competition in the response layer around the time of response execution, and continued stimulus processing after the response serves only to reinforce this dominance. Conflict is therefore observed primarily prior to the response, before the increasingly activated correct response completely suppresses incorrect response activity. These simulation results predict that the conflict-related N2 should peak prior to the response, a prediction borne out in empirical studies (Fig. 1c).

RESEARCH OVERVIEW

In the present research, we applied this model to the findings of ERN-N2 dissociations. In each simulation, we first parameterized the model to fit the reported behavioral findings, then assessed the impact of these parameters on simulated ERPs (i.e., we did not directly fit the empirical ERP data). Although we propose that the ERN and N2 both reflect conflict monitoring, we predict that these two components should be sensitive to different aspects of task processing. In particular, ERN amplitude should reflect primarily whether subjects attend to relevant stimulus information (which determines whether error-correcting responses will be produced), whereas N2 amplitude should reflect primarily whether subjects process irrelevant stimulus information (which determines the level of incorrect response activity on incongruent trials). In the present research, we investigated whether such dissociations can provide new accounts of the cognitive impairments caused by alcohol consumption (Ridderinkhof et al., 2002) and ACC lesions (Swick & Turken, 2002).

SIMULATION 1: ALCOHOL AND THE ERN

Ridderinkhof et al. (2002) had subjects perform the flanker task after consuming a high dose of alcohol, a low dose, or a placebo drink. Subjects were trained to achieve constant levels of flanker interference and constant error rates across alcohol conditions, to prevent confounds with these variables. Thus, the primary behavioral effect of alcohol was overall slowing of response time (RT; Fig. 2, upper left panel). (For simplicity, we focus here on the placebo and high-dose conditions only.) The ERP results indicated that alcohol consumption substantially reduced the ERN but did not affect the N2, suggesting that alcohol had a relatively specific impact on error processing. However, alcohol consumption also led to reduced amplitude of the P3 (K.R. Ridderinkhof, personal communication, April 17, 2005), a component thought to reflect stimulus-categorization processes (Donchin & Coles, 1988).

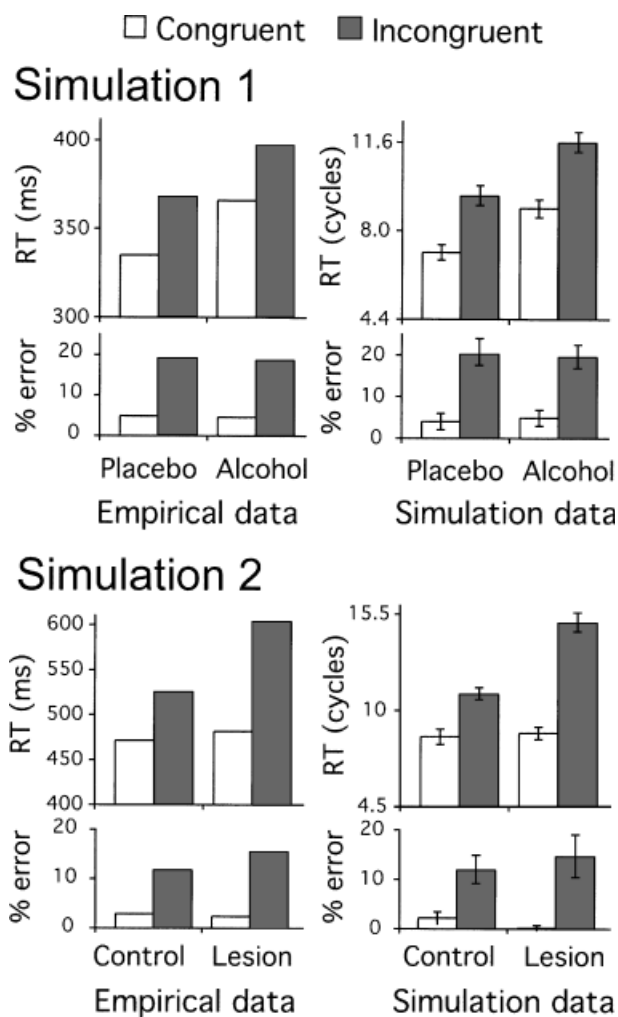


Fig. 2. Empirical (left) and simulated (right) behavioral results for the placebo and alcohol conditions of Simulation 1 (upper panel) and the control and lesion conditions of Simulation 2 (lower panel). RT = response time. The upper left panel plots empirical data from the study by Ridderinkhof et al. (2002); the lower left panel plots empirical data from the study by Swick and Turken (2002).

One interpretation of these findings is that the ERN reflects an error signal conveyed by the mesencephalic dopamine system, and that alcohol consumption specifically disrupts this system (Holroyd & Yeung, 2003). In contrast, if we assume that alcohol directly impairs a conflict-monitoring system indexed by the ERN, then we would expect alcohol to have a corresponding impact on the N2. Thus, these findings initially seem difficult for our theory to explain. However, a detailed analysis suggests that the primary effect of alcohol in the flanker task might be impaired stimulus processing, rather than a specific performance-monitoring deficit. The possibility of impaired stimulus processing is suggested by the fact that Ridderinkhof et al. found that alcohol consumption caused increased RTs and reduced P3 amplitude. This possibility is also consistent with previous reports that alcohol impairs stimulus categorization (Colrain et al., 1993; Marinkovic, Halgren, & Maltzman, 2001; Tzambazis & Stough, 2000) and attentional control of stimulus processing

(Curtin & Fairchild, 2003; Fillmore, Vogel-Sprott, & Gavrilescu, 1999; Koelega, 1995).

On the basis of these observations, we simulated alcohol effects in terms of two specific deficits in stimulus processing: reduced quality of stimulus representation and reduced attentional focus. Together these changes tend to increase RTs, an effect that is consistent with the notion that the depressant effects of alcohol produce general RT slowing. These changes also tend to increase error rates in the model. However, as we have noted, Ridderinkhof et al. trained their subjects to produce equivalent error rates across conditions. We simulated this behavioral control as leading subjects in the alcohol condition to adopt more cautious response strategies (i.e., increased response thresholds) in order to avoid increased error rates.

Method

Simulation parameters for the placebo condition were taken from our previous research (Yeung et al., 2004), except that gain on the center attention unit was increased (from 1.0 to 1.4) in order to match the empirical error rates. Parameter values for the alcohol simulation were then chosen to fit behavioral data from the high-dose condition. The stimulus-processing deficit was simulated by reducing external inputs to the stimulus layer (from 0.15 to 0.11). The attentional deficit was simulated by reducing external input to the center attention unit (by a factor of 0.8). The change in response threshold was simulated by increasing the value of response-unit activation that was required for a response to be produced (from 0.18 to 0.20). These three parameter changes were used to simulate alcohol effects on eight empirical data points: four behavioral results (RT and error rate on congruent and incongruent trials) and four ERP results (N2 amplitude on congruent and incongruent trials, ERN amplitude following correct and error responses). The model was run for 10 simulations of 1,000 trials in each condition. The simulated ERN was quantified as the average difference in postresponse conflict between trials with correct and error responses. The simulated N2 was quantified as the difference in peak conflict between congruent and incongruent trials with correct responses.

Results

Overall RTs were increased in the alcohol simulation relative to the placebo condition, but there were no corresponding increases in overall error rate or amount of flanker interference (Fig. 2, upper right panel). The absence of between-condition differences in flanker interference reflects the fact that whereas attentional impairments increase flanker interference, stimulus degradation has the opposite and therefore offsetting effect. That is, stimulus degradation differentially impairs processing of flanker stimuli—which unlike the target do not receive top-down attentional support—and hence reduces flanker interference. The model therefore accurately simulates the behavioral data reported by Ridderinkhof et al. (2002). Of interest, then, are the simulated ERP components in the model (Fig. 3, top row of

results for Simulation 1), which are defined in terms of patterns of response conflict on correct and error trials (Fig. 3, second row of results for Simulation 1). Critically, the model replicated the pattern of ERP results observed empirically: The simulated ERN was substantially reduced in the alcohol simulation relative to the placebo control, $F(1, 18) = 45.6, p < .01, \eta_p^2 = .72$, whereas the N2 was unaffected by this manipulation, $F(1, 18) = 1.98, p > .15, \eta_p^2 = .10$.

As we have described, the conflict-monitoring theory explains the ERN in terms of conflict between an initial incorrect response and error-correcting activity produced by continued stimulus processing. The simulated ERN therefore depends critically on the degree to which the correct response becomes activated following errors. As shown in Figure 3 (third row of results for Simulation 1, black lines), at the peak latency of the simulated ERN (5 cycles after response), error-correcting activity was approximately halved in the alcohol condition (0.018) relative to the placebo condition (0.035), accounting for the approximate halving of ERN amplitude in the alcohol simulation. Error-correcting activity was reduced in the alcohol condition because deficits in stimulus representation and attentional input lead to impaired processing of the target stimulus (on which error-correcting activity depends).

In contrast to the ERN, the simulated N2 did not differ in amplitude between conditions. In the model, N2 amplitude depends critically on activation levels of the conflicting, incorrect response, which in turn depend on the amount of processing of irrelevant stimulus information. Incorrect response activity (Fig. 3, bottom row of results for Simulation 1 panels, gray lines) differed little between conditions because, as described, alcohol-induced deficits in attentional focus and stimulus representation had offsetting effects on flanker processing. As a consequence, the simulated N2 did not differ between conditions.

In explaining the simulation results, we have focused on the impact of deficits in stimulus representation and attentional input because these parameters directly influence the dynamics of response selection, and hence affect the dynamics of response conflict. In contrast, variations in response threshold have relatively little impact on patterns of response conflict, because threshold determines the point at which a response is generated, but does not directly affect activation dynamics. Thus, increasing the response threshold in the alcohol simulation was important in simulating the empirical behavioral data (in particular, the equivalent error rates across conditions), but had relatively little effect on the simulated ERN-N2 dissociation. Indeed, in other simulations, we have observed corresponding dissociations while keeping response threshold constant.

SIMULATION 2: ACC LESIONS AND THE ERN

Swick and Turken (2002) compared a patient (RN) with a focal left-ACC lesion against age-matched control subjects in two versions of the Stroop task (which, for simplicity, we average

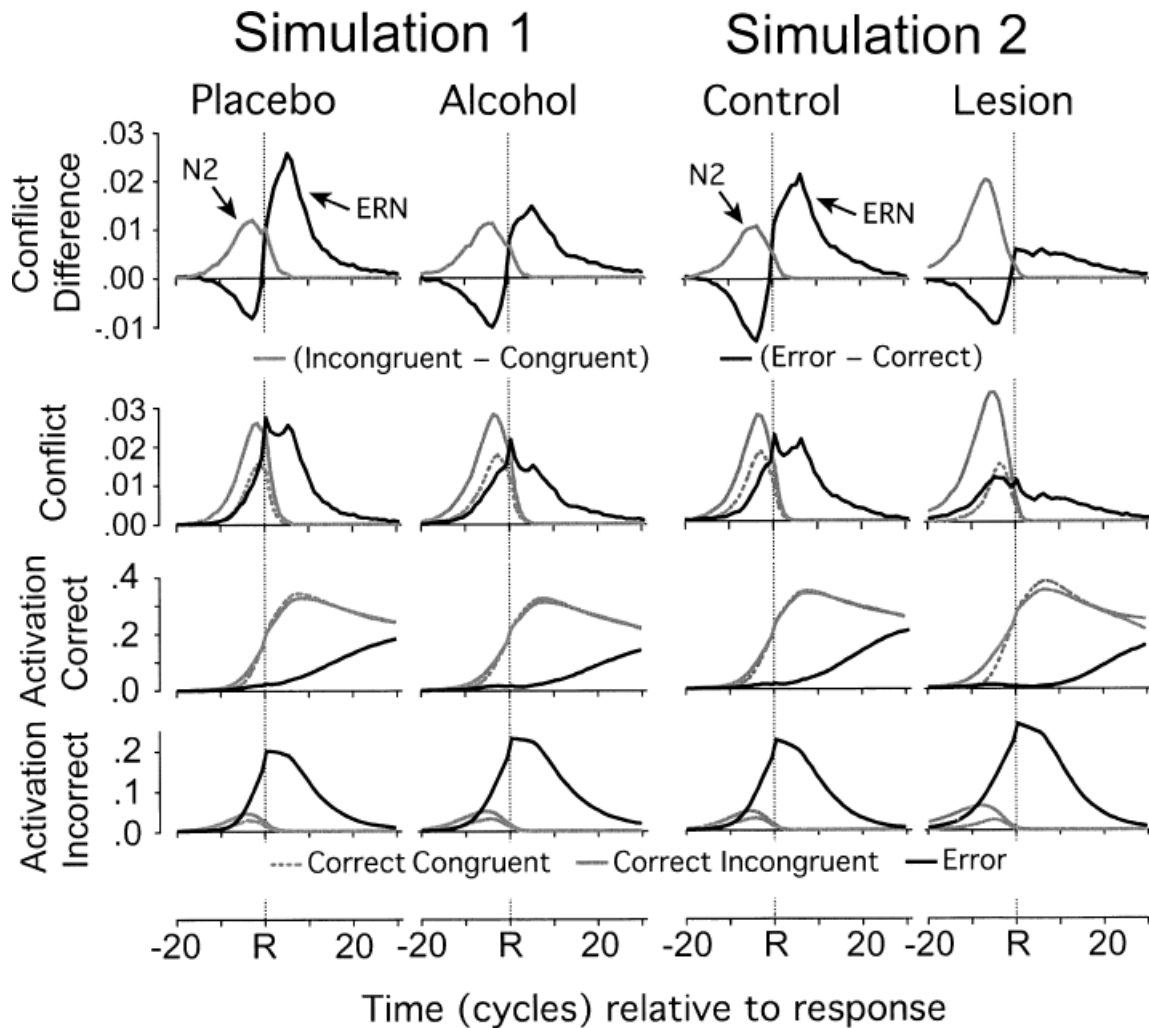


Fig. 3. Simulated effects of alcohol consumption and anterior cingulate cortex lesions on the error-related negativity (ERN) and N2. The results are shown separately for the placebo and alcohol conditions of Simulation 1 (left panel) and the control and lesion conditions of Simulation 2 (right panel). Data are shown time-locked to the response (R, at time zero). The top panels plot the conflict difference between trials with correct and error responses (ERN) and between congruent and incongruent trials with correct responses (N2). The upper middle panels show the raw conflict scores for each condition, calculated as the product of the activation levels of the competing response units. The activation levels of the correct and incorrect response units are shown in the lower middle and lower panels, respectively (note that different vertical scales are used in these panels).

across here). As with the flanker task, a critical feature of the Stroop task is that the stimulus contains irrelevant information that is sometimes associated with the correct response (congruent trials) and sometimes associated with the incorrect response (incongruent trials). Behaviorally, RN was particularly sensitive to this irrelevant stimulus information, showing markedly increased RTs on incongruent trials (Fig. 2, lower left panel). Concurrently recorded ERP data indicated a dissociation between the ERN and N2, with RN showing a greatly reduced ERN but substantially increased N2 relative to the control subjects.

Swick and Turken (2002) interpreted their results as being “at odds with the contention that the exact same area of dorsal ACC mediates both conflict detection and error monitoring”

(p. 16358). However, a detailed simulation of these findings suggests an alternative interpretation. Specifically, we propose that the primary deficit caused by RN’s lesion is a disruption of the recruitment of attentional control by the conflict-monitoring system in ACC. In our model, recruitment of attention occurs via a conflict-control loop: Detection of conflict on one trial leads to increased attentional focus on subsequent trials (Botvinick et al., 2001; Cohen, Botvinick, & Carter, 2000). In the present simulation, we investigated whether our theory might explain Swick and Turken’s results in terms of a damaged conflict-control loop. That is, we speculate that RN has a relatively spared ability to detect conflict—an ability supported by the intact right ACC and spared portions of left ACC—but cannot use this conflict signal effectively to regulate attention.

Method

Parameter values for the control condition were taken from previous research except that we increased gain on the center attention unit (to 1.3) and increased response threshold (to 0.21) to match the low error rates observed empirically in the Stroop task. We simulated the effects of RN's lesion by varying two parameters. The first parameter, α , governs the degree to which detection of conflict leads to increases in attentional control ($\alpha_{\text{control}} = 4.41$, $\alpha_{\text{lesion}} = 1.10$). In effect, the lesioned model behaves as if there is less conflict than there actually is, and hence sets attentional control at a reduced level. The second parameter, response threshold, was increased in the lesion simulation (to 0.25) in order to match the overall error rate across conditions (the error rates of Swick and Turken's, 2002, patient were roughly equal to those of control subjects). As in Simulation 1, variability in response threshold was used to fit the behavioral data, but had relatively little effect on response conflict. Indeed, in other simulations keeping threshold constant, we have observed simulated ERP results similar to those we describe here. Thus, in what follows, we focus on the extent to which the simulated deficit in the recruitment of attentional control (α) can account for the effects of ACC lesions on behavioral and ERP measures. The simulation results reported here are based on 10 simulations of 1,000 trials in each condition (control and lesion).

Results

The simulated behavioral results (Fig. 2, lower right panel) replicate Swick and Turken's (2002) finding that interference was increased in the lesion condition relative to the control condition. This increase in interference is a straightforward consequence of reduced recruitment of attentional input, which results in poor performance when irrelevant stimulus information cues the incorrect response. Of interest, then, are the simulated ERPs in the model, which were again defined in terms of patterns of response conflict on correct and error trials (Fig. 3, second row for Simulation 2). Critically, the simulated ERP data (Fig. 3, top row for Simulation 2) capture the pattern of results observed by Swick and Turken: The simulated ERN was greatly reduced in the lesion condition relative to the control condition, $F(1, 18) = 108.6, p < .01, \eta_p^2 = .86$, whereas the simulated N2 was greatly increased, $F(1, 18) = 130.4, p < .01, \eta_p^2 = .88$.

The attentional deficit therefore has opposing effects on the simulated ERN and N2, even though both components reflect conflict monitoring. This dissociation can be understood in terms of the complementary effects that attentional deficits have on processing of target and flanking stimuli. As described earlier, factors that impair target processing tend to reduce ERN amplitude because they reduce the strength of postresponse stimulus processing and corresponding error-correcting activity. Meanwhile, factors that increase processing of flanking stimuli tend to increase N2 amplitude because they increase interference from this irrelevant information on incongruent trials.

Critically, a deficit in attentional focus produces both of these effects (Fig. 3, third and fourth rows for Simulation 2), and hence has dissociable effects on the simulated ERN and N2. These simulation results therefore suggest a new account of Swick and Turken's (2002) findings in terms of a lesion-induced deficit in attentional focus.

DISCUSSION

The present research provides a new account of the impact of alcohol consumption and ACC lesions on cognitive functioning as reflected in behavior and ERP recordings: We simulated the effects of alcohol consumption in terms of impaired stimulus processing and simulated the effects of an ACC lesion in terms of a deficit in conflict-based adjustments in attention. In each case, our account of the empirical findings contrasts with earlier explanations of the results in terms of specific error-monitoring deficits. The simulation results demonstrate that the empirical results can be explained by the theory that the ERN, like the N2, reflects the involvement of ACC in conflict monitoring.

An important implication of the simulation results is that, although our theory explains both the ERN and the N2 in terms of conflict monitoring (Yeung et al., 2004), it does not predict that the amplitude of these components must necessarily covary. Instead, dissociations are possible because the two components are sensitive to different aspects of task processing. Specifically, ERN amplitude depends critically on processing of target stimulus information (which underlies the ability to produce error-correcting responses), whereas N2 amplitude depends primarily on processing of irrelevant stimulus information (which determines the level of incorrect response activation). Thus, factors that differentially affect processing of target and distracting stimulus information should have differential effects on the ERN and N2. In Simulation 2, for example, lesion-induced attentional deficits led to impaired target processing (hence a reduced ERN) while causing increased processing of irrelevant stimulus information (hence an increased N2).

The results of the two simulations suggest new interpretations of the cognitive deficits caused by alcohol consumption and ACC lesions. In each case, we argue that the primary deficit does not lie within the monitoring system itself. Instead, our findings suggest that the primary effect of alcohol consumption in the study by Ridderinkhof et al. (2002) was impaired stimulus processing, whereas the primary deficit caused by the ACC lesion in Swick and Turken's (2002) study was in conflict-based recruitment of attentional control. Given that we are offering these specific interpretations of our simulation results, it is important to emphasize that we did not arrive at these hypotheses in an arbitrary manner. That is, it is not the case that our model is sufficiently underconstrained that it could account for the data in multiple ways (i.e., using many different types of parameter changes) that would support alternative accounts of the underlying cognitive deficits. To the contrary, target features

of the empirical data, together with the structure of our computational model, tightly constrain the possible hypotheses that can be considered. For example, a critical constraint in the data of Ridderinkhof et al. is that alcohol consumption increased RTs without increasing behavioral flanker interference. In our model, almost all manipulations that increase RTs also tend to increase flanker interference. In fact, to our knowledge, the only manipulation that does not have this property is the manipulation we use—stimulus degradation—which increases RTs but does not increase interference because both target and flankers suffer from degradation effects. We feel that something more interesting than coincidence is at work here, when the sole manipulation that can account for the behavioral data leads directly to an account of the observed ERP effects.

In suggesting new interpretations of the cognitive deficits caused by alcohol consumption and ACC lesions, the present research leads to testable predictions that could form the basis for future research. First, we propose that a deficit in stimulus processing is the primary cognitive impairment caused by alcohol consumption. One could test this hypothesis using a modified version of the design of Ridderinkhof et al. (2002). Specifically, the stimuli presented in the placebo condition could be degraded such that performance in this condition matches performance in the alcohol condition (cf. Holroyd & Yeung, 2003). If alcohol causes a deficit specific to error monitoring, as Ridderinkhof et al. (2002) suggested, then ERN amplitude should continue to be affected by alcohol consumption. In contrast, our theory predicts that if performance is matched in this way, then the ERN and N2 should be similarly matched across conditions. Along similar lines, one could induce an attentional “deficit” in control subjects—for example, by presenting additional distracting stimuli—in order to match RN’s behavioral deficit. If the primary effect of the ACC lesion is impaired error monitoring, then ERN amplitude should continue to be reduced in RN relative to these control subjects. In contrast, we predict that there should be no difference in the ERN and N2 between this patient and performance-matched control subjects.

In this way, the present research suggests important avenues for future studies. More generally, this research demonstrates the value of computational modeling in investigating implications of theories, and interpretations of empirical data, that may be subtle and nonintuitive: Without a model, it is not obvious how the conflict-monitoring theory can account for dissociations between the ERN and N2. Using our model, we have provided a formal demonstration of how such dissociations might arise and, in so doing, have provided new insight into the impact of alcohol consumption and brain lesions on monitoring functions that are critical to normal cognitive functioning.

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