Published in final edited form as:
*J Cogn Neurosci*. 2011 September ; 23(9): 2211–2221. doi:10.1162/jocn.2010.21595.

# Integrating Conflict Detection and Attentional Control Mechanisms

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## Abstract

Human behavior involves monitoring and adjusting performance to meet established goals. Performance-monitoring systems that act by detecting conflict in stimulus and response processing have been hypothesized to influence cortical control systems to adjust and improve performance. Here we used fMRI to investigate the neural mechanisms of conflict monitoring and resolution during voluntary spatial attention. We tested the hypothesis that the ACC would be sensitive to conflict during attentional orienting and influence activity in the frontoparietal attentional control network that selectively modulates visual information processing. We found that activity in ACC increased monotonically with increasing attentional conflict. This increased conflict detection activity was correlated with both increased activity in the attentional control network and improved speed and accuracy from one trial to the next. These results establish a long hypothesized interaction between conflict detection systems and neural systems supporting voluntary control of visual attention.

## INTRODUCTION

Human performance is supported by two major cognitive functions: a monitoring process that assesses performance success and a control process that adjusts behavior to achieve optimal performance (Gratton, Coles, & Donchin, 1992; Schneider & Shiffrin, 1977). Neuroimaging and electrophysiological studies have implicated the ACC as a brain region involved with monitoring performance (Emeric et al., 2008; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Carter et al., 1998), particularly via its sensitivity to conflict in stimulus and response processing (Kerns, 2006; Botvinick, Cohen, & Carter, 2004; Botvinick, Braver, Barch, Carter, & Cohen, 2001; Carter et al., 2000; MacDonald, Cohen, Stenger, & Carter, 2000). According to the conflict-control model (Botvinick et al., 2001), after conflict detection by ACC, a cortical control system in the dorsolateral pFC (dlPFC) is engaged to bring future performance in line with established goals.

The conflict-control model is largely based on evidence from the case of conflict involving prepotent response inhibition, such arises in the Stroop color–word interference task (Stroop, 1935). This popular task generates conflict by asking subjects to name the color of the ink of a printed word when the word meaning itself may specify either the same color as the ink or a conflicting color. For example, Kerns et al. (2004) demonstrated that increased ACC activity on high-conflict trials in the Stroop task (e.g., the word RED in green ink) was correlated with increased activity in dlPFC on trials immediately after high-conflict trials.



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Although the conflict-control model specifies a neural mechanism involving ACC and dlPFC, the model has broader theoretical implications for the neuroscience of control.

Perhaps the most widely studied domain of top–down control involves selective attention. Converging evidence from neuropsychological (Corbetta, 1998; Posner, Walker, Friedrich, & Rafal, 1984; Mesulam, 1981), electrophysiological (Reynolds & Chelazzi, 2004; Colby & Goldberg, 1999), and brain imaging (Corbetta, Kincade, Ollinger, McAvoy, & Shulman, 2000; Hopfinger, Buonocore, & Mangun, 2000; Gitelman et al., 1999; Kastner, Pinsk, De Weerd, Desimone, & Ungerleider, 1999) studies has shown that the voluntary control of selective attention is supported by a network of brain regions that includes dorsal frontal areas surrounding the FEFs and posterior parietal areas in and around the intraparietal sulcus. An interaction between ACC conflict-monitoring system and this frontoparietal attentional control network has been hypothesized (Casey et al., 2000), but little direct evidence has been provided that could demonstrate a direct link between these systems in performance monitoring and control of attention.

We investigated the hypothesis that during attentional conflict, ACC interacts with the frontoparietal attentional control system to modulate selective attention. The working model was that conflict in one trial (*N*) should result in adjustments in top–down attentional control to reduce conflict in a subsequent trial (*N* + 1). Such an interaction would be observed as activity in ACC for trial *N* where conflict was induced and then as changes in activity in the frontoparietal attentional control network as well as in behavioral performance for trial *N* + 1.

To test our hypothesis, subjects were presented with attention directing cues that instructed them where to covertly direct attention on each trial to discriminate the orientation of the cued-location target; stimuli in the opposite visual field were to be ignored (see Figure 1). Conflict in attentional orienting was induced by varying the discriminability of the cues unpredictably across trials (see Figure 1B). We measured performance and brain activity (using fMRI) as subjects performed the attention task. The results confirmed our hypotheses: Conflict in attentional orienting on one trial affected both performance and brain activity on the next trial. Importantly, increased ACC activity on conflict trials was followed by both increased activity in the frontoparietal attention network and improved target discrimination on the next trial.

## METHODS

### Subjects

Twenty right-handed volunteers (aged 20–31 years; 10 women) were paid $10/hr to participate in the study. Three subjects were excluded from functional analysis because of excessive head movement in the scanner (>0.5 voxel movement in any direction). Participants were informed of all procedures and provided written consent as specified in the protocol approved by the institutional review board at the University of California, Davis.

### Stimuli

Attend cues (to which subjects shifted attention) consisted of two vertical lines, one on either side of a small fixation square. The distance from each line to fixation was 0.9°; the length of each line ranged from 1.1° to 1.7°, depending on the type of cue. Neutral cues (to which subjects did not shift attention) were similar to attend cues, except the vertical lines were of equal length (1.1°) and had short (0.3°) horizontal lines on the top and bottom of each vertical line to make them easily and immediately distinguishable from attend cues without being excessively different than attend cues as a visual stimulus (Figure 2). Targets were circular Gabor gratings (1.5° diameter, located bilaterally 6.2° from fixation in the

upper left and right visual fields), each of which could be horizontal or vertical in orientation.

## Procedure

Subjects performed 16 blocks of 45 trials each, pseudo-randomized using an m-sequence (Buracas & Boynton, 2002). Each trial began with a briefly presented cue (100-msec duration) that instructed subjects to covertly attend (without removing eyes from fixation square) to the upper right or upper left visual field. For the attend left cues, the left line was longer than the right; for the attend right cues, the right line was longer than the left; and for the neutral cues to which subjects did not shift attention, the lines were of equal length. The amount of conflict generated by the attend cues was manipulated systematically by varying the difference in length of the two lines. Accordingly, there were three categories of attend cues: low, medium, and high conflict-generating cues, determined by the time required for subjects to judge which of the pair of lines was longer (tested empirically in previous behavioral experiments). On half of the attend trials, after a 1400-msec blank postcue interval, bilateral targets briefly appeared (200-msec duration), followed immediately by pattern masks (300-msec duration). Subjects were required to respond by pressing one of two buttons with their right hand to indicate whether the target in the attended field had been horizontal or vertical (attended and unattended targets could either be both of the same orientation or each be of different orientations). On the other half of the attend trials and for all neutral cue trials, no targets were presented and no response was required. The m-sequence was generated using nine trial types: low, medium, and high-conflict cues with targets, low, medium, and high-conflict cues without targets, neutral cues, and two no-stimulus conditions. Thus, each trial type comprised 11% of the total trials in the experiment. Subjects were instructed to maintain fixation at all times, verified by infrared eye-tracking using an eye-tracker (ASL model 504), and to shift attention immediately after determining the appropriate direction as instructed by the cue.

## Training

Each subject was required to complete a 45-min training session before participation in the fMRI experiment. The first part of this training session consisted of eight blocks of trials that were similar to the fMRI task, except that targets were presented unilaterally in an 80% valid to 20% invalid ratio (in 80% of the trials the target appeared in the cued hemifield; in the other 20%, the target appeared in the uncued hemifield). The point of these blocks of trials was for subjects to practice shifting their attention and for the experimenters to obtain behavioral measures (RT and accuracy), which would indicate that subjects were indeed shifting attention to the cued hemifield. The second part of the training session consisted of four blocks of trials with bilateral targets, identical to the task subjects would later perform for the fMRI experiment. The point of these blocks was to ensure that subjects could perform the task with reasonable accuracy (>80%) and to minimize any learning effects associated with being introduced to the task in the scanner.

## Imaging Methods

Magnetic resonance images were acquired at the UC Davis Imaging Research Center, using a GE Healthcare (Waukesha, WI) 1.5-T Signa neuro-optimized (NV/i) MRI system running LX 9.1 operating system software. After a T1-weighted sagittal localizer, T2-weighted fast spin echo (FSE) images were acquired in an axial oblique orientation aligned with the AC/PC line and covering the supra-tentorial brain volume. This sequence was followed by a series of fMRI scans using single-shot EPI, with slices acquired at the same locations and orientations as used for the T2 FSE images. After completion of all fMRI scans, three-dimensional high spatial resolution T1-weighted images were acquired for structural detail. The T2-weighted images provided a convenient validation and check of anatomical

structures shown at relatively low resolution in the EPI scans. The three-dimensional T1 images provided near-isotropic structural information to BrainVoyager QX for high-resolution reslicing of the data (e.g., axial oblique slices to sagittal or to coronal; see Figure 3) and optimal overlaying of the activation maps computed from the fMRI time series.

The scan parameters for the localizer sagittal T1-weighted spin-echo images were as follows: repetition time (TR) = 500 msec, echo time (TE) = 9 msec, flip angle (FA) = 90°, field of view (FOV) = 24.0 × 24.0 cm, slice thickness = 3.0 mm, slice spacing = 0.5 mm, matrix = 256 (frequency) × 224 (phase), number of slices = 19, number of averages (NEX) = 1, bandwidth = 15.625 kHz, inferior SAT pulse on, Autoshim on, voxel size = 0.94 × 1.07 × 3.0 mm, scan time = 2 min. The scan parameters for the axial-oblique T2-weighted FSE images were as follows: TR = 3000 msec, TE = 100 msec, FA = 90°, FOV = 22.0 × 22.0 cm, slice thickness = 6.0 mm, slice spacing = 0.0 mm (interleaved in two acquisitions), matrix = 256 (frequency) × 256 (phase), number of slices = 16, number of averages (NEX) = 2, bandwidth = 31.25 kHz, echo train length = 24, flow compensation = slice direction, inferior SAT pulse on, fat saturation on, Autoshim on, phase correct on, voxel size = 0.86 × 0.86 × 6.0 mm, scan time = 2 min 36 sec. The scan parameters for the axial oblique three-dimensional T2*-weighted gradient-echo, echo-planar images were as follows: TR = 1500 msec, TE = 40 msec, FA = 90°, FOV = 22.0 × 22.0 cm, slice thickness = 6.0 mm, slice spacing = 0.0 mm (interleaved within one acquisition), matrix = 64 (frequency) × 64 (phase), number of slices = 16, number of averages (NEX) = 1, bandwidth = 62.5 kHz, echo train length = 64 (single shot), voxel size = 3.44 × 3.44 × 6.0 mm, number of temporal phases = 207, scan time = 5 min 10 sec. Custom image reconstruction included automated suppression of ghost artifacts (Buonocore & Gao, 1997). The scan parameters for the three-dimensional axial-oblique T1-weighted inversion-recovery fast RF-spoiled gradient-echo (IR-FSPGR) images were as follows: TR = 9.1 msec, TE = 2.0 msec, TI = 400 msec, FA = 15°, FOV = 22.0 × 22.0 cm, slice thickness = 1.2 mm, matrix = 256 (frequency) × 256 (phase), number of slices = 128, number of averages (NEX) = 1.0, bandwidth = 15.625 kHz, voxel size = 0.86 × 0.86 × 1.2 mm, scan time = 8 min 36 sec.

Image processing was performed using BrainVoyager QX, Version 1.3. Functional images were preprocessed to correct for differences in slice acquisition order and head motion. Each subject's anatomical scans were co-registered with their functional images and spatially normalized to stereotaxic space using Talairach coordinates. Resulting parameters were used to spatially normalize the functional images, which were smoothed spatially with an 8-mm FWHM Gaussian kernel.

## Data Analysis

Individual subjects' hemodynamic responses (HDRs) to the attend cues were isolated by convolving a vector of onset times of the cues and targets with a synthetic HDR function, emphasizing transient activity in response to the events (Hopfinger et al., 2000; Friston, Josephs, Rees, & Turner, 1998; Friston, Frith, Turner, & Frackowiak, 1995). The general linear model was used to estimate the relative contributions of each condition to the overall time course of neural activity for each individual subject. A random effects analysis was performed on spatially smoothed data from each of the 17 subjects to investigate generalizable effects across subjects.

Cortical activity masks were created from the event-related responses of cues versus baseline activity (no stimulus condition), with statistical significance evaluated with a threshold of $p < .0000001$, uncorrected, and a spatial extent threshold of 50 contiguous voxels, parameters that yielded a false discovery rate of <0.001 (Benjamini & Hochberg, 1995). A general linear model was run on these cortical masks, and contrasts of interest were then examined by comparing (subtracting) sets of conditions. Areas exhibiting significant

activity ($p < .005$, 50 contiguous voxels, appropriate parameters determined from "Cluster Threshold Estimator" plug-in, Fabrizio Esposito, BVQX) from the subtractions were then further examined as ROIs, for which beta values for each individual condition were obtained by running a general linear model on each ROI. For contrasts of interest, *t* tests were performed to compare sets of conditions. All cue-related data presented in this manuscript were generated using cue-only trials to avoid signal contamination related to target processing. Similarly, trial-to-trial analyses were performed using exclusively cue-only trials in trial *N* to avoid signal contamination from target processing and motor response between trial *N* and trial $N + 1$.

HDRs were obtained by event-related averaging the activity within particular ROIs. Difference HDRs (d-HDRs) were created by subtracting the averaged HDR waveform for one condition in a given ROI from the HDR waveform for another condition in the same ROI at each time point, resulting in a waveform representing the difference between two conditions (or sets of conditions) over time. For all HDR graphs, stimulus (cue) onset occurred at time = 3, with baseline signal change equal to the average of the three time points leading up to and including stimulus onset (mean of Time Points 1, 2, and 3). For statistical comparison of the trial $N + 1$ neurophysiological adjustment in frontoparietal regions, HDR measures at Time Points 6 and 7 TRs after the trial *N* cue were averaged across right and left regions. This averaged activity after high conflict was then compared with activity after low-conflict conditions using a *t* test.

## RESULTS

### Behavior Shows Reduced Accuracy on High-conflict Trials

Overall, subjects ($n = 17$) performed well on the task, responding accurately to the cued target on 94% of trials. Subjects were slightly more accurate for targets preceded by a low-conflict cue than they were for targets preceded by a high-conflict cue (95.3% vs. 92.8%), $t(16) = 2.21$, $p = .042$. Pilot data, in which subjects responded only to the cue stimuli, indicated that subjects misjudged the high-conflict cues on approximately 8% of trials and low-conflict cues on approximately 2% of trials. Because the bilateral targets were identical to each other on half the trials and opposite on half the trials, the 6% difference in improper target selection ought to result in a 3% error difference in judging targets, similar to the observed difference of 2.5%. RTs did not differ for targets as a function of conflict engendered by the cue (all *p* values > .2; e.g., low vs. high: 911.4 vs. 925.9 msec), $t(16) = 1.09$, $p = .29$. In this fMRI study, the subjects were not required to respond to targets in the uncued visual field, and therefore there is no behavioral correlate of selective visual attention per se. However, to ensure that the task parameters were suitable to induce the subjects to covertly orient selective spatial attention to the different cue types, we conducted a separate behavioral test using identical cues and timing. In this test, cues predicted the likely location of a unilateral target with a .80 probability, and responses were required to targets at the cued as well as the uncued (.20 probability) locations. Significant cueing effects (improved performance for a cued-location target vs. uncued-location target) were observed for all three levels of cue conflict for both RT (all *p* values < .01) and accuracy (all *p* values < .05) (see Supplemental Figure 1). These additional behavioral findings demonstrate that the cueing parameters were sufficient to induce subjects to covertly orient spatial attention during the cue-target interval regardless of cue discriminability level. This conclusion is supported by related fMRI analyses of biasing activity to cues in visual cortex described below.

### Cues Activated Attentional Control Systems and Modulated Visual Cortex

To identify the frontoparietal attentional control network, activity associated with attend cues was compared with activity to neutral cues (e.g., Woldorff et al., 2004; Hopfinger et al., 2000). Because neutral cues were never followed by targets, strictly "cue-only" attend cues (not followed by target) were used for this comparison. Independent of the direction of cueing or discriminability level, attention-directing cues elicited greater activity than neutral cues in and around the FEFs, in the posterior portion of middle frontal gyrus, and in the intraparietal sulcus region of posterior parietal cortex (PPC). These areas are consistent with the frontoparietal attentional control network reported in numerous prior studies (Slagter et al., 2007; Woldorff et al., 2004; Giesbrecht, Woldorff, Song, & Mangun, 2003; Corbetta et al., 2000; Hopfinger et al., 2000) (see Figure 2A; Table 1). Note that these regions are those that would be predicted to be activated to exert top–down attentional control in trial $N + 1$ in response to the detection of conflict in trial $N$.

Previous studies have shown that activity increases in areas of retinotopically organized visual cortex contralateral to the direction of attention in response to attention-directing cues (Giesbrecht et al., 2003; Hopfinger et al., 2000). We interrogated activity in visual cortex and found that extrastriate visual cortical regions contralateral to the cued hemifield were activated in response to the cues and before the targets (see Figure 2B), independent of cue discriminability. This pattern of pretarget biasing activity in visual cortex suggests that subjects were successfully processing the cues and shifting spatial attention as instructed.

### High-conflict Cues Activated ACC

Because there has been no well-established neuronal circuitry described for processing conflict generated by competing attentional orienting responses, we performed a whole-brain analysis comparing BOLD activity associated with high-conflict attend cues to activity associated with low-conflict attend cues. This analysis allowed us to identify the network of regions involved with monitoring conflict in deployment of selective attention. Across the entire brain, the (high-conflict–low-conflict) contrast yielded greater activity in the dorsal ACC, bilateral anterior insular cortex, and right lateral parietal cortex (Table 2). These regions are similar to regions previously reported to be involved in cognitive control and/or conflict monitoring (Liston, Matalon, Hare, Davidson, & Casey, 2006; MacDonald et al., 2000; Botvinick et al., 1999). Thus, the brain areas activated by conflict during attentional orienting in the present task are highly overlapping with areas associated with conflict monitoring in response inhibition tasks such as the Stroop task.

We further explored the areas active in the high-conflict–low-conflict contrast to determine which regions were sensitive to the parametric manipulation of cue conflict. Beta values for each cue condition in each ROI were compared in pairwise $t$ tests. Areas showing significant differential activity for the high versus medium cue conflict comparison included ACC (high > med), $t(16) = 3.34$, $p = .004$, right insula (high > med), $t(16) = 3.29$, $p = .005$, and right lateral parietal cortex (high > med), $t(16) = 2.72$, $p = .015$. Only ACC was also significantly modulated in the medium versus low-cue conflict comparison (med > low), $t(16) = 2.79$, $p = .013$. In contrast, no regions of the frontoparietal attentional control network were modulated by cue conflict (all $p > .1$). Therefore, the only brain region that showed a parametric modulation of activity across the three levels of cue conflict was ACC (see Figure 2D), supporting the hypothesis that ACC is involved with performance monitoring during shifts of covert spatial attention.

### Frontoparietal Attention Network Activity Increased after Conflict Trials

To evaluate the time courses of activity in the ACC and the frontoparietal network as a function of cue conflict and directed attention, we extracted the HDRs in these brain regions

and computed d-HDRs. In FEF and PPC, the attend minus neutral cue d-HDRs showed a clear peak three to four TRs (i.e., 4.5–6 sec) after cue onset, as would be expected of a response time locked to the attention directing cues that were related to attentional control for the current trial (trial *N*) (see Figure 3A, left panels). During this same period, however, in ACC there is no significant response visible in the d-HDR (see Figure 3A, right panel). In contrast, in the d-HDR formed by subtraction of low-conflict from high-conflict cues, there is a clear peak three to four TRs after cue onset in ACC (see Figure 3B, right panel), but no response at this period in FEF and PPC (see Figure 3B, left panel). Overall then, the pattern for the current trial (trial *N*) is that attentional control engages the frontoparietal network but not ACC, whereas cue conflict engages ACC but not the frontoparietal network. Next we ask whether cue conflict on trial *N* results in changes in attentional control in trial *N* + 1, the subsequent trial.

The effects of cue conflict on trial *N* on brain activity in trial *N* + 1 can be observed by looking at the d-HDRs in Figure 4 at six to seven TRs after cue onset of trial *N* (each trial duration was three TRs). There is a clearly discernable peak in the frontoparietal network during the period of six to seven TRs after cue onset of trial *N* [combined FEF: high > low, $t(16) = 2.96$, $p = .009$; combined PPC: high > low, $t(16) = 2.63$, $p = .018$], which is three to four TRs after cue onset of trial *N* + 1 (see Figure 3B, left panel), but no such activity in ACC (see Figure 3B, right panel). In other words, activity in the frontoparietal network during trials after high-conflict trials was significantly greater than activity during trials after low-conflict trials, across all trial types. Thus, high conflict in trial *N* generated high ACC activity in trial (*N*), which was followed by increased frontoparietal activity in trial (*N* + 1).

## Trial *N* ACC Activity Is Correlated with Trial *N* + 1 Frontoparietal Activity

Previous studies of next-trial effects have examined the correlation between conflict-generated ACC activity and top–down control activity in the following trial (Liston et al., 2006; Kerns et al., 2004). In our paradigm, the most appropriate comparison for such a correlational analysis was between the difference in ACC activity in response to high- versus low-conflict trials and the difference in frontoparietal activity after a high- versus low-conflict trial. Hence, we performed a between-subjects correlational analysis between differential (high–low) ACC activity by conflict level and differential frontoparietal activity by previous trial conflict level. We found a strong positive correlation between conflict-generated ACC activity (trial *N*) and postconflict (trial *N* + 1) adjustments in frontoparietal attentional control regions ($r = .757$, $p = .0004$) (see Figure 3C). There was no significant relation between differential ACC activity and total ACC activity ($r = .07$, $p > .5$), nor between differential frontoparietal activity and total frontoparietal activity ($|r| < .2$, $p > .4$ for all regions), indicating that the magnitude of the differential activity

## Performance Improved Significantly after High-conflict Trials

Next, we investigated whether the observed neurophysiological adjustments were related to improved performance. Mean RT was significantly faster (934.7 vs. 896.7 msec), $t(16) = 4.89$, $p = .0002$, and accuracy was significantly higher (96.4% vs. 92.4%), $t(16) = 3.36$, $p = .004$, for trials (*N* + 1) after high-conflict trials (*N*) compared with trials after low-conflict trials (see Figure 4A). Moreover, performance was more significantly influenced by the conflict level of the previous trial than by the conflict level of the trial currently being performed. In other words, although conflict in trial *N* had a slightly negative effect on performance in trial *N*, it had a significantly positive effect on performance in trial *N* + 1. Presumably, this is because the conflict in trial *N* stimulates ACC activity, which then leads to increased frontoparietal activity and increased attentional control in trial *N* + 1.

### Neurophysiological Increases Correlated with Performance Improvements

To further investigate the idea that the increased frontoparietal activity was responsible for the task improvements, we tested whether a correlation existed between the amount of frontoparietal activity after high-versus low-conflict trials and the degree of performance improvement on those trials. As can be seen from the scatterplots in Figure 4B, there is a positive correlation between frontoparietal activity and accuracy rate for trials after high-versus low-conflict trials ($r = .586$, $p = .013$) as well as a positive correlation between frontoparietal activity and response speed for these trials ($r = .606$, $p = .009$). Although speed and accuracy were both positively correlated with increased frontoparietal activity, they were not correlated with each other ($r = .28$, $p > .2$), and partial correlations existed between frontoparietal activity and accuracy, controlling for speed ($r_p = .545$, $p = .029$), and between frontoparietal activity and speed, controlling for accuracy ($r_p = .566$, $p = .022$). A positive correlation also existed between ACC activity during high-versus low-conflict trials and accuracy rate for trials after high- versus low-conflict trials ($r = .696$, $p = .002$) but not between ACC activity during high- versus low-conflict trials and response speed for trials after high- versus low-conflict trials ($r = .371$, $p = .14$). All correlations reported are between-subjects.

## DISCUSSION

Our results support the hypothesis that ACC monitors conflict and engages the frontoparietal network to control the focus of attention. In our paradigm, high conflict in trial (*N*) generated high ACC activity in trial (*N*), which led to increased frontoparietal attention network activity in trial (*N* + 1) as well as improved behavioral performance in trial (*N* + 1). This pattern of activity is indicative of a functional interaction between the two major cognitive systems supporting performance. The performance-monitoring system detects conflict and leads to the amelioration of the conflict via task-appropriate modulation of activity within top–down control areas (Liston et al., 2006; Egner & Hirsch, 2005; Kerns et al., 2004; Botvinick et al., 2001; Berlyne, 1960). Our data establishes an interaction between the ACC conflict-monitoring system and the frontoparietal attentional control system.

Response conflict occurs when two or more response programs are activated simultaneously (for a review, see Coles, Gratton, & Donchin, 1988). Typically, investigations of conflict use paradigms in which a prepotent response (e.g., reading the word in a Stroop task) competes with the task-relevant response (i.e., naming the color in the Stroop task) (Kerns et al., 2004; MacDonald et al., 2000). When these signals are in opposition, conflict occurs and ACC is significantly more active than during conditions when the responses are complimentary (i.e., when word and color require same response). Our paradigm induces conflict somewhat differently; rather than requiring the inhibition of a prepotent response, the task's inherent perceptual uncertainty induces coactivation of competing response programs (i.e., shift attention leftward vs. shift attention rightward). This manipulation generates varying degrees of conflict, reflected by the different levels of ACC activation to each level of perceptual uncertainty (see Figure 2D).

A recent review of ACC's performance-monitoring activity has outlined a distinction between two types of trial-to-trial adjustments that occur after ACC activation: one type that results in a more cautious response mode and another that results in an increase in the efficiency of information processing (Ridderinkhof et al., 2004). In a spatial attention paradigm such as the one presented here, the task-appropriate adjustment is an increase in information processing efficiency via enhanced spatial attention. Thus, one would expect to observe increased activity in control regions supporting spatial attention, namely, the frontoparietal attentional control network. Although other studies have shown increased activity in small regions of dlPFC (Kerns et al., 2004) and perceptual processing regions

such as FFA (Egner & Hirsch, 2005) on trials after ACC activation, this study is the first to show increased activity throughout the frontoparietal control network after ACC activation.

ACC is heavily interconnected with the supplementary eye fields (Luppino, Matelli, & Rizzolatti, 1990), a brain region implicated in the planning and generation of saccades and in covert visual attention paradigms. It has also been shown to project directly to both FEFs (Stanton, Bruce, & Goldberg, 1993; Huerta, Krubitzer, & Kaas, 1987) and PPC (Selemon & Goldman-Rakic, 1988; Pandya, Van Hoesen, & Mesulam, 1981), critical elements of the frontoparietal attentional control network. In fact, a portion of ACC itself has been shown to be involved in oculomotor control (Paus, Petrides, Evans, & Meyer, 1993) and as such is sometimes referred to as the "cingulate eye fields." Considering this functional connectivity, it is possible that a top–down signal from ACC could influence attentional control, similarly to what has been observed with subthreshold microstimulation of FEF (Moore & Fallah, 2004) or superior colliculus (Muller, Philiastides, & Newsome, 2005). Thus, the modulation of attentional control regions that follows conflict detection between competing attentional orienting responses may take place via mono- or polysynaptic connections.

Our current investigation manipulated cue discriminability to modulate the level of conflict generated by processing the cues. Frontoparietal activity increased on trials after high-conflict trials and performance improved on these trials as well. Typically, postconflict adjustments are directed toward the source of conflict, which in this paradigm is the cue. However, because subjects' manual responses were to the targets rather than the cues, it is difficult to determine whether the elevated performance in trial $N + 1$ reflects enhanced processing of the cues, targets, or both. Unfortunately, because of the temporal smearing inherent with the HDR, the precise timing at which the postconflict frontoparietal modulation occurs is not possible to determine definitively from the fMRI data. However, given that enhanced spatial attention over trial $N + 1$ would improve discrimination of both cue and target stimuli, it is possible that the neurophysiological and behavioral adjustments could reflect improved processing of the cues themselves, improved efficiency of shifting attention during the cue-target interval, improved target processing, or some combination of the above processes.

Although the current investigation has used manipulations of conflict level to selectively activate ACC, other models suggest that ACC is sensitive to various aspects of performance, such as response selection (Turken & Swick, 1999), conscious effort (Naccache et al., 2005; Paus, 2001), and error probability (Brown & Braver, 2005). Although the results reported in this data set do not preclude such other interpretations of ACC function, a converging and parsimonious explanation is that ACC generally monitors performance and is activated under conditions for which the current influence of "control exertion" regions (e.g., dlPFC, frontoparietal network) is insufficient for optimal performance. This explanation is consistent with reports that decreased activity in frontal control regions can be associated with attentional lapses and suboptimal performance (Weissman, Roberts, Visscher, & Woldorff, 2006). Furthermore, the fact that activity in control regions tend to be enhanced for trials after high ACC activity could be taken as evidence that insufficient activity in control regions was contributing to ACC activation.

In the paradigm we have used, task difficulty was manipulated to modulate conflict level, making it difficult to rule out the interpretation that ACC activity in our paradigm is simply a reflection of the level of task difficulty. This situation is ubiquitous in standard conflict manipulations because, for example, incongruent trials are naturally more difficult than congruent trials in Stroop and flanker paradigms. In fact, some proponents of the conflict-monitoring hypothesis have even suggested that conflict is directly related to task difficulty (Aston-Jones & Cohen, 2005). Although our paradigm does not establish a distinction

between task difficulty and conflict, the lack of such a distinction does not preclude the interpretation that ACC monitors performance and conflict in our spatial attention study and is correlated with increased performance and enhanced activity in task relevant (frontoparietal) areas on the following trial.

The present findings establish that conflict-related ACC activity in a voluntary spatial attention task is predictive of subsequent activity in major attentional control regions (frontoparietal network) outside the dlPFC, which is commonly found in studies of conflict and cognitive control. The behavioral results show a next-trial effect of both increased accuracy and faster RT. Taken together with the fact that ACC was the only cortical region whose activity level modulated parametrically with conflict level, we believe that this set of results indicates that the role of ACC as a performance monitor and potential signal to top–down control centers extends beyond typical cognitive control paradigms.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

## Acknowledgments

The authors thank John Ryan, Santani Teng, and Sean Fannon for assistance. Special thanks to Barry Giesbrecht for the collaboration. This study was supported by the NEI Traineeship (grant no. T32 EY015387 to B. J. W.) and the National Institute of Mental Health (grant no. MH 055714 to G. R. M. and grant no. MH059883 to C. S. C.).

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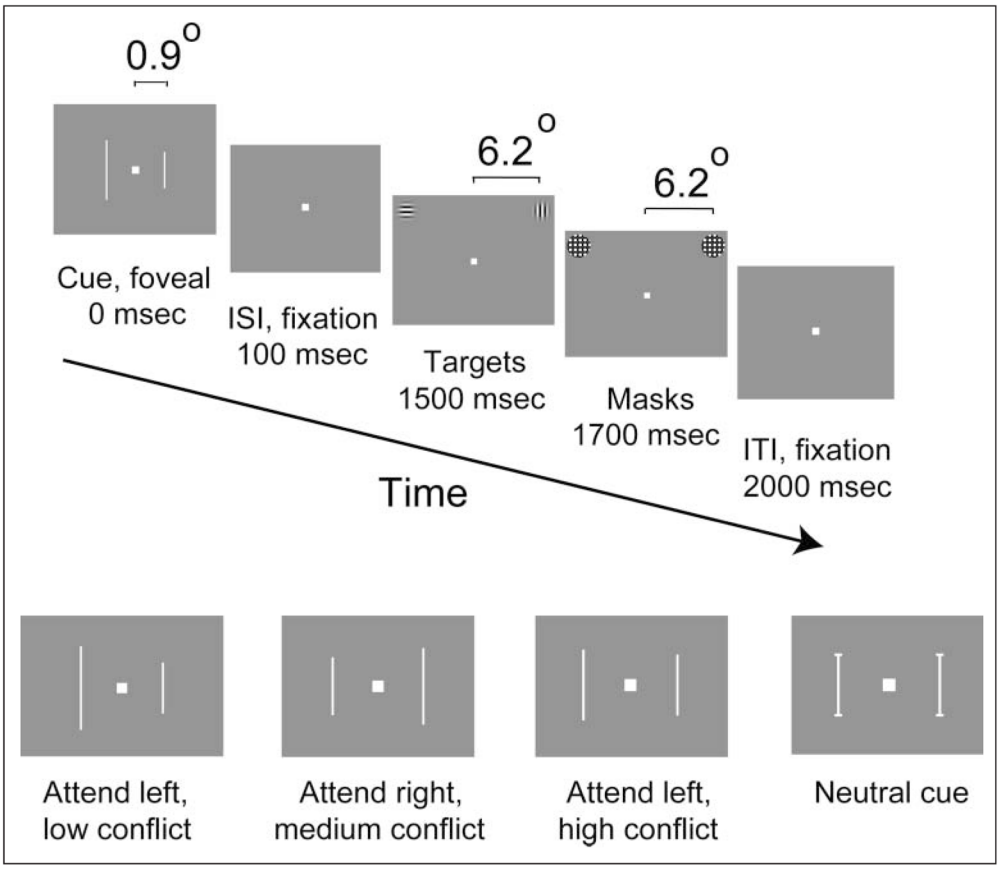


**Figure 1.**

Experimental sequence and sample cue types. (Top) Example of a low-conflict, attend left trial. Participants were required to maintain fixation throughout each trial on the central square. Cues were flashed for 100 msec and followed 1500 msec after onset by a bilateral target display of oriented gratings flashed for 200 msec, which was immediately masked by a patterned stimulus. (Bottom) Sample cue types showing three of the six possible attend cue types (2 spatial directions × 3 conflict levels) plus the neutral cue (at far right).

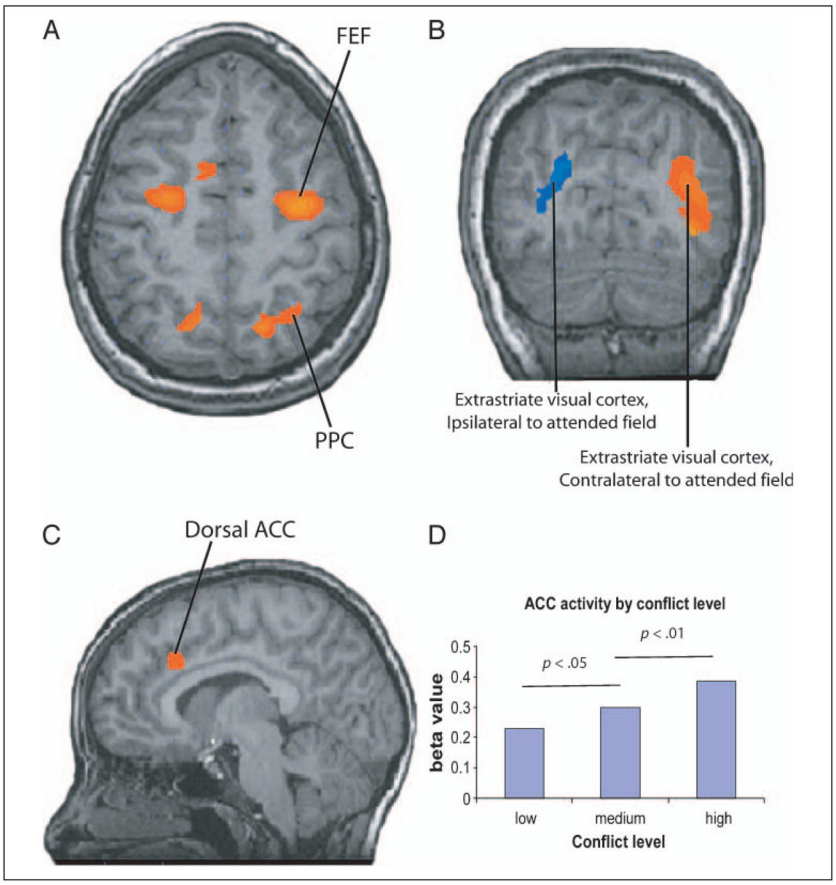


**Figure 2.**

BOLD activity differences between conditions. (A) Frontoparietal attentional control network, as defined by (attend–neutral) contrast. (B) Extrastriate visual cortex activity for (attend left–attend right) contrast. (C) Anterior cingulate activity for (high- to low-conflict) contrast. (D) ACC was the only area across brain whose activity was parametrically modulated across the three conflict levels.

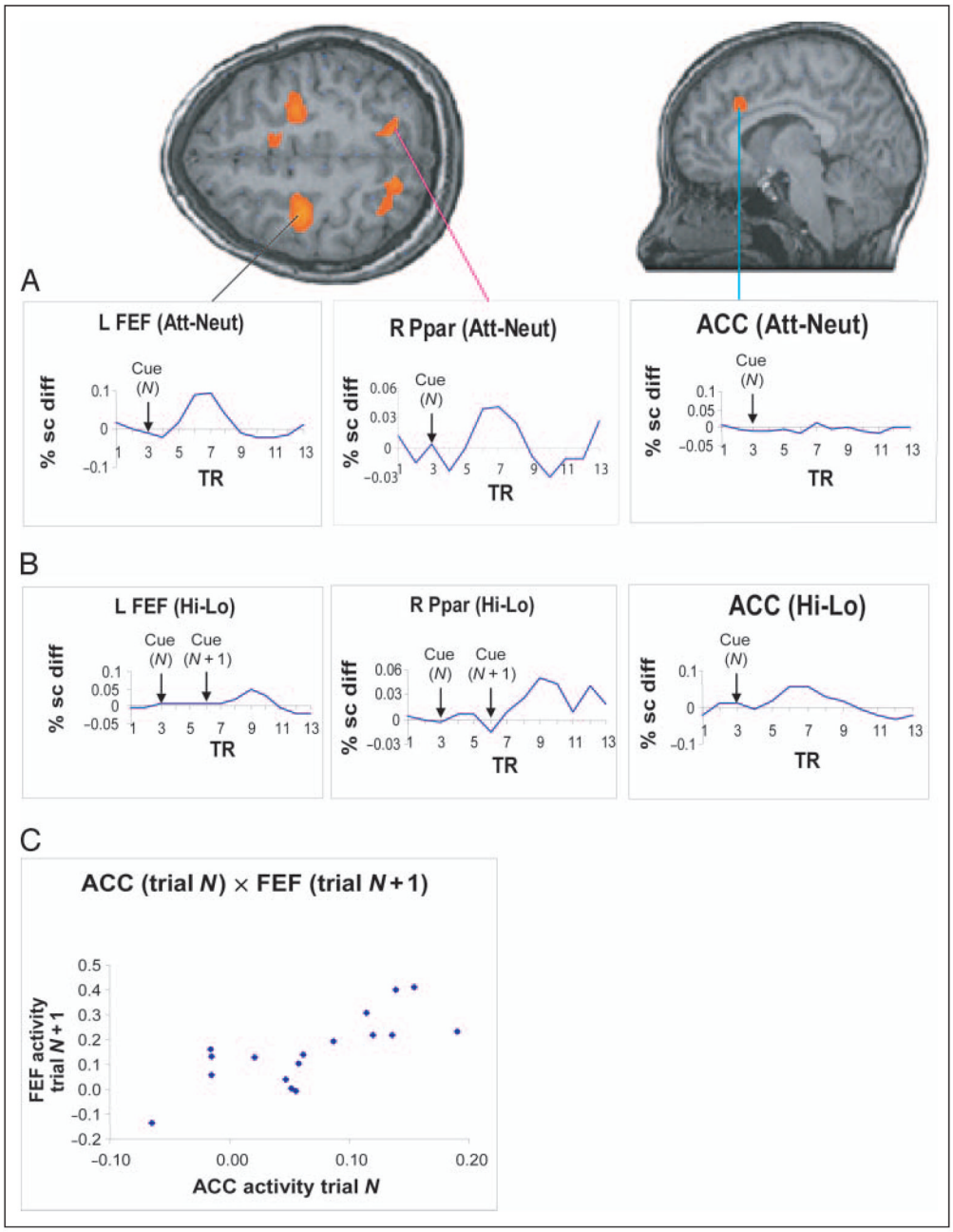


**Figure 3.**

Time course differences for frontoparietal regions and ACC. (A) Time course differences showing activity differences between attend and neutral conditions. TR = 1.5 sec, trial length = three TRs, trial (*N*) onset at TR 3. (B) Time course differences showing next-trial effects of increased frontoparietal activity in trial *N* + 1 after high versus low conflict in trial *N* (bottom-left and bottom-middle panels). TR = 1.5 sec, trial length = three TRs, trial (*N*) onset at TR 3, trial (*N* + 1) onset at TR 6. (C) Scatterplot showing the significant positive correlation between ACC activity on trial *N* and frontoparietal activity on trial *N* + 1 on the basis of high- versus low-conflict level in trial *N*.

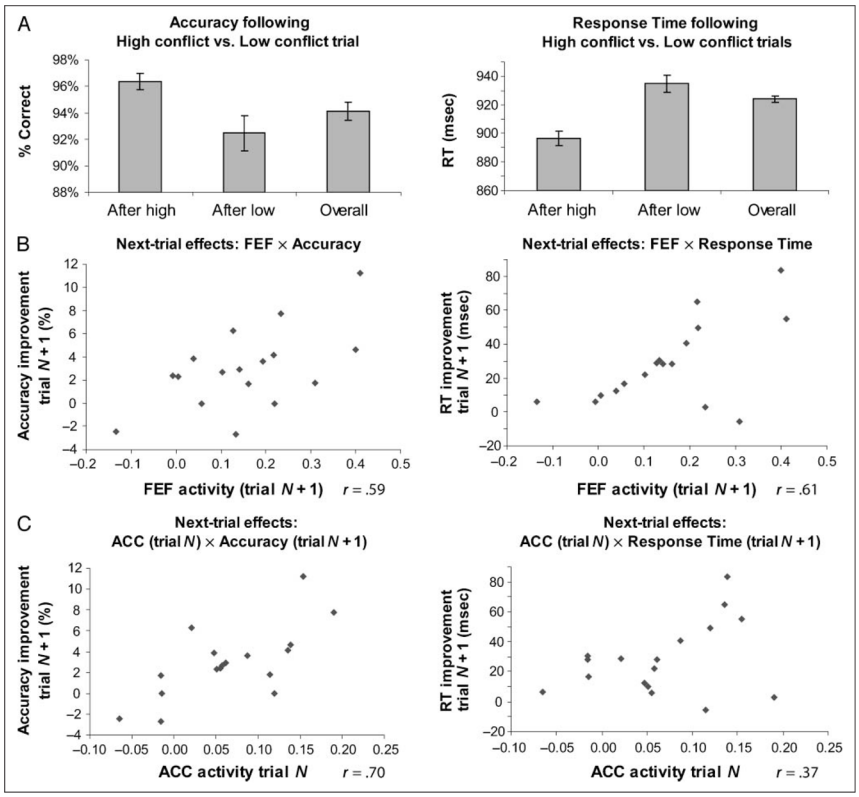


**Figure 4.**

Task performance on trial $N + 1$. (A) Participants were more accurate ( $p < .005$) and faster ( $p < .001$) responding to trial $N + 1$ after high- versus low-conflict trials. (B) FEF activity in trial $N + 1$ was positively correlated with accuracy ( $p < .05$) and response speed ( $p < .01$) on trial $N + 1$ after high- versus low-conflict trials. ACC activity (high vs. low conflict) in trial $N$ was positively correlated with accuracy ( $p < .005$) on trial $N + 1$ after high- versus low-conflict trials. is not simply a function of the magnitude of overall activity in these regions (ACC and FEF/PPC).

**Table 1**

Talairach Coordinates, Cluster Sizes, and Statistical Values of Major Clusters Activated in the (Attend – Neutral) Contrast

| Brain Area | Cluster Size | x | y | z | Z Score |
|---|---|---|---|---|---|
| L middle frontal gyrus (FEF) | 761 | −28 | −7 | 46 | 5.65 |
| R middle frontal gyrus (FEF) | 660 | 24 | −7 | 44 | 4.67 |
| L PPC (intraparietal sulcus) | 472 | −15 | −57 | 50 | 3.75 |
| R PPC (intraparietal sulcus) | 262 | 25 | −54 | 46 | 3.64 |
| L superior frontal gyrus (supplementary eye field) | 64 | −8 | 2 | 51 | 3.15 |

**Table 2**

Talairach Coordinates, Cluster Sizes, and Statistical Values of Major Clusters Activated in the (High Conflict – Low Conflict) Contrast

| Brain Area | Cluster Size | x | y | z | Z Score |
|---|---|---|---|---|---|
| R dorsal ACC | 579 | 6 | 21 | 35 | 4.95 |
| L dorsal ACC | 283 | −4 | 16 | 36 | 4.14 |
| R insula | 946 | 31 | 21 | 12 | 5.32 |
| L insula | 763 | −38 | 13 | 11 | 5.02 |
| R PPC | 68 | 27 | −54 | 31 | 3.52 |

## Table 3

Effect of Conflict Level on Accuracy and Response Time, for Both the Current ("Same") Trial and Following ("Next") Trial

| | Conflict Level | | |
|---|---|---|---|
| | **Low** | **Medium** | **High** |
| *Accuracy* | | | |
| Same trial | 95.3% | 95.2% | 92.8% |
| Next trial | 92.4% | 93.1% | 96.4% |
| *Response Time* | | | |
| Same trial | 911.4 | 919.2 | 925.9 |
| Next trial | 934.7 | 924.9 | 896.7 |