

**Common and unique components of response inhibition  
revealed by fMRI**

**Tor D. Wager**

**Department of Psychology**  
**Columbia University**

**Ching-Yune C. Sylvester**  
**Steven C. Lacey**  
**Derek E. Nee**  
**Michael Franklin**  
**John Jonides**

**Department of Psychology**  
**University of Michigan**

**Correspondence should be addressed to:**

**Tor D. Wager**  
**Department of Psychology**  
**Columbia University**  
**1190 Amsterdam Ave.**  
**New York, NY 10027**

**Email:** [tor@paradox.psych.columbia.edu](mailto:tor@paradox.psych.columbia.edu)  
**Telephone:** (734) 717-1595

**Abstract**

The ability to inhibit inappropriate responses is central to cognitive control, but whether the same brain mechanisms mediate inhibition across different tasks is not known. We present evidence for a common set of frontal and parietal regions engaged in response inhibition across three tasks: the go/no-go task, the flanker task, and the stimulus-response compatibility task. Regions included bilateral anterior insula and anterior prefrontal, right dorsolateral and premotor, and parietal cortices. Principal components analysis showed a coherent pattern of individual differences in these regions that was positively correlated with interference costs in behavioral performance in all three tasks. (Of individual regions, the insula was the most consistently correlated across tasks.) Other regions were uniquely activated by and uniquely correlated with performance in only one task: thalamus and right parietal for go/no go, left anterior insula for flanker, and left parietal for go/no go. Overall, the results provide evidence for multiple mechanisms for response inhibition, with common mechanisms activated in response to cognitive/motor conflict.

## Introduction

Withholding inappropriate responses is a hallmark of human behavior. This can be observed in many individual tasks in which the tendency to make an automatic or natural response must be suppressed in order to make an appropriate but unnatural response. Much research has been concerned with cases in which conflicting motor responses of this sort are engaged (e.g., Dagenbach & Carr, 1994; Dejong, Coles, & Logan 1995; Anderson & Spellman, 1995; Kornblum, Hasbroucq, & Osman, 1990). It is often argued that resolving cases of motor conflict requires the engagement of inhibitory processes that dampen the tendency to make the inappropriate response in favor of the appropriate one (e.g., Kornblum & Requin, 1995; Logan, 1985; Logan & Cowan, 1984; Lowe, 1979). The issue we address is: Are these inhibitory processes all of a sort? That is, when one has to inhibit an inappropriate motor response in one situation, are the same mechanisms recruited as when one has to inhibit a response in another situation?

We engaged this issue by conducting a simple experiment, collecting behavioral and fMRI data: All participants were faced with three tasks in which avoiding an inappropriate response was required. The three tasks varied a great deal in their specific demands and perceptual components, but they shared a common need to inhibit inappropriate responses. One was a stimulus-response compatibility task (SRC), in which a compatible mapping of stimuli to responses had to be overcome in order to respond appropriately to each stimulus. We compared incompatible responses to compatible ones to isolate any inhibition-related processes that might be involved in this task. A second task involved the go/no-go (GNG) paradigm, in which a response had to

be withheld on presentation of one imperative stimulus but a response had to be executed upon presentation of a different stimulus. We compared “no-go” trials, which required response inhibition, with “go” trials in which the response was executed. The third situation was a flanker task in which a response had to be made to a central stimulus while ignoring flanking stimuli. In this task, we compared trials in which the flanking stimuli were mapped onto incompatible responses relative to the target response with trials on which the flanking stimuli mapped onto compatible responses.

The question we asked of these three cases was this: Is the same signature of brain activation obtained across tasks? Alternatively, are there distinct patterns of brain activation that are tied to individual situations in which inhibition is required? In either case, we were also interested in whether the pattern or patterns of brain activation would correlate with the corresponding behavioral measures of inhibition.

The question at hand is a pressing one because there is a good deal of controversy in the literature about whether there are common mechanisms of inhibition at work in these and other similar tasks. On the one hand, some previous research with response interference paradigms has shown a relatively cohesive set of areas that are involved in these inhibitory tasks. The anterior cingulate, dorsolateral prefrontal, inferior prefrontal, premotor, and parietal cortices have all been implicated in SRC tasks (Dassonville et al., 2001; Iacoboni, Woods, & Mazziotta, 1996; Merriam et al., 2001; Peterson et al., 2002; Schumacher & D'Esposito, 2002), GNG tasks (Casey et al., 1997; Klingberg & Roland, 1997; Konishi et al., 1999; Liddle, Kiehl, & Smith, 2001; Menon, Adleman, White,

Glover, & Reiss, 2001; Rubia et al., 2001), and flanker tasks (Bunge, Hazeltine, Scanlon, Rosen, & Gabrieli, 2002; Casey et al., 2000; Hazeltine, Poldrack, & Gabrieli, 2000). On the other hand, when examined behaviorally, there is very little if any correlation in performance between one inhibitory task and another (Burgess, Alderman, Evans, Emslie, & Wilson, 1998; Duncan, Johnson, Swales, & Freer, 1997; Fan, Flombaum, McCandliss, Thomas, & Posner, 2003).

One issue unresolved in previous research is that studies that putatively target “inhibitory processes” generally use a single task and rely on subtraction methods or parametric variation to isolate the inhibitory processes of interest. The Stroop task, in which participants must name the ink color of a color word (e.g., “RED” printed in blue ink) while ignoring the word form, is perhaps the classic example of an “inhibitory” task. However, behavioral research has shown that the Stroop task is likely to involve multiple types of interference, with multiple ways of resolving them (Kornblum et al., 1990). Irrelevant information may be inhibited at perceptual, semantic, or response-selection stages of processing. Activations in these tasks could arise from several factors that differ between “inhibition” and control conditions, including enhanced attention to the task in general, increased demand for divided attention, enhanced long-term memory demands when maintaining the less automatic task-set in the inhibition task, and other factors.

Two features of our design help circumvent these problems. First, by studying three tasks that share a common requirement for response inhibition, we can make stronger conclusions about areas that are activated in all three tasks. Second, we chose tasks such that response inhibition is ostensibly the only type of inhibition common to all

three tasks. In the GNG task, inhibition may occur only at the response-selection or execution stages. In the Flanker task, inhibition may occur at the perceptual or response-selection levels. In the SRC task, inhibition may occur at the stimulus-response rule mapping level (Rubenstein, Meyer, & Evans, 2001) or the response-selection level. In addition, by studying the three tasks in the same participants, we circumvented problems that arise when comparing across studies that use different individual brains, different standard brain spaces, and different analysis methods.

There are two previous reports in the literature that compare different inhibitory tasks within the same individuals, and they do report common regions of activation due to inhibitory processes (Fan et al., 2003; Peterson et al., 2002). However, a second problem is not addressed by these studies: There may be common regions of activation across different tasks that require inhibitory processes, but these regions, in fact, are not critical to inhibitory control, but are engaged by other common processes among the tasks. So, merely finding common regions of activation across tasks is not sufficient to argue that there are common mechanisms of inhibition. What is needed beyond this is the analysis of the correlations of activation in these regions with behavioral measures of inhibitory control. Thus, our analysis specifically searched for regions that showed both activation and correlations between activation and task performance in each inhibitory task. In addition, we asked whether activated regions are organized into coherent networks showing similar patterns of task-related individual differences, and whether these networks are the same across inhibitory tasks.

## **Methods**

### ***Participants***

14 undergraduate students (ages 18-25) from the University of Michigan were recruited through advertisements placed in the campus paper and flyers posted in campus buildings. All participants completed a self-report health screen for neurological and psychiatric diagnoses, as well as drug or alcohol abuse. They signed informed consent forms approved by the University Institutional Review Board and were compensated up to \$40 for their participation.

### ***Behavioral tasks***

There were 6 runs in each scanning session, two for each task. Each run was preceded and followed by a 20s baseline block in which participants fixated on a cross that appeared in the center of the screen. A run consisted of 6 alternating experimental (A) and control (B) blocks of 18 seconds each, in BABA order, for a total of 12 blocks.

Prior to the beginning of each task, participants were given oral instructions and a short practice session of 2 blocks. Participants were further instructed to respond as quickly and as accurately as possible and were informed that they could earn bonuses for speed and accuracy. Before each run the instructions were displayed as long as the participant needed.

To rule out the possibility that experimental tasks might involve more overt or intended eye movements than their controls, and that mechanisms controlling these eye movements might produce activations that would complicate the interpretation of the task related activations, one run of a saccade task was also included. We describe these tasks now.

**Go/No-Go Task.** In this task, participants saw a random sequence of letters one at a time in the center of the screen. In each block, 12 letters and fixation crosses were presented. Whenever the participants saw any letter other than X, they made a keypress with their right index finger. Each of the letters subtended approximately 2 degrees of visual angle, appeared for 440 ms; letters were separated by a 1000 ms central fixation cross.

The two block-types in this experiment were High No-Go (A) and Low No-Go (B). On average, in the High No-Go blocks, 50% of the letters were Xs (i.e., “no-go” trials); in the Low-No-Go blocks, only 20% of the letters were Xs. The beginnings and ends of each block were not marked in any way, and no participant reported noticing the blocked design during a debriefing session. Note that this design allows the data to be analyzed in either a blocked or event-related fashion.

**Flanker Task.** On each trial, participants were shown three colored circles in the middle of the screen. The center circle was the target stimulus, and the two circles to the left and right of it were the flankers. Participants responded to the target, while ignoring

the flankers. The circles could appear in one of four different colors: red, green, yellow, or blue. If the target circle was red or green, participants responded with their left index finger. If the target circle was yellow or blue, participants responded with their right index finger. The two flankers were always of the same color, and that color was always different from the target circle.

There were two types of trials: congruent and incongruent. A trial was congruent if the responses indicated by the target and the flankers were the same. A trial was incongruent if the responses indicated by the target and the flankers were different. The stimuli were presented in alternating blocks of incongruent (A) and congruent (B) trials. For each trial, the three circles were displayed simultaneously for 1000 ms followed by a 440 ms central fixation cross. As is true for the GNG task, the blocked design was not directly revealed to the participants. No participant stated that he/she noticed the blocked nature of the task in a debriefing session.

**Stimulus-Response Compatibility Task.** Participants were presented with a series of left or right pointing arrows in the center of the screen. Each arrow consisted of an arrowhead and a rectangular stem, subtending approximately 3 degrees of visual angle. Each block of trials was preceded by an instruction screen informing the participants of the response required to each arrow: “same” or “opposite.” In the “same” or compatible blocks (B), participants responded with the index finger indicated by the direction of the arrow. Conversely, in the “opposite” or incompatible blocks (A),

participants responded with the index finger that opposed the direction indicated by the arrow.

In this task, each block consisted of a 2000 ms instruction screen followed by 11 arrows each presented for 1000 ms separated by 440 ms fixation crosses. Note that the instructions given at the beginning of each block made participants explicitly aware of the compatibility or incompatibility of the upcoming trials.

**Saccade Task.** As a control for possible confounding eye movements, a saccade task was included in which participants directed their gaze to the location of a series of fixation crosses on the screen. Each block consisted of 11 fixation crosses, each presented for 440 ms and separated by a 1000 ms central fixation cross. The crosses could appear in one of 8 random locations. Blocks of saccade-trials alternated with 18s baseline control blocks, in which participants fixated on a single centrally located cross.

***Image acquisition and pre-processing***

MRI images were acquired using a 3T GE Signa scanner equipped with the standard quadrature headcoil (General Electric, Milwaukee, WI). Head movement was minimized using foam padding and a cloth restraint strapped across participants' foreheads. Experimental tasks were presented using E-Prime (Beta 5.0) software (Psychology Software Tools, Inc.) and the IFIS 9.0 system with a 10-button response unit (MRI Devices Corp.).

Functional T2\*-weighted BOLD images were acquired using a spiral sequence of 15 contiguous axial 5 mm slices (TR = 1500 ms, TE = 30 ms, flip angle = 90°, field of view (FOV) = 24 cm). Two structural images were also acquired: a T1-weighted gradient echo (GRE) image was acquired using the same FOV and slices as the functional scans (TR = 300, TE = 6.8, flip angle = 65°); a high-resolution spoiled GRASS (Gradient Recalled Acquisition in Steady State; SPGR) image was also acquired (TR = 6.4, TE = 1.5, TI = 600, flip angle = 15°, FOV = 24 cm, 2.5 mm slice thickness). The T1 GRE images were acquired before the functional runs, and SPGR images were acquired after.

Functional images were corrected for slice acquisition timing differences using a local, 17-point sinc interpolation program (Oppenheim, Schafer, & Buck 1999) and corrected for head movement using the realignment routines in the Automated Image Registration (AIR) package (Woods, Grafton, Holmes, Cherry, & Mazziotta, 1998). Subsequent preprocessing and analysis was done using SPM99 (Wellcome Department of Cognitive Neurology, London). Individual SPGR images were corrected for signal inhomogeneity (Kristoff & Glover,

psych.stanford.edu/~kalina/SPM99/Tools/vol\_homocor.html), and then co-registered to the corresponding T1 GRE images. SPGR images were then normalized to the SPM99 T1 template, which is in Montreal Neurological Institute (MNI) space, and those normalization parameters were applied to the T2\* (functional) images. After spatial normalization, T2\* images were smoothed using a 10 mm FWHM Gaussian filter. All of the analyses included a temporal high-pass filter (100 s) and each image was scaled to have a global mean intensity of 100.

### ***Image Analysis***

All analyses were performed using the General Linear Model implemented in SPM99, with separate regressors and intercepts for each run. For the flanker and stimulus-response compatibility tasks, epochs the length of each task block were convolved with a standard hemodynamic response function (HRF). For the go/no-go task, event onset times for the no-go trials and go trials were convolved with the HRF. Contrast images for each participant were subjected to a random-effects analysis, and all statistical results were thresholded using a False Discovery Rate (FDR) correction for multiple comparisons (Genovese, Lazar, & Nichols, 2002) of  $p < .05$  and 4 contiguous voxels. The FDR correction ensures that no more than an average of 5% of activated voxels for each contrast will be false positives (the extent threshold is in addition to this requirement). Peak coordinates in MNI space were converted into Talairach coordinates using a transformation by Matthew Brett ([www.mrcbu.cam.ac.uk/Umaging/mninspace.html](http://www.mrcbu.cam.ac.uk/Umaging/mninspace.html)). Brodmann areas were identified using the Talairach atlas (Talairach & Tournoux, 1988) as implemented by the Talairach

Daemon ((Lancaster et al., 2000); <http://ric.uthscsa.edu/projects/talairachdaemon.html>); both MNI and Talairach coordinates are reported.

**Classifying voxels into common and unique areas.** We were interested in whether a set of brain regions showed consistent responses across all types of inhibition, and/or whether some brain regions were unique to particular inhibitory tasks. Finding the former would be evidence for general mechanisms shared across response inhibition tasks, whereas finding the latter would identify mechanisms associated with unique inhibitory processes.

To achieve this goal, we had to determine which brain regions were activated in each task, and whether these brain regions were or *were not* activated in the other tasks. We first identified regions that were activated in at least one task (FDR-corrected,  $p < .05$ ). We then classified voxels in this set of regions into the following categories of interest. *Common* regions showed activation in all three tasks at the FDR corrected threshold. However, the corrected statistical threshold for concluding that an area was activated in a whole-brain analysis is high, and areas that do not meet this stringent threshold may still show evidence for activation at a lower threshold. To overcome this thresholding issue, we also determined which regions were common at a lower threshold of uncorrected  $p < .05$  in all three tasks (Fan et al. 2003). *Unique* regions showed activation (FDR-corrected,  $p < .05$ ) in one task, but not in the other two. We chose to search for regions that showed such qualitative differences because they can more readily

be interpreted as involving unique processes, whereas quantitative comparisons are less easily interpretable (Sternberg, 2001).

For both common and unique regions, we calculated correlations between brain activation in each task and corresponding behavioral performance measures. This allowed us to identify regions that are likely to be functionally related to task performance in each task (for common regions) or in only one task (for unique regions). Because individual outliers may heavily influence results with small sample sizes, particularly in correlational analyses, we also calculated robust correlation values by identifying and removing outliers using the Minimum Covariance Determinant algorithm (Rousseeuw & Van Driessen, 1999). However, with small sample sizes, two random variables may also be coplanar by chance, potentially inflating false-positive rates for robust correlations (Hubert, 2001). To avoid this problem, we examined correlations that showed significant values for both ordinary correlations and robust correlations to provide converging evidence that correlations were not induced by outliers. In our study, where the ordinary and robust correlation values produced similar results, we proceeded to interpretation. However, we refrained from interpreting correlations where the two estimates differed by more than  $r = .4$ . Although other combinations of activation and correlation patterns exist—for example, a region may be activated in two tasks, and correlated with performance in two tasks—we focus on regions with consistent common (across all three tasks) or unique (only in one task) patterns as they are of the greatest theoretical interest.

## Results

### **Behavioral Results**

Behavioral analyses revealed significant interference caused by the active inhibition component of each task. In all three tasks, the behavioral data were analyzed on a block-level, comparing accuracy and response times for the low-conflict blocks with those in the high-conflict blocks (i.e., high no-go, congruent, and compatible blocks compared to low no-go, incongruent, and incompatible blocks).

In the Go/No-go task (GNG), the false alarm rate (FAR) was the measure of interference because, of course, responses were not made on no-go trials. FARs were significantly greater during low no-go ( $\bar{x} = .19$ ) than high no-go blocks ( $\bar{x} = .03$ ;  $\bar{x}$  difference = .17,  $\sigma = .094$ ,  $t(13) = 6.57$ ,  $p < .0001$ ). Overall reaction times were equivalent for low no-go ( $\bar{x} = 394$  ms) and high no-go ( $\bar{x} = 392$  ms) blocks.

In the Flanker task, reaction times were greater for incongruent than congruent blocks (609 and 551 ms, respectively;  $\bar{x}$  difference = 58 ms,  $\sigma = 20$  ms,  $t(13) = 10.6$ ,  $p < .0001$ ). Accuracy showed effects in the same direction as in the GNG task, with higher accuracy in the low conflict blocks (92.4% and 96.8% for incongruent and congruent blocks, respectively,  $t(13) = 3.28$ ,  $p < .05$ ).

In the Stimulus-Response Compatibility (SRC) task, reaction times were greater for incompatible than compatible blocks (408 and 371 ms, respectively;  $\bar{x}$  difference = 37

ms,  $\sigma = 20$  ms,  $t(13) = 7.03$ ,  $p < .0001$ ). In this task, there were no differences in accuracy across high and low conflict blocks—accuracy was virtually the same for incompatible and compatible trials (97.4% and 97.8%).

Correlations among behavioral inhibition measures (inhibition - control RTs for SRC and Flanker, and inhibition – control in FAR for go/no-go) revealed negligible relationships among performance on the three tasks: For GNG & Flanker,  $r = -.17$ , for GNG & SRC,  $r = -.25$ , and for Flanker & SRC,  $r = .12$ , all nonsignificant. Robust correlations produced similar results.

These inhibition measures were used as the behavioral measures for brain-behavior correlations for the Flanker and SRC tasks. Higher scores on each measure indicate less efficient inhibition, and thus greater task interference. Notably, every participant showed an interference effect in the expected direction on each task, providing further evidence of the behavioral robustness of these effects.

## **Imaging Results**

**Individual inhibition task activations.** Task comparisons designed to isolate inhibition-related activity produced reliable activations in all three tasks, as shown in Table 1 and Figure 1. Activated areas, corrected for multiple comparisons ( $p < .05$  corrected) using FDR (Genovese et al., 2002) in random effects analyses, included regions in 1) superior and inferior parietal cortex, 2) anterior prefrontal, 3) premotor, 4)

insula, 5) caudate/putamen, 6) anterior cingulate, 7) posterior cingulate, and 8) premotor cortex bilaterally, as well as 9) right dorsolateral prefrontal and 10) left thalamus.

**Saccade task activations.** We wanted to rule out the possibility that inhibitory activations were related to differences in eye movements (overt or intended). To aid in localizing tasks relative to the frontal and parietal eye fields, and other regions known to be involved in producing eye movements, we identified voxels activated in the saccade task at a lenient threshold ( $p < .05$  uncorrected, shown in cyan in Figure 2). As expected, activated regions included bilateral frontal and parietal eye fields, supplementary eye field, and a large contiguous region in the medial and lateral occipital cortex. Saccade regions were not considered as candidates for common or unique inhibition regions, described below.

**Common regions.** We classified voxels as belonging to *common* regions if they met the corrected threshold in all three tasks. Three regions shown in Table 2 met this criterion: Anterior cingulate (extending into the right superior frontal sulcus) and right caudate/putamen. At a lower threshold of 0.05 uncorrected in all three tasks, used in previous studies comparing inhibition tasks (Fan et al. 2003), several additional regions emerged (Table 2), including bilateral anterior insula and anterior PFC, right DLPFC, left caudate and posterior intraparietal sulcus (IPS) and right anterior IPS.

Correlations between activations in these common regions and reaction time performance indicated that among the regions, only the anterior insula (bilaterally)

showed significant correlations between activation and behavioral performance in all three tasks. The minimum correlation for any task in left or right insula was  $r = 0.46$ , with a critical  $r$  of 0.46 one-tailed or  $r = 0.53$  two-tailed. Robust correlation values ranged from  $r = .61$  to  $.76$  for each task, all  $p < .05$ . At most one outlier was removed from each correlation. Figure 3A shows the common region in right insula. Figure 3B shows the magnitude of activation in the critical comparison for each task, and reveals that the right anterior insula showed no evidence for saccade-related activity (the left insula, not shown for space reasons, revealed the same pattern of results). Figure 3C shows robust correlations between behavioral performance measures (x-axis, higher scores reflect more task interference) and activation (y-axis), both expressed in units of standard deviations for easy comparison across tasks. These correlations were significant for each of the three tasks in both left and right anterior insula.

**Unique regions.** We next classified voxels as belonging to *unique* regions if they were significant at corrected  $p < .05$  in one task, but showed no evidence for significance in the other tasks at a lower threshold (uncorrected  $p < .05$ ). Table 4 shows results of all such regions. We then examined correlations with behavioral performance on each of the three tasks. If a region is really uniquely activated in one task, it should show brain-performance correlations for that task only. Regions that showed such unique patterns of correlation as well as activation are separated in Table 4 from other regions whose pattern of correlations were less consistent with the activation pattern. It is these uniquely activated + uniquely correlated regions that we interpret as providing strong evidence for involvement in only one task.

Two such regions were found for the GNG task—right thalamus and left posterior parietal cortex, shown in Figure 4A and Table 3. The left panel of Figure 4A shows the location of the right posterior parietal region unique to the GNG task. The center panel shows reliability of activation for each task averaged across voxels in the region, and indicates that neither Flanker, SRC, nor saccade tasks showed activation in this region. The right panel shows a robust positive correlation between false alarm rate (x-axis) and brain activation in the GNG task (y-axis), indicating that participants with higher interference scores showed more activation in the right parietal cortex. The same pattern of results was found for the thalamus.

The Flanker task showed only one uniquely activated region that also correlated with behavioral interference scores, left putamen/caudate, shown in Table 3 and Figure 4B. The panel descriptions are of the same format as Figure 2.

The SRC task also showed one region that was both uniquely activated in the SRC task and uniquely correlated with SRC performance, in the left inferior parietal cortex. This region, located just medial to saccade-activated regions, is shown in Figure 4C. A second area in left mid-cingulate—19 mm to the right and 15 mm posterior to the common anterior cingulate region—was correlated with SRC performance, but it was not uniquely related to the SRC task because activation in this region was also negatively correlated with performance in the GNG task.

**Network analysis.** Principal components analysis (PCA) was performed on participants' activation scores (contrast values) in what we have defined as common regions. Scores within each contiguous common region were averaged across voxels, so that the ten variables in the analysis were the average activity in each of the ten common regions—bilateral anterior insula and anterior PFC, anterior cingulate, right DLPFC, right SFS (2 regions), right inferior parietal, and intraparietal sulcus (IPS; Table 2). Examination of the correlations among regions revealed generally positive correlations in each of the three tasks. Thus, participants with high activation in one region tended to show high activation in the other regions on the same task. PCA eigenvectors were compared with a null-hypothesis distribution of eigenvalues generated using a permutation test. One principal component in each analysis was significant (eigenvalues of 6.00 for GNG, 5.18 for Flanker, and 6.18 for SRC, all  $p < .001$ , with no other significant eigenvalues). Common regions were considered part of the “network” in each task if they correlated at least  $r = .53$  ( $p < .05$ ) with the principal component. The full set of networked common regions (that loaded highly on the principal component) for each task is shown in the right columns of Table 2.

We first tested the hypothesis that activation in the networks as a whole predicted behavioral performance in each task. Component scores (the measure of network activation) in each task were correlated positively with performance,  $r = .37$ , n.s. for GNG,  $r = .57$ ,  $p < .05$  for Flanker, and  $r = .55$ ,  $p < .05$  for SRC. We next tested whether behavioral performance on each task was *uniquely* predicted by brain activation for that task. This was done by entering component scores on all three tasks as predictors in

multiple regression, with performance on each task as the dependent variable and one regression analysis per task. This allowed us to examine whether performance was related to activation in multiple tasks, suggesting common interference-prevention mechanisms, or by only activation in the same task, suggesting independent mechanisms in the same brain regions. We found that in general, component scores uniquely predicted behavioral performance on each task, with partial correlations of  $r = .57$ ,  $p < .05$  for GNG,  $r = .56$ ,  $p < .05$  for Flanker, and  $r = .60$ ,  $p < .05$  for SRC. The only exception was that GNG activation predicted SRC performance, partial  $r = .54$ ,  $p < .05$ , as well as GNG performance.

Finally, we tested the hypothesis that the activation in common regions for each task was correlated with activation for other tasks. Positive correlations among tasks and with behavioral scores would indicate that individuals' activation is consistent across the tasks and predicts a common component underlying behavioral performance. The absence of correlations between task activations (in the presence of correlations between activation and performance in each task) would suggest that the networks are tracking the amount of behavioral interference experienced in a particular task. In this way, we have the beginnings of a conceptual model for determining whether activation is due to common mechanisms of interference prevention or resolution after it occurs. Importantly, the component scores for the three tasks were not inter-correlated, with all correlation values between .006 and .10, suggesting that the networks track interference experienced in each task (evidenced by the activation-performance correlations) but are less likely to reflect common mechanisms that prevent interference across tasks. Thus, if these brain regions perform the same underlying function in each task, it is likely a function that

becomes active in response to interference, such as conflict monitoring, response selection or decision making, or suppression of late, inappropriate motor tendencies (Kornblum & Requin, 1995). We return to this point in the discussion.

**Meta-analysis regions.** A previous meta-analysis of brain activations from these same inhibitory tasks has isolated a set of regions that the tasks may share in common (Nee, Jonides, & Wager, 2004). Using sets of contiguous voxels in these common regions, we created regions of interest (ROIs). Analysis of the average activity in these ROIs corresponded closely with results from the analyses presented above. Meta-analysis revealed consistent inhibition regions summarized in Table 5. Two of the meta-analysis regions—DLPFC and anterior cingulate—were active in all three tasks, paralleling findings from the common-region analyses.

The left insular meta-analysis region was 7 mm rostral and 10 mm superior to the common region reported earlier, and was uniquely activated and correlated with performance in the Flanker task, as shown in Figure 5B. This result parallels the anterior spread of activation only in the Flanker task (Figure 2). The right angular gyrus meta-analysis region shared an identical center of mass with the right posterior parietal region found to be unique to the GNG task, and thus paralleled results from the previous *unique* region analysis, as shown in Figure 5C. The results of the meta-analysis thereby support our previous findings.

## Discussion

This study demonstrates that diverse inhibitory tasks—one that required withholding of a response (Go/No-go), one that required inhibiting encoding and/or responses (Flanker), and one that required re-mapping stimulus-response associations (SRC)—shared substantial overlap in neural activations in the same participants. A core set of commonly activated regions, including bilateral insula, anterior PFC, right DLPFC and right SFS, were also correlated with one another, suggesting that they are activated in each task “of a piece.” Activation in this network, and in the bilateral insula individually, tracked behavioral performance in each task, suggesting that the network is sensitive to the amount of interference encountered by each individual subject on each task. However, the degree to which an individual activated this network varied from task to task, according to that individual’s performance on the task: More activation was associated with increasing behavioral interference on all three tasks.

The positive correlations we observed may seem counterintuitive, as activation of inhibitory mechanisms that prevent interference might be expected to produce low behavioral interference scores, but they are consistent with similar individual differences effects in task switching (Smith et al., 2001; Wager, 2003). One hypothesis is that these regions are involved in the central decision-making process that fails in poor inhibitors, or in mechanisms of interference detection or resolution that become active in response to conflict (rather than in the prevention of conflict). Another idea is that these areas are involved in inhibiting irrelevant representations (and preventing interference), but they work more efficiently in good inhibitors, requiring and/or producing less excess blood

flow. Other possibilities exist as well: activation during interference-task periods could be more protracted for good performers and extend into the control-task periods, producing poorer model fits in good performers. Demonstrating correlations with individual performance is a first step, and future research may distinguish among these possibilities. Our working hypothesis is that these regions implement interference resolution mechanisms that are engaged relatively late, after conflict has been created.

The common regions found in this study corresponded well with those reported as common activations in previous studies (Fan et al. 2003) and in an original meta-analysis of previous neuroimaging studies of inhibition (Nee et al., 2004). These regions were anatomically distinct from areas related to the basic orienting of attention and control of eye movements, as they showed no evidence for activation in the saccade task.

Previous studies investigating relationships among performance on multiple inhibitory tasks have found that correlations are generally low but (usually) significant, suggesting that there is some common underlying ability of response inhibition (Boone, Ponton, Gorsuch, Gonzalez, & Miller, 1998; Fan et al., 2003; Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). The current study has identified a candidate neural mechanism implementing this ability: an insular-prefrontal-cingulate network that resolves interference between competing responses in each of the three tasks studied here and perhaps others as well.

In a previous study, Fan et al. (2003) investigated the relationships between inhibition tasks and reported common activations in a subset of the regions we report here. Like the present study, they found low correlations among measures of task performance, and concluded that the neural overlap they observed was not strong

evidence for common mechanisms. However, the present study provides stronger evidence for a common mechanism, based on the relationship between activation and task performance across these diverse tasks.

If there are common mechanisms in response interference, why is behavioral performance uncorrelated? One possible answer lies in the locus of the effects. Mechanisms of interference resolution may be common, but the mechanisms that *give rise to* interference may vary from task to task. Prior evidence supports the idea that different amounts of interference may be created in particular individuals on particular tasks. For example, printed color words may interfere with naming ink colors (the standard Stroop effect), but only in those who can read (Ehri & Wilce, 1979). Likewise, hearing-impaired individuals show reduced interference from irrelevant semantic content in an auditory task (Jerger et al., 1993). The degree of interference may in fact reflect both the relative processing speeds and automaticity of relevant and irrelevant information (Dyer, 1973; MacLeod & Dunbar, 1988; Wager, 2003). Both of these factors affect interference *independent of* executive ability, perhaps explaining low correlations among behavioral interference scores, and their effects fit within the framework of computational modeling accounts of interference effects (Cohen, Dunbar, & McClelland, 1990; Kornblum et al., 1990).

Thus, although common interference resolution mechanisms may exist, the amount of behavioral interference varies from task to task depending on how an individual's existing biases (strategic or otherwise) interact with the perceptual and motor characteristics of the task. In such a case, the activity in the interference resolution system of the brain should track the degree of interference experienced on each task.

That is, if a participant is worse on the flanker task than on the go/no-go task, that person should show relatively greater activation in the flanker task.

One important alternative candidate function for the interference resolution network identified in this study is the inhibition of perceptual representations (irrelevant stimuli). Two pieces of evidence argue against this alternative. First, if the network served to inhibit perceptual representations, its activation would not be expected to be related to performance in all three tasks, as neither the Go-no/go task nor the SRC task contain stimuli whose suppression might facilitate task performance. Second, if the network were responsible for suppression of unwanted percepts, then worse performance (more interference) would be expected to be associated with less activation in these control structures. However, we found correlations in the opposite direction.

Paralleling the low correlations in performance among the tasks, we found brain areas uniquely involved in performance of each task we studied. In each case, at least one uniquely activated area was also uniquely correlated with behavioral performance, strengthening the region's candidacy as a mediator of task-specific processes. The Go-no/go task, for example, involves only one stimulus and a single, simple response. Thus, it may place the highest demand on systems for pure motor inhibition and controlling the timing of response execution. It also places high demand on the flexible linking of stimuli with the intention to act. Thalamic and right parietal activations unique to Go-no/go may reflect these processes.

The Flanker task is the only task that involves space-based perceptual inhibition, as flankers can be excluded from further processing based on their location—a particularly effective strategy if stimuli are separated by more than one degree of visual

angle (Humphreys & Bruce, 1989). The flanker task might also uniquely involve selecting which stimulus to act upon. Unlike the Go-no/go task, a response is always made; and unlike the SRC task, the relevant stimulus must be selected rather than the relevant response. Unique Flanker activation and correlation in the head of the caudate may reflect the selection-for-action of relevant stimuli. This seems the most likely working hypothesis given the caudate's widely acknowledged role in motor planning and its apparent involvement in task-switching (Sylvester et al., 2002; Wager, 2003).

The SRC task requires a response on every trial, like the Flanker, but in this task it is the relevant stimulus-response mapping rule that must be selected. Previous research has pointed to parietal cortex involvement in this task (Iacoboni et al., 1996). We found superior and inferior parietal activations in all three tasks (and in the saccade task as well; Figure 2), although one region in left inferior parietal cortex around the temporal-parietal junction was unique to the SRC task. Thus, this region is the best candidate for a mediator of the stimulus-response rule selection unique to the SRC task. However, due to our stringent requirements for unique activation and correlation, the approach we have taken here may miss a number of important regions in all tasks, particularly those regions in superior parietal cortex that overlap with saccade activations but are otherwise unique to SRC.

### References

- Anderson, M. C., & Spellman, B. A. (1995). On the status of inhibitory mechanisms in cognition: memory retrieval as a model case. *Psychol Rev*, *102*(1), 68-100.
- Boone, K. B., Ponton, M. O., Gorsuch, R. L., Gonzalez, J. J., & Miller, B. L. (1998). Factor analysis of four measures of prefrontal lobe functioning. *Archives of Clinical Neuropsychology*, *13*(7), 585-595.
- Bunge, S. A., Hazeltine, E., Scanlon, M. D., Rosen, A. C., & Gabrieli, J. D. E. (2002). Dissociable contributions of prefrontal and parietal cortices to response selection. *Neuroimage*, *17*(3), 1562-1571.
- Burgess, P. W., Alderman, N., Evans, J., Emslie, H., & Wilson, B. A. (1998). The ecological validity of tests of executive function. *Journal of the International Neuropsychological Society*, *4*(6), 547-558.
- Casey, B. J., Thomas, K. M., Welsh, T. F., Badgaiyan, R. D., Eccard, C. H., Jennings, J. R., et al. (2000). Dissociation of response conflict, attentional selection, and expectancy with functional magnetic resonance imaging. *Proceedings of the National Academy of Sciences*, *97*, 8728-8733.
- Casey, B. J., Trainor, R. J., Orendi, J. L., Schubert, A. B., Nystrom, L. E., Giedd, J. N., et al. (1997). A developmental functional MRI study of prefrontal activation during performance of a go-no-go task. *Journal of Cognitive Neuroscience*, *9*, 835-847.

- Cohen, J. D., Dunbar, K., & McClelland, J. L. (1990). On the control of automatic processes: a parallel distributed processing account of the Stroop effect. *Psychol Rev*, 97(3), 332-361.
- Dassonville, P., Lewis, S. M., Zhu, X. H., Ugurbil, K., Kim, S. G., & Ashe, J. (2001). The effect of stimulus-response compatibility on cortical motor activation. *Neuroimage*, 13(1), 1-14.
- Duncan, J., Johnson, R., Swales, M., & Freer, C. (1997). Frontal lobe deficits after head injury: Unity and diversity of function. *Cognitive Neuropsychology*, 14(5), 713-741.
- Dyer, F. (1973). The Stroop phenomenon and its use in the study of perceptual, cognitive, and response. *Memory & Cognition*, 1(2), 106-120.
- Ehri, L., & Wilce, L. (1979). Does word training increase or decrease interference in a Stroop task? *Journal of Experimental Child Psychology*, 27(2), 352-364.
- Fan, J., Flombaum, J. I., McCandliss, B. D., Thomas, K. M., & Posner, M. I. (2003). Cognitive and brain consequences of conflict. *Neuroimage*, 18(1), 42-57.
- Genovese, C. R., Lazar, N., & Nichols, T. E. (2002). Thresholding of statistical maps in functional neuroimaging using the false discovery rate. *Neuroimage*, 15(4), 870-878.
- Hazeltine, E., Poldrack, R., & Gabrieli, J. D. E. (2000). Neural activation during response competition. *Journal of Cognitive Neuroscience*, 12, 118-129.
- Hubert, M. (2001). Multivariate outlier detection and robust covariance matrix estimation - Discussion. *Technometrics*, 43(3), 303-306.

- Humphreys, G. W., & Bruce, V. (1989). Visual attention. In *Visual cognition: Computational, experimental, and neuropsychological perspectives* (pp. 143-190). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Iacoboni, M., Woods, R. P., & Mazziotta, J. C. (1996). Brain-behavior relationships: Evidence from practice effects in spatial stimulus-response compatibility. *Journal of Neurophysiology*, 76(1), 321-331.
- Jerger, S., Stout, G., Kent, M., Albritton, E., Loiselle, L., Blondeau, R., et al. (1993). Auditory Stroop Effects in Children with Hearing Impairment. *Journal of Speech and Hearing Research*, 36(5), 1083-1096.
- Klingberg, T., & Roland, P. E. (1997). Interference between two concurrent tasks is associated with activation of overlapping fields in the cortex. *Brain Res Cogn Brain Res*, 6(1), 1-8.
- Konishi, S., Nakajima, K., Uchida, I., Kikyo, H., Kameyama, M., & Miyashita, Y. (1999). Common inhibitory mechanism in human inferior prefrontal cortex revealed by event-related functional MRI. *Brain*, 122, 981-991.
- Kornblum, S., Hasbroucq, T., & Osman, A. (1990). Dimensional overlap: Cognitive basis for stimulus-response compatibility: A model and taxonomy. *Psychological Review*, 97(2), 253-270.
- Kornblum, S., & Requin, J. (1995). *LRP evidence of automatic response activation by irrelevant stimuli*. Paper presented at the Second Annual Meeting of the Cognitive Neuroscience Society, San Francisco, CA.

- Lancaster, J. L., Woldorff, M. G., Parsons, L. M., Liotti, M., Freitas, C. S., Rainey, L., et al. (2000). Automated Talairach atlas labels for functional brain mapping. *Hum Brain Mapp, 10*(3), 120-131.
- Liddle, P. F., Kiehl, K. A., & Smith, A. M. (2001). Event-related fMRI study of response inhibition. *Human Brain Mapping, 12*(2), 100-109.
- Logan, G. D. (1985). Executive control of thought and action. *Acta Psychologica, 60*, 193-210.
- Logan, G. D., & Cowan, W. B. (1984). On the ability to inhibit thought and action: A theory of an act of control. *Psychol Rev, 91*, 295-327.
- Lowe, D. G. (1979). Strategies, context, and the mechanism of response inhibition. *Memory & Cognition, 7*, 382-389.
- MacLeod, C. M., & Dunbar, K. (1988). Training and Stroop-like interference: evidence for a continuum of automaticity. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 14*, 126-135.
- Menon, V., Adleman, N. E., White, C. D., Glover, G. H., & Reiss, A. L. (2001). Error-related brain activation during a Go/NoGo response inhibition task. *Human Brain Mapping, 12*(3), 131-143.
- Merriam, E. P., Colby, C. L., Thulborn, K. R., Luna, B., Olson, C. R., & Sweeny, J. A. (2001). Stimulus-response incompatibility activates cortex proximate to three eye fields. *NeuroImage, 13*, 794-800.

- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., & Howerter, A. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*, 49-100.
- Nee, D., Jonides, J., & Wager, T. D. (2004). *A meta-analysis of inhibitory tasks in neuroimaging. Manuscript in preparation.* Unpublished manuscript.
- Oppenheim, A. V., Schaffer, R. W., & Buck, J. R. (1999). *Discrete-time signal processing* (2nd ed.). Upper Saddle River, NJ: Prentice Hall.
- Peterson, B. S., Kane, M. J., Alexander, G. M., Lacadie, C., Skudlarski, P., Leung, H. C., et al. (2002). An event-related functional MRI study comparing interference effects in the Simon and Stroop tasks. *Cognitive Brain Research, 13*(3), 427-440.
- Rousseeuw, P. J., & Van Driessen, K. (1999). A Fast Algorithm for the Minimum Covariance Determinant Estimator. *Technometrics, 41*, 212-223.
- Rubenstein, J. S., Meyer, D. E., & Evans, J. E. (2001). Executive control of cognitive processes in task switching. *Journal of Experimental Psychology: Human Perception and Performance, 27*, 763-797.
- Rubia, K., Russell, T., Overmeyer, S., Brammer, M. J., Bullmore, E. T., Sharma, T., et al. (2001). Mapping motor inhibition: Conjunctive brain activations across different versions of go/no-go and stop tasks. *NeuroImage, 13*, 250-261.
- Schumacher, E. H., & D'Esposito, M. (2002). Neural implementation of response selection in humans as revealed by localized effects of stimulus-response compatibility on brain activation. *Human Brain Mapping, 17*(3), 193-201.

- Smith, E. E., Geva, A., Jonides, J., Miller, A., Reuter-Lorenz, P., & Koeppel, R. A. (2001). The neural basis of task-switching in working memory: effects of performance and aging. *Proc Natl Acad Sci U S A*, 98(4), 2095-2100.
- Sternberg, S. (2001). Separate modifiability, mental modules, and the use of pure and composite measures to reveal them. *Acta Psychol (Amst)*, 106(1-2), 147-246.
- Sylvester, C. Y., Jonides, J., Wager, T., Lacey, S., Smith, E., Cheshin, A., et al. (2002). Processes of interference resolution as revealed by functional magnetic resonance imaging. *Journal of Cognitive Neuroscience*, 129-129.
- Talairach, J., & Tournoux, P. (1988). *A coplanar stereotactic atlas of the human brain*: Theime, Stuttgart.
- Wager, T. D. (2003). *Brain and behavioral mechanisms of switching attention*. Unpublished Dissertation, University of Michigan, Ann Arbor.
- Woods, R. P., Grafton, S. T., Holmes, C. J., Cherry, S.R., & Mazziotta, J. C. (1998). Automated image registration: I. General methods and intrasubject, intramodality validation. *Journal of Computer Assisted Tomography*, 22, 141-154.

Table 1.

Region	Brodmann Area		Flanker Talairach Coordinates			Z score	SRC Talairach Coordinates			Z score	Go/No-Go Talairach Coordinates			Z score
			x	y	z		X	Y	z		x	y	z	
Parietal	7	R					22	-48	58	4.10	8	-60	44	3.38
		L	-15	-64	49	3.09	-22	-45	58	3.66	-11	-48	53	3.12
	40	R					26	-41	48	4.21	45	-38	39	4.20
		L					-38	-35	48	4.03	-64	-35	29	3.84
Anterior prefrontal	10	R	34	41	16	3.78	22	48	7	3.21	30	49	25	4.20
		L	-34	51	16	3.54	-26	48	11	3.55	-34	45	25	3.08
Superior frontal sulcus (premotor)	6	R					19	-5	56	4.87				
		L					-19	2	46	4.17				
Dorsolateral Prefrontal	9/46	R									41	44	7	3.92
Insula	13	R									41	15	-1	4.31
		L	-45	11	-1	3.78								
Anterior cingulate	32	R	8	20	36	4.49					8	21	45	3.39
		L					-8	13	45	4.03				
Posterior cingulate	23	R									4	-13	28	3.79
		L									-4	-17	33	3.71
Caudate		R	19	1	18	3.40	11	-3	23	3.02				
		L	-15	1	18	3.55	-11	-3	23	3.93				
Putamen		R	22	11	9	4.84					15	4	4	3.00
		L	-22	15	8	4.38								
Thalamus		L					-11	-17	19	3.35	8	-21	6	4.87

Table 1. Regions activated at whole-brain corrected thresholds ( $p < .05$ ) in each task.

Table 2.

Common Regions	Volume			Activity (Peak Z-score)			Correlations (Pearson's r)			Robust correlations			Network Affiliation			
	BA	x	y	z	(mm3)	Go/No go	Flanker SRC	Saccade	Go/No go	Flanker SRC	Go/No go	Flanker SRC	Go/No go	Flanker SRC		
<b>Common Regions (FDR corrected in each task)</b>																
Left caudate	-	8	4	5	141	1.97*	3.12*	2.47*	-0.64	0.33	-0.20	0.04	0.33	-0.20	0.16	0.78
Anterior cingulate	32	4	11	45	633	2.95*	4.14*	2.45*	1.58	-0.04	0.24	0.50*	0.90*	0.05	0.65*	0.61
Right caudate	-	11	4	5	492	2.99*	3.37*	3.11*	1.40	0.41	-0.10	-0.10	0.44	-0.10	-0.28	0.91
<b>Common Regions (p &lt; .05 uncorrected in each task)</b>																
L anterior insula	13	-34	19	5	633	3.09*	3.29*	1.79*	1.05	0.46+	0.64*	0.50+	0.61*	0.64*	0.76*	0.74
R anterior insula	13	34	19	5	352	3.35*	3.85*	1.82*	0.44	0.65*	0.47+	0.58*	0.65*	0.64*	0.69*	0.77
L anterior PFC	10	-30	49	20	70	3.02*	3.08*	1.75*	-1.70	0.37	0.36	0.47+	0.90*	0.36	0.69*	0.75
R anterior PFC	10	30	41	15	70	1.73	2.91*	1.68*	-2.52	0.20	0.37	0.23	0.20	0.37	0.63*	0.93
R DLPFC	46/9	41	34	25	281	3.43*	2.00*	2.07*	-2.77	0.32	0.14	0.34	0.32	-0.37	0.65*	0.81
R superior frontal sulcus	6	15	11	55	70	3.53*	2.19*	1.71*	1.04	0.02	0.22	0.65*	0.02	-0.24	0.52	0.69
R premotor cortex	6	19	8	60	70	2.90*	2.88*	1.69*	1.37	-0.03	0.46+	0.61*	0.74*	0.46+	0.61*	0.83
R inferior parietal cortex	40	49	-34	45	70	1.86	1.70	3.89*	0.95	-0.09	0.53*	-0.46+	-0.08	0.78*	-0.78*	0.54
L posterior IPS	7	-15	-60	50	70	1.97*	2.89*	1.85	1.40	-0.17	0.48+	0.11	-0.17	0.69*	0.37	-0.57

Table 2. Regions activated by all three inhibition tasks. \* =  $p < .05$ , + =  $p < .10$ . P-values are based on a one-tailed test for activation, and a 2-tailed test for correlations. Common regions at the lower, uncorrected threshold met the additional restriction of corrected significance in at least one task. Volume indicates the number of cubic mm of brain tissue activated in common. 70 mm of brain tissue equals one 3.75 x 3.75 x 5 mm voxel (used in this study), or 8.75 standard 2 x 2 x 2 mm voxels. Network affiliations columns list the correlation between the average value of each common ROI and the first principal component from PCA analysis; a correlation of  $r > .53$  ( $p < .05$ ) was chosen as the cutoff for considering a region to be part of the network.

Table 3.

Region	BA	x	y	z	Volume (mm <sup>3</sup> )	Activity (Peak Z-score)				Correlations (Pearson's r)			Robust correlations		
						Go/No go	Flanker	SRC	Saccade	Go/No go	Flanker	SRC	Go/No go	Flanker	SRC
<b>Go/no-go unique regions with unique brain-behavior correlations</b>															
Thalamus	-	8	-23	0	2602	4.52*	1.06	1.58	0.27	0.65*	-0.35	-0.07	0.76*	0.11	0.07
R posterior parietal	7/40	34	-64	40	1125	3.31*	0.00	1.30	1.00	0.51+	-0.39	0.18	0.51+	-0.11	0.14
<b>Other Go/no-go unique regions</b>															
L inferior parietal	40	-60	-34	30	281	3.55*	0.63	1.20	1.30	-0.25	0.16	0.14	0.02	0.59*	-0.42
L DLPFC	9	-30	41	30	211	3.29*	1.58	1.03	-2.38	0.35	-0.20	0.08	-0.13	0.15	0.56*
Mid-cingulate	23	4	-19	30	563	3.53*	1.04	0.28	-2.88	0.62*	-0.37	0.62*	0.57*	-0.37	0.61*
R SFS	6	15	19	55	703	3.71*	1.47	1.49	1.26	0.11	0.15	0.70*	0.11	-0.03	0.70*
R anterior PFC	10	30	45	30	352	3.98*	1.15	0.23	-1.16	0.11	-0.39	0.04	0.44	-0.09	-0.15
R inferior parietal	40	49	-45	30	2602	4.49*	1.63	1.63	1.45	0.03	-0.27	0.01	-0.35	-0.86*	0.43
R DLPFC	46/9	45	30	25	422	3.43*	1.39	1.56	-2.85	0.41	0.23	0.47+	0.41	-0.11	0.20
R DLPFC	9	45	8	30	2602	3.91*	0.04	1.60	1.42	0.18	-0.09	0.36	0.18	0.62*	0.50+
R temporal cortex	21/41	49	-45	5	352	3.43*	0.53	-0.14	1.56	-0.08	0.21	0.01	-0.07	0.03	0.37
R sensorimotor cortex	40/2	45	-38	55	211	3.25*	1.35	1.59	-0.15	0.11	0.50+	-0.06	0.11	0.24	-0.12
<b>Flanker unique regions with unique brain-behavior correlations</b>															
L putamen	-	-23	23	10	773	1.47	3.39*	1.64	1.23	0.19	0.53*	0.47+	0.19	0.53*	0.47+
<b>Other Flanker unique regions</b>															
Anterior cingulate	24/32	0	8	40	1688	1.63	4.19*	1.53	0.09	0.07	0.05	0.17	0.55	0.05	0.62*
Rostral anterior cingulate	24	-4	23	25	211	1.37	3.54*	0.78	-2.21	0.12	0.44	0.65*	0.12	0.90*	0.83*
R Caudate	-	26	23	5	1406	1.59	3.70*	1.63	1.39	0.62*	0.42	0.24	0.62*	0.42	0.22
R Putamen	-	23	-8	0	281	0.61	3.51*	1.48	1.31	0.29	0.10	0.01	0.29	-0.73*	0.01
<b>SRC unique regions with unique brain-behavior correlations</b>															
L inferior parietal	40	-34	-45	30	211	-0.42	0.18	3.16*	1.48	-0.15	-0.10	0.44	-0.15	0.39	0.74*
<b>Other SRC unique regions</b>															
Thalamus / Caudate	-	-15	-15	15	3867	1.27	1.63	3.78*	1.27	0.11	-0.33	0.12	0.11	-0.55*	-0.37
Mid-cingulate	6/24	-11	-4	55	352	-0.58	1.43	3.24*	1.56	-0.24	0.14	0.41	-0.57*	0.14	0.80*
R sensorimotor	40/7	26	-45	50	2883	1.59	1.40	4.03*	1.60	0.00	0.38	0.14	0.00	0.38	-0.19
R premotor cortex	6	23	-19	55	2602	1.05	1.59	4.45*	1.56	0.05	0.49+	0.11	0.05	0.49+	-0.38

Table 3. Regions uniquely activated by only one task.  
Significance values are as in Table 2.

Table 5.

Meta-analysis regions	Activity (Peak Z-score)				Correlations (Pearson's r)				Robust correlations						
	BA	x	y	z	Volume (mm <sup>3</sup> )	Go/No go	FlankerSRC	Saccade	Go/No go	FlankerSRC	Go/No go	FlankerSRC			
R DLPFC	9/46	41	19	25	8930	3.55*	3.41*	1.88*	0.31	0.08	0.13	0.30	0.33	0.13	-0.02
Anterior cingulate	32/8	0	23	45	141	1.90*	3.79*	2.09*	-1.05	0.22	0.23	0.57*	0.89*	0.23	0.65*
Medial PFC	8	4	15	55	70	3.32*	2.10*	1.43	1.01	0.26	0.19	0.59*	0.28	0.19	0.59*
L anterior insula/IFG	13/45	-38	26	15	211	0.18	2.94*	1.28	-0.66	0.07	0.59*	0.41	0.07	0.41	0.41
R angular gyrus	39	34	-64	40	70	3.31*	-0.67	-0.58	-3.25	0.51*	-0.34	0.25	0.51	-0.02	0.25

Table 5. Summary of results from the inhibition meta-analysis regions. Meta-analysis regions are listed at the left of the table. R DLPFC – Right Dorsolateral Prefrontal Cortex, MPFC – Medial Prefrontal Cortex, IFG – Inferior Frontal Gyrus.

## Figure Captions

Figure 1. Slices showing activations in each task: go/no go (top row), flanker (middle row), and stimulus-response compatibility (bottom row). All regions shown were significant at  $p < .05$  corrected using False Discovery Rate control in at least one task. Regions significant at  $p < .05$  (uncorrected) in all three tasks were considered common inhibitory regions. The display threshold is  $p < .05$  uncorrected.

Figure 2. Common and unique regions in inhibition tasks. All regions shown are significant  $p < .05$  corrected using False Discovery Rate control in at least one task. Yellow regions are significant in all tasks at  $p < .05$  (uncorrected). Red: significant activation only in go/no-go. Green: significant only in the flanker task. Blue: significant only in the stimulus-response compatibility (SRC) task. Black: significant in at least one task, but not classified as either common or unique. Cyan: Significant in the saccade task ( $p < .05$  uncorrected). Saccade regions were excluded from further analysis.

Figure 3. Right anterior insula and correlations with task performance. Left anterior insula (not shown) also showed consistent positive correlations between task performance and brain activity across the three inhibition tasks. A) Slice showing the location of the common anterior insula region. B) Barplot showing the max Z-values (random effects analysis, y-axis) for each task (x-axis). Horizontal lines indicate statistical thresholds. C) Correlation scatterplot showing robust brain-behavior correlations between activation and performance for each task. Red circles: Go/No-go task; Green squares: Flanker task; Blue triangles: SRC task. D) Network regions in which activation among regions was significantly intercorrelated for each of the three tasks (principal components analysis of contrast values across participants in common regions). E) Partial correlation scatterplots for the first principal component (network scores) for each task and behavior in that task. The plots show that performance in each task uniquely predicted network activation in that task, controlling for other performance variables.

Figure 4. Selected unique regions showing correlations between activation and task performance. A) Shows the Thalamus with corresponding activations for the four tasks, and the correlation plot for false alarm rate in the Go/NoGo task, B) Left Putamen with corresponding activations, and the correlation plot for inhibition in the flanker task, and C) Left Inferior Parietal with corresponding activations, and the correlation plot for inhibition in the SRC task.

Figure 5. Shows voxels selected from the meta-analysis for A) Left Anterior Insula with corresponding activations for the three tasks, and the correlation plot for inhibition in the flanker task, B) Right Posterior Parietal with corresponding activations, and the correlation plot for false alarm rate in the Go/NoGo task, and C) Medial Prefrontal Cortex with corresponding activations for the three tasks, and the correlation plot for inhibition in the SRC task.

Figure 1.

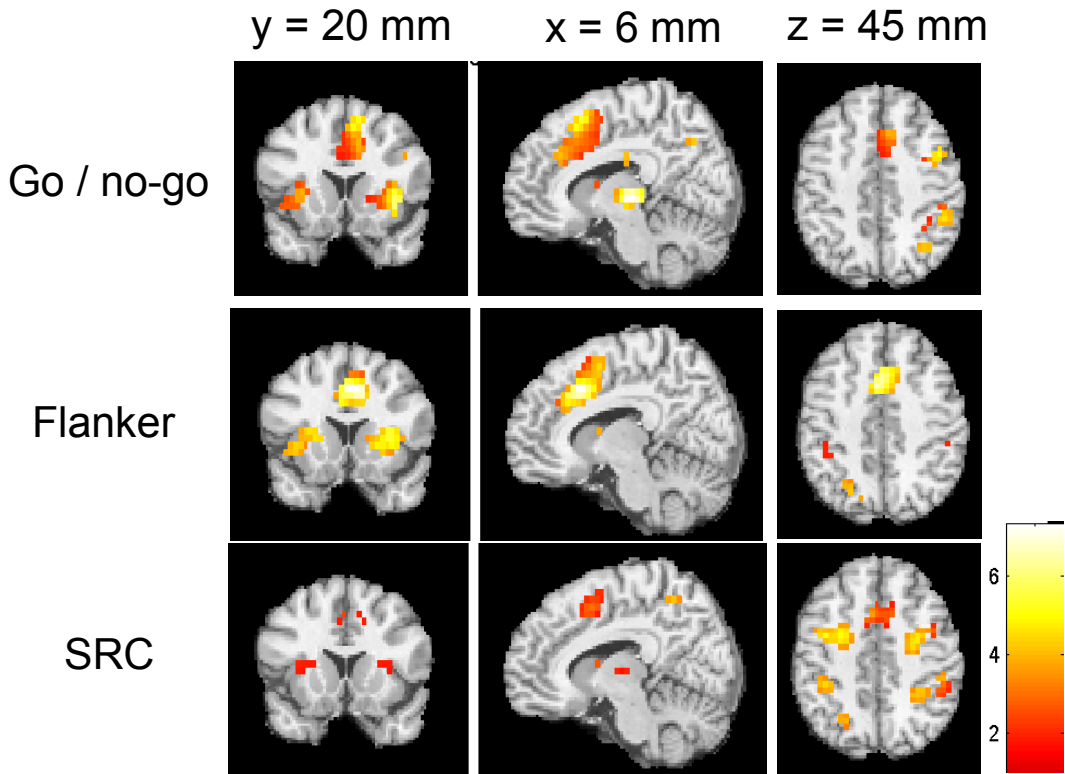


Figure 2.

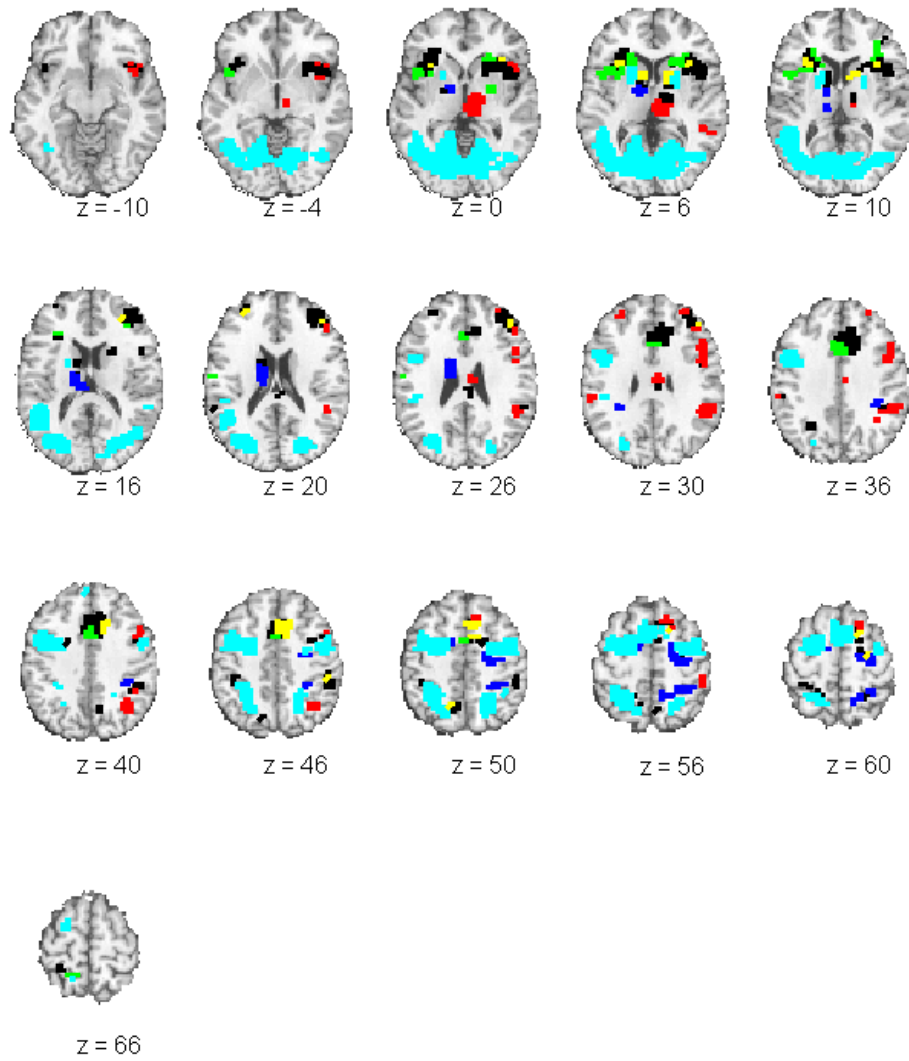


Figure 3.

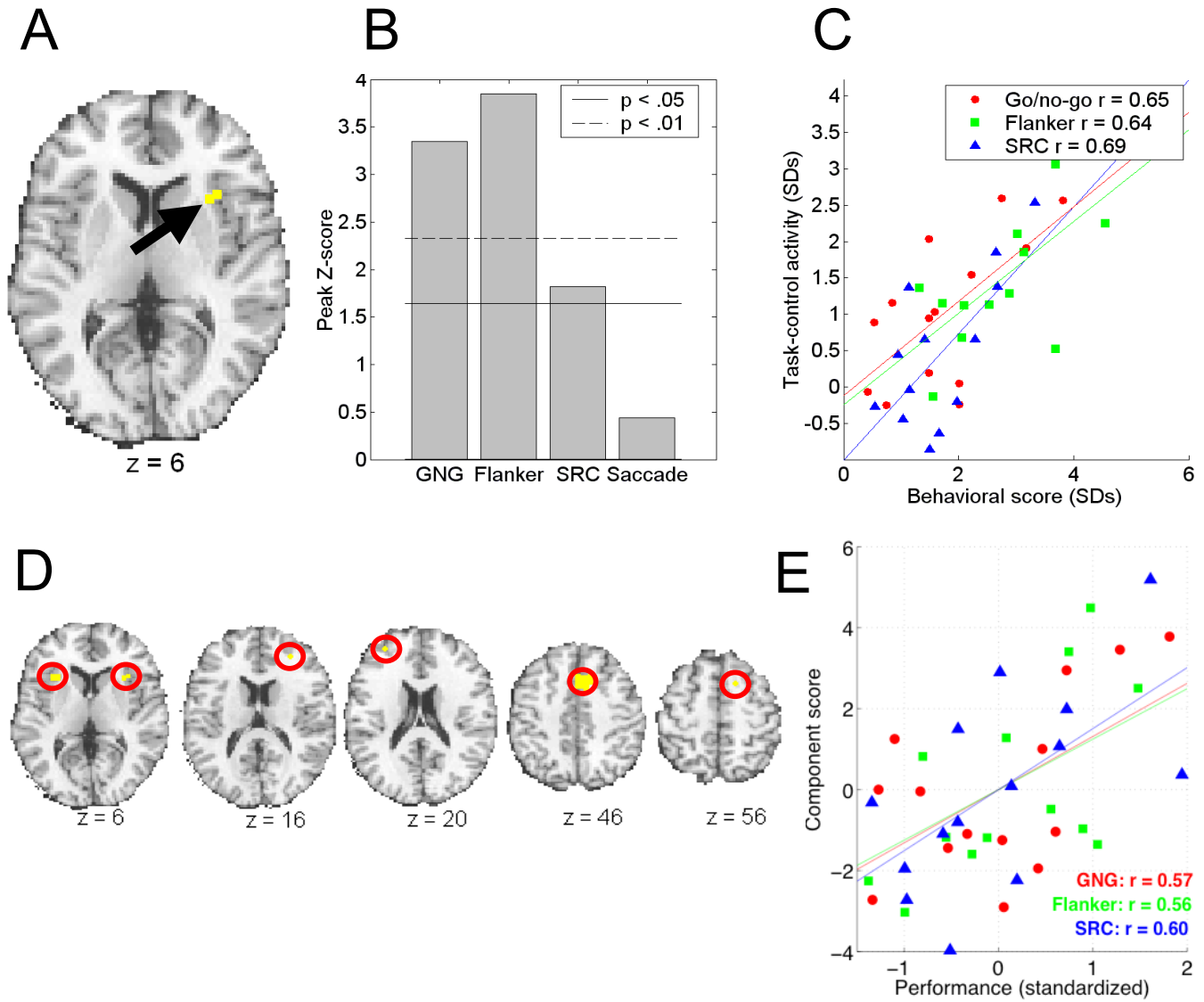


Figure 4.

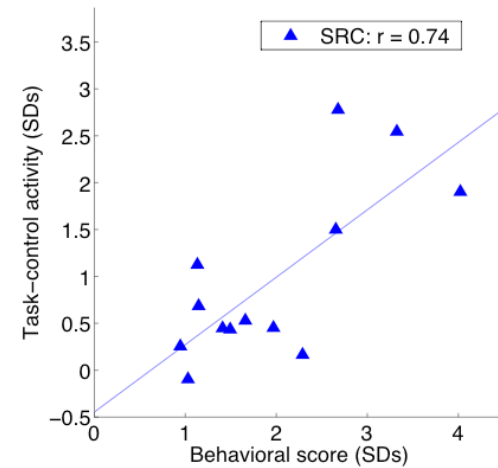
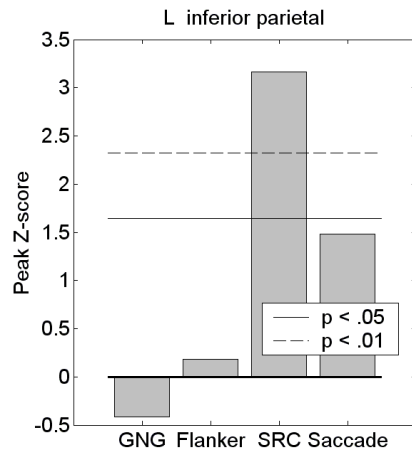
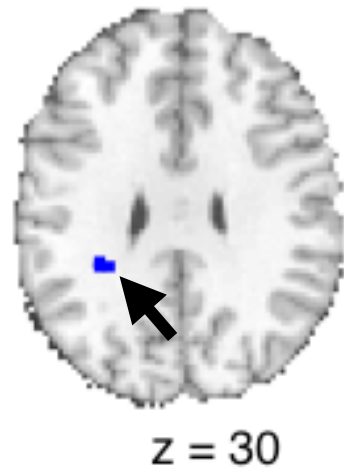
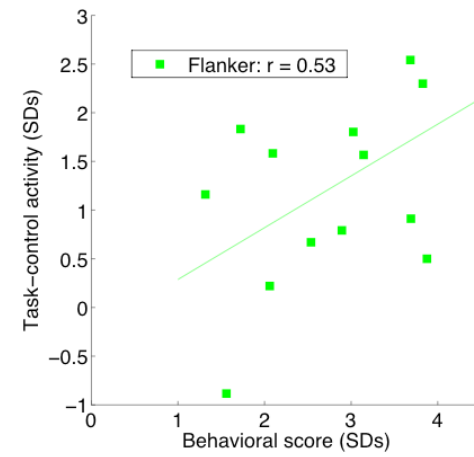
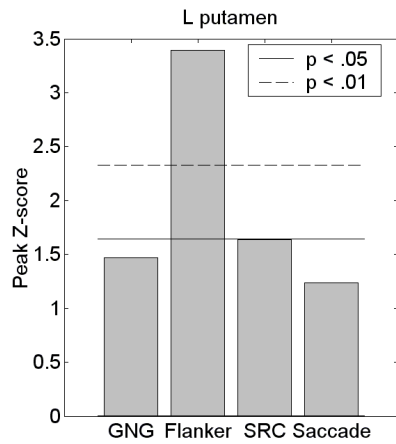
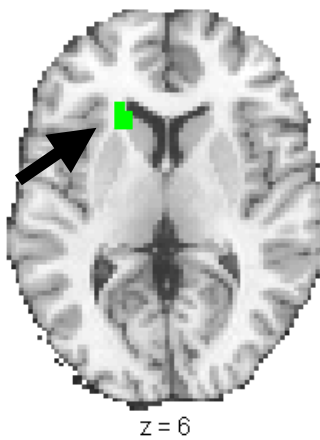
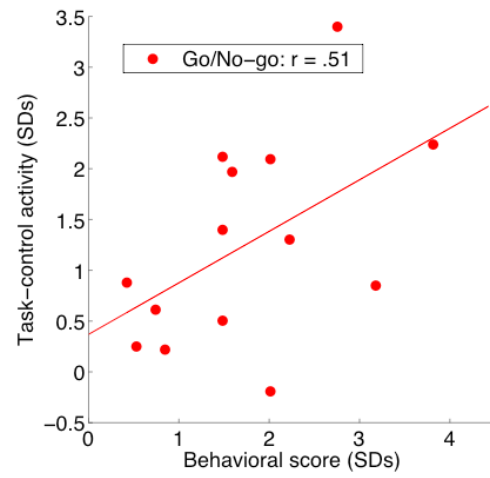
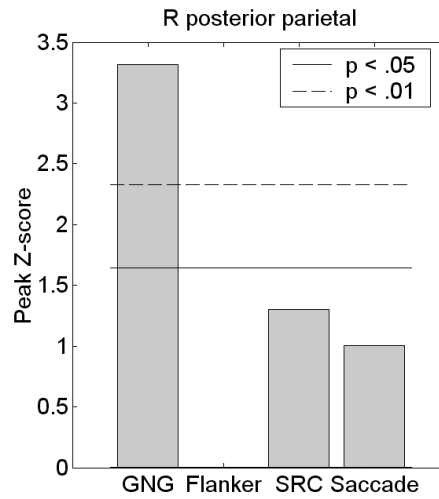
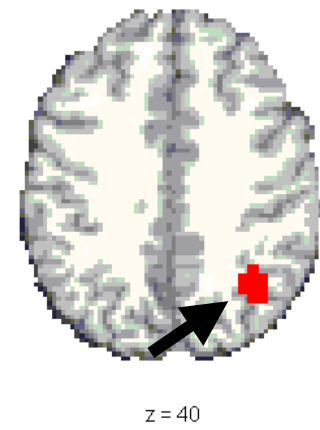


Figure 5.

