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Common and unique components of response inhibition revealed by fMRI

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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: a go/no-go task, a flanker task, and a stimulus–response compatibility task. Regions included bilateral anterior insula/frontal operculum and anterior prefrontal, right dorsolateral and premotor, and parietal cortices. Insula activity was positively correlated with interference costs in behavioral performance in each task. Principal components analysis showed a coherent pattern of individual differences in these regions that was also positively correlated with performance in all three tasks. However, correlations among tasks were low, for both brain activity and performance. We suggest that common interference detection and/or resolution mechanisms are engaged across tasks, and that inter-task correlations in behavioral performance are low because they conflate measurements of common mechanisms with measurements of individual biases unique to each task.

Keywords: Response inhibition; fMRI; Executive; Frontal

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 and Carr, 1994; Dejong et al., 1995; Anderson and Spellman, 1995; Kornblum et al., 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 and Requin, 1995; Logan, 1985; Logan and 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 presented with three tasks in which avoiding an inappropriate response was required. The three tasks varied 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), which, on some trials, required overcoming a compatible stimulus–response mapping in order to respond appropriately to each stimulus. For example, upon viewing an arrow pointing to the left, one might be required to either press a key with the left hand (compatible mapping) or the right hand (incompatible mapping). We compared responses in the incompatible mapping condition to those in the compatible one to isolate interference-resolution processes (including response inhibition) involved in this task. A second task involved the go/no-go paradigm (GNG), in which two types of stimuli were presented, one requiring a response and the other requiring the withholding of a response. Participants were instructed to respond as quickly as possible to each target stimulus – for example, the letter X– and withhold a response to a non-target stimulus (e.g., the letter Y). 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 (Eriksen and Eriksen, 1974). For example, a centrally presented color patch (blue or yellow) was mapped to a right-hand response. The two flanking color patches were mapped to the same response (congruent) or a different

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response (incongruent) as the target. In this task, we compared trials in which the flanking stimuli were mapped onto incongruent vs. congruent responses.

The type of response competition in this flanker task may be similar to that in the SRC task, but the differences may be important as well: incongruent flankers interfere with incorrect responses through a newly learned color–response association rather than an inherent compatibility effect, as in SRC tasks (Kornblum et al., 1990; Zhang et al., 1999). Also, spatial selective attention may be profitably used in the flanker task, as flankers are spatially distinct from the target, but they are not in the SRC task. Similarly, the inhibition required in the GNG task may be different from that of the flanker and SRC tasks as it involves the withholding of a prepotent response, rather the production of an alternate response.

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 et al., 1996; Merriam et al., 2001; Peterson et al., 2002; Schumacher and D'Esposito, 2002), GNG tasks (Casey et al., 1997; Klingberg and Roland, 1997; Konishi et al., 1999; Liddle et al., 2001; Menon et al., 2001; Rubia et al., 2001), and flanker tasks (Bunge et al., 2002a,b; Casey et al., 2000; Hazeltine et al., 2000). On the other hand, when examined behaviorally, the correlations in performance between one inhibitory task and another, while sometimes significant, generally explain little of the individual variability in these tasks (Burgess et al., 1998; Duncan et al., 1997; Fan et al., 2003; Miyake et al., 2000).

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" (i.e., interference-resolution) processes of interest. For example, 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. Thus, 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. Through our selection of tasks, we attempt to reduce the ambiguity in interpretation resulting from these multiple types of interference.

Common and unique types of interference resolution

Two features of our design help circumvent the abovementioned problems. First, by studying three tasks that share the 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/selection is ostensibly the only type of inhibition common to all three tasks.

In the GNG task, one has to inhibit the prepotent tendency to execute a response. This sort of inhibition may occur only at the response-selection or execution stages (Rubia et al., 2001), as there is only a single imperative stimulus that must be attended at any given time, and no prior learning builds an association between the "no-go" stimulus and either the "go" stimulus or a "go" response. Thus, there is little stimulus or response overlap that could lead to other forms of interference (Kornblum et al., 1990; Zhang et al., 1999).

In the flanker task, flanking stimuli are spatially distinct from the target, and incompatible flankers overlap with the target response but not with the target stimulus (Kornblum et al., 1990). Thus, inhibition may occur at two levels. First, one may inhibit the perceptual processing of the competing stimuli. However, should this type of inhibition fail, the competing stimuli may activate a competing response, which must be inhibited during response selection (van Veen et al., 2001). As this is the only task with spatially distinct stimuli that could be perceptually filtered (Broadbent, 1977; Mangun, 1995), regions uniquely activated by the flanker task may map most directly to perceptual selection.

In the incompatible trials of the SRC task, a prepotent tendency is developed through overlap between the automatic response elicited by the stimulus (e.g., left arrow) and the incorrect response (left button), competing with the correct response (right button) (Kornblum et al., 1990). Since interference resolution cannot occur during perceptual selection, it is likely to occur at the stage of response selection. However, it is also possible that rules directly compete, and priming of the incorrect rule or task set produces interference (Allport et al., 1994; Monsell et al., 2000; Rubenstein et al., 2001).

Therefore, although each task may recruit unique processes (response execution inhibition in GNG, perceptual inhibition in flanker, and stimulus–response mapping inhibition or rule selection in SRC), what these tasks share in common is inhibition at the level of response selection (e.g., Nee et al., 2004). 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 a few 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). These studies report low behavioral correlations among inhibition tasks, and they concluded that overlapping regions alone do not provide strong support for the existence of common mechanisms underlying inhibitory tasks. However, these studies did not correlate behavioral interference measures with regions of activation (but see Bunge et al., 2002a,b). Additional evidence about relationships between brain activity and behavioral performance could strengthen the case for common brain regions for interference resolution and provide leverage in interpreting the functional significance of overlapping activations. Beyond replicating and extending previous work, another critical

issue is an examination of the correlations between activation in these carefully isolated "common" regions with behavioral measures of inhibitory control. Our experiment was designed to address these issues, and in our analysis we specifically searched for regions that showed both activation and correlations with performance in each inhibitory task. In addition, we used connectivity analyses on the pattern of individual differences (e.g., Lin et al., 2003) to ask (a) whether activated regions are organized into coherent, distributed networks of brain regions showing similar patterns of task-related individual differences, and (b) whether these networks are the same across inhibitory tasks.

Methods

Participants

Fourteen 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 US$40 for their participation.

Behavioral tasks

There were 6 runs in the scanning session, two for each task. Each run was preceded and followed by a 20-s 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 s each, in BABA order, for a total of 12 blocks per run. One issue with using a fixed BABA order is that the predicted task effect is somewhat collinear with the effect of practice. In principle, a smooth decrease of activation with practice, without a true B–A effect, could produce a significant B–A result. However, high-pass filtering at 0.013 Hz removes any such linear trends, avoiding this potential confound.

We chose a block design because it maximizes power to detect stable effect magnitudes (Liu et al., 2001; Wager and Nichols, 2003), sacrificing knowledge provided by event-related designs about whether activations reflect general task-set-related or specific trial-related processes in favor of maximizing stability of effects for individual differences analyses. However, the GNG task was analyzed as an event-related task because we expected activation in interference-processing regions to be elicited by "no-go" trials. These trials were distributed across blocks containing different relative "no-go" trial densities.

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 below.

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 12 fixation crosses were presented. Whenever the participants saw any letter other than X, they made a keypress with their right index finger; each time they saw an X, they were not to make any response. Each of the letters subtended approximately 2° of visual angle and appeared for 440 ms; letters were separated by a 1000-ms central fixation cross. There were a total of 288 trials.

Although the GNG task included blocks with high (50%) and low (20%) no-go trials, activation in interference-resolution regions is expected to occur for "no-go" trials in both blocks. This design, which was fundamentally different from the other tasks, required an event-related analysis, with separate regressors for "go" and "no-go" trials. The critical contrast, and the measure of response-selection difficulty in brain activity, was the subtraction of "no-go"–"go" trials. The interference measure used to assess behavioral performance was the false alarm rate (FAR) for no-go trials.

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. Although using an event-related design prevents us from directly comparing the magnitude of activation in the GNG task with magnitudes for the other tasks (Wager et al., 2005), this design does not preclude looking for correlations between brain activity and performance within each task. In addition, we may test for differences in reliability of activation across tasks (see Image Analysis section). Notably, the GNG task employed a rapid event-related design with clustered events, which is expected to maximize power (Liu, 2004), so experimental power is expected to be relatively consistent across all tasks.

Flanker task

We used a task similar to that of Hazeltine et al. (2000). 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. There were 12 trials per block for a total of 288 trials. As 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° 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. A total of 242 trials were presented. 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

One issue is whether activation in the inhibition tasks is reducible to differences between actual and intended eye movements. While previous studies of inhibition tasks have not raised this issue, the brain regions involved in spatial attention appear to be very similar to those involved in generating saccades (e.g., Corbetta et al., 1998). Thus, in inhibition tasks, spatial attention might be more heavily recruited during incompatible/incongruent conditions, as participants may covertly focus on different parts of the stimulus (e.g., the tails of the arrows) to avoid interference. In this study, we chose to avoid some problems in interpretation by mapping saccade activity at a low threshold and excluding it from analysis. Thus, common regions we report are likely to be activated by processes unique to interference resolution rather than being basic mechanisms of directed attention. To achieve this goal, 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 on the screen. Six blocks of saccade-trials alternated with 18 s 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 = 1000 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 et al., 1999) and corrected for head movement using the realignment routines in the Automated Image Registration (AIR) package (Woods et al., 1998). Subsequent preprocessing and analysis was done using SPM99 (Wellcome Department of Cognitive Neurology, London). Individual SPGR images were corrected for signal inhomogeneity (G. Glover and K. Kristoff, http://www-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 canonical 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 et al., 2002) of $P < 0.05$ 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 (http://www.mrccbu.cam.ac.uk/Umaging/mnispace.html). Brodmann areas were identified using the Talairach atlas (Talairach and Tournoux, 1988) as implemented by the Talairach Daemon (Lancaster et al., 2000; http://www.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, $P < 0.05$). We then classified voxels in this set of regions into the following categories of interest.

Common regions were determined to be areas that showed activation in all three tasks at $P < 0.05$ (uncorrected). This threshold has been used in previous studies (e.g., Fan et al., 2003) and requires a minimum t value of 2.16 in each of the three tasks. Even using a threshold of uncorrected $P < 0.05$, the threshold is higher than a required minimum t value of 0.56 for an SPM99 conjunction analysis, which does not require each test to be significant individually. The conjunction threshold is

low because the conjunction analysis tests the global null hypothesis that all tasks do not activate the voxel against the alternative that one or more tasks activate the voxel (Brett et al., 2004; Nichols et al., in press). For this reason, we prefer the intersection test we employed here to the SPM99 conjunction test. In addition to reporting common regions at $P < 0.05$ uncorrected in each task, for completeness, we separately report the smaller set of common regions significant at $P < 0.05$ FDR-corrected in each task.

Unique regions were determined to be areas that met three qualifications: (a) activation (FDR-corrected, $P < 0.05$) in only one task, (b) significantly more reliable activation in that task than the next most reliable task, and (c) no activation in the other two (or the saccade task) even at the lower threshold of $P < 0.05$ uncorrected. Importantly, although identifying such regions is important for making inferences about differences among executive functions, the limited power in this study makes identification of unique regions exploratory only at this point, and further studies must confirm the results we present here. Identifying activated regions in this manner provides stronger evidence for qualitative differences in activation among tasks (e.g., active in flanker but not the other tasks). Such qualitative differences can more readily be interpreted as identifying unique processes (Sternberg, 2001).

Since we cannot directly compare activation magnitudes between the GNG task (event related) and the other tasks (blocked) as the regressors for events and blocks may have different scales (Wager et al., 2005), one solution is to compare reliability scores (Z scores) across the different tasks. The difference between two Z scores is distributed with zero mean and a standard deviation of $\sqrt{2}$. Thus, the test statistic is the minimum difference between Z scores for the "uniquely activating" task and all other tasks, multiplied by $\sqrt{2}$, and this statistic is compared with a normal distribution to obtain two-tailed P values. We refer to this quantity as the minimum Z difference, and a significant value indicates more reliable activation, in the sense of lower between-subjects error, in one task than the others.

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 and Van Driessen, 1999). 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 r estimates differed by more than 0.4. 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 (GNG, flanker, and SRC), 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 GNG task, the false alarm rate (FAR) on no-go trials was used as the measure of interference. (Reaction times were not available because, of course, responses were not made on no-go trials.) The FAR is the proportion of no-go trials on which participants failed to inhibit the prepotent response and pressed the button. FARs were significantly greater during low no-go ($\bar{x} = 0.19$) than high no-go blocks ($\bar{x} = 0.03$; $\bar{x}$ difference = 0.17, $\sigma = 0.094$, $t(13) = 6.57$, $P < 0.0001$), although the differences between high-percentage and low-percentage blocks were not used in individual differences analyses. The individual differences measure in performance was the overall no-go FAR, and the measure in brain activity was the overall no-go-go event-related contrast. 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, $t(13) = 10.6$, $P < 0.0001$) and this difference was used as the interference score. Accuracy showed effects in the same direction as in the go-no-go task, with higher accuracy in the low conflict blocks (92.4% and 96.8% for incongruent and congruent blocks, respectively, $t(13) = 3.28$, $P < 0.05$).

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, $t(13) = 7.03$, $P < 0.0001$). 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 (experimental-control RTs for SRC and flanker, and FAR in low-high for GNG) revealed negligible relationships among performance on the three tasks: for GNG and flanker, $r = -0.17$; for GNG and SRC, $r = -0.25$; and for flanker and SRC, $r = 0.12$, 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 greater task interference, and thus less efficient inhibition. 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 Fig. 1. Activated areas, corrected for multiple comparisons ($P < 0.05$) using FDR (Genovese et al., 2002) in random effects analyses,

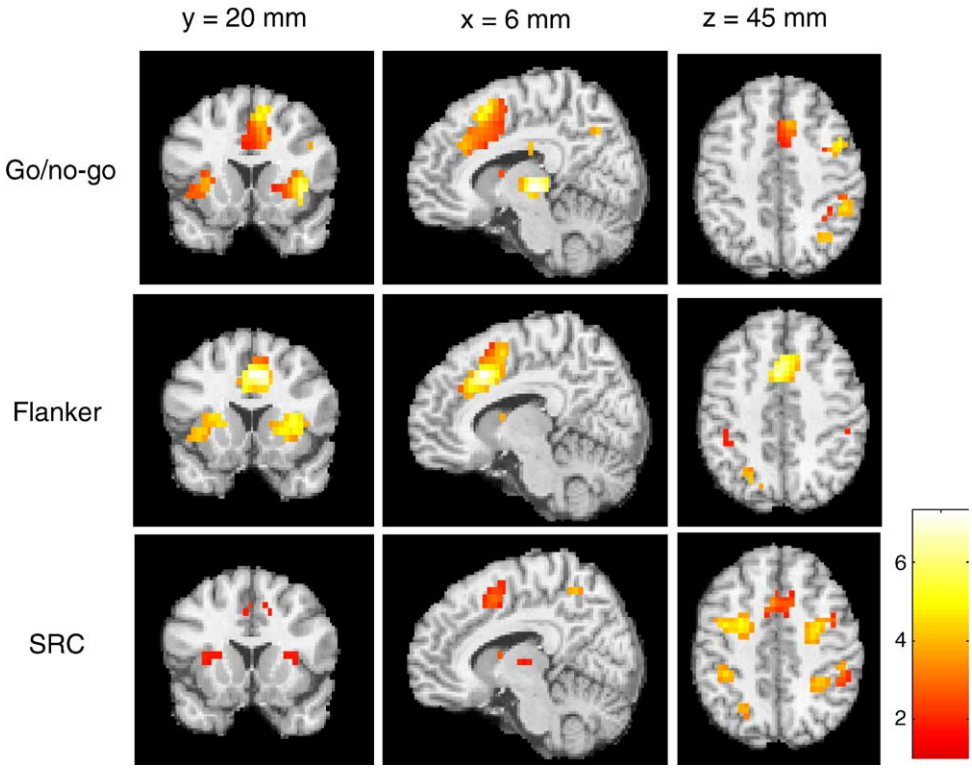

Fig. 1. Slices showing activations in each task: go/no-go (top row), flanker (middle row), and stimulus–response compatibility (bottom row). All regions shown were significant at $P < 0.05$ corrected using False Discovery Rate control in at least one task. Regions significant at $P < 0.05$ (uncorrected) in all three tasks were considered common inhibitory regions. The display threshold is $P < 0.05$ uncorrected.

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) thalamus (please see Table 1 for specific areas activated in each task). FDR corrected significance thresholds were $t > 3.47$ for flanker, $t > 3.60$ for GNG, and $t > 3.50$ for the SRC task.

Saccade task activations

We wanted to rule out the possibility that inhibitory activations were related to differences in utilization of basic spatial attention or eye movements. 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 ($P < 0.05$ uncorrected, shown in cyan in Fig. 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 specified threshold in all three tasks. These regions, listed in Table 2, included bilateral anterior insula and anterior PFC, right DLPFC, left caudate, posterior intraparietal sulcus (IPS), and right anterior IPS. Three regions (Table 2) were also significant at $P < 0.05$ FDR-corrected in each task: anterior cingulate (extending into the right superior frontal sulcus) and bilateral caudate/putamen.

Correlations between activations in these common regions and reaction time performance (or false alarms for GNG) indicated that among all of these regions, only the anterior insula (bilaterally) showed significant correlations between activation and behavioral performance in all three tasks. However, the lack of significant correlations in other regions is inconclusive, as the small sample size used in this study necessarily affords low sensitivity to true correlations; it could be that the true correlation value is consistently positive across all regions. We proceed to describe and interpret regions that did show significant correlations. The minimum brain activity–performance correlation for any task in left or right insula was $r = 0.46$, with a critical r of 0.53 ($P < 0.05$, two-tailed) or 0.46 ($P < 0.10$, two-tailed) for a marginal trend. Robust correlation values ranged from $r = 0.61$ to 0.76 for each task, all $P < 0.05$. At most one outlier was removed from each correlation. Fig. 3A shows the common region in right insula. Fig. 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. Fig. 3C shows robust correlations between behavioral performance measures (x-axis, higher scores reflect more task interference) and activation in the right insula (y-axis), both expressed in units of standard deviations (SDs, e.g., performance scores divided by the SD of performance scores across subjects) for easy comparison across tasks. SD scores are equivalent to z scores, but without subtracting the mean response, so that the mean activity and performance levels are shown. These correlations were significant for each of the three tasks in both right and left anterior insula. (Note: details for the left insula are not shown in Fig. 3 for space reasons but revealed the same pattern of results as

Table 1
Regions activated at whole-brain FDR corrected thresholds ($P < 0.05$) in each task

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

the right insula; peak activation coordinates and correlation values for both are in Table 2.) Figs. 3D and E are described in the Network analysis section below.

Unique regions

We next classified voxels as belonging to unique regions if they (a) were significant at corrected $P < 0.05$ in one task, (b) showed no evidence for significance in the other tasks at a lower threshold (uncorrected $P < 0.05$), and (c) showed more reliable activation in that task than any of the others.

Regions were found that met or approached significance on these criteria for each of the three tasks. Candidate regions that met the first two criteria are shown in Fig. 2, in red (GNG), green (flanker), and blue (SRC), and described in Table 3. Few of these regions met the third criterion of greater reliability of activation in one task than the others in a direct comparison (see the Min Z diff column in Table 3). Also of note, these candidate regions were almost always adjacent to common regions (yellow in Fig. 2) or other regions active in multiple tasks (black in Fig. 2); and finally, uniquely activated regions frequently showed evidence for correlations between activity and performance for multiple tasks, as listed in Table 3. Collectively, these considerations weaken the argument for task-specific processing in these areas.

However, some of these regions were consistently task specific in our meta-analysis and are partially confirmed here, and we discuss the most likely candidates for task-unique regions below.

In the GNG task, unique regions that met all criteria were found in thalamus, right inferior parietal cortex, and right anterior prefrontal cortex (Table 3). Of these, only the thalamus showed activity-performance correlations only with the GNG task; other regions showed correlations in other tasks, weakening the argument for task-unique activation in these regions. The left panel of Fig. 4A shows the location of the thalamic region uniquely activated in the GNG task. The center panel shows reliability of activation for each task averaged across voxels in the region and indicates that none of the flanker, SRC, or 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 go-no-go task (y-axis), indicating that participants with higher interference scores showed more activation in the right parietal cortex. The right posterior parietal cortex also showed significant activity–performance correlations only in the GNG task, although it did not meet all criteria for a task-unique region; we mention it because a nearby region derived from meta-analysis (see below) did meet all criteria.

In the flanker task, no regions met all criteria for unique regions, although the anterior cingulate approached significance on the comparison of activation reliability (Table 3; min Z difference = 1.81, $P > 0.10$). Of the candidate regions, the left caudate/putamen (Fig. 4B) showed a significant activity–performance correlation for the flanker task alone ($r = 0.53$, $P < 0.05$), although the correlation for SRC showed a trend towards significance ($r = 0.47$, $P < 0.10$).

In the SRC task, only the right premotor cortex met the criteria for a unique region, although it was not significantly correlated with performance in any task, and correlations approached significance for the flanker task ($r = 0.49$, $P < 0.10$; see Table 3). The only region that was correlated with performance only in the SRC task was in white matter near left inferior parietal cortex; the functional significance of this activation is unclear. A portion of the anterior cingulate cortex was also correlated with SRC performance, but it was also negatively correlated with performance in the GNG task.

Network analysis

Correlations among individual activation scores in common regions (Table 2) were positive in each task, indicating that participants with high activation in one region tended to show high activation in the other regions on the same task. Principal

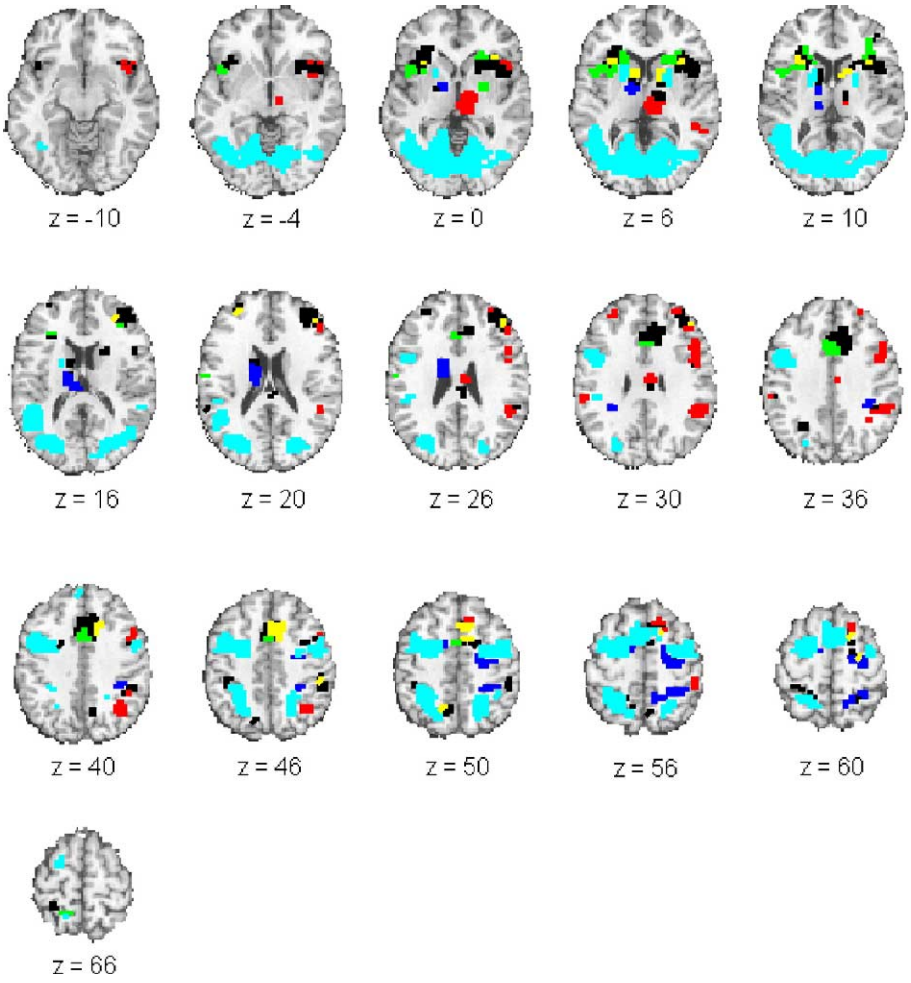

Fig. 2. Common and unique regions in inhibition tasks. All regions shown are significant $P < 0.05$ corrected using False Discovery Rate control in at least one task. Yellow regions are significant in all tasks at $P < 0.05$ (uncorrected). Red: significant activation only in go/no-go. Green: significant 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 ($P < 0.05$ uncorrected). Saccade regions were excluded from further analysis.

components analysis (PCA) scores revealed a single common principal component in each of the three tasks, tested for significance using a permutation test. Eigenvalues were 6.00 for GNG, 5.18 for flanker, and 6.18 for SRC, all $P < 0.001$, with no other significant eigenvalues. Common regions were considered part of the "network" in each task if they correlated at least $r = 0.53$ ($P < 0.05$) with the principal component. We focus on regions, shown in Fig. 3D, that were found to be part of the common network in each of the three tasks. However, the full set of networked common regions (that loaded highly on the principal component) for each task is shown in the last three 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 for each task were calculated as the weighted average across all common regions for each participant, where the weights are the first eigenvector for that task. Thus, highly inter-correlated regions are given more weight in the average, and the component scores are thus a measure of how strongly a participant activated the network on each task. Component scores were then correlated with behavioral performance for each task and were shown to be positively correlated in each case, $r = 0.37$, n.s. for GNG, $r = 0.57$, $P < 0.05$ for flanker, and $r = 0.55$, $P < 0.05$ for SRC. Robust correlation values were higher and statistically significant in each case; these are plotted in Fig. 3E. Direct comparisons between Pearson's correlations and robust correlations can be found in Table 2.

We next tested whether behavioral performance in 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 a multiple regression, with performance on each task as the dependent variable and one regression analysis per task. This allowed us to examine whether behavioral 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 $r = 0.57$, $P < 0.05$ for GNG, $r = 0.56$, $P < 0.05$ for flanker, and $r = 0.60$, $P < 0.05$ for SRC. The only exception was that GNG activation predicted SRC performance, partial $r = 0.54$, $P < 0.05$, 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 (e.g., high insula activation in Flanker is correlated with high

Table 2
Regions activated by all three inhibition tasks

Common regions	BA	*x*	*y*	*z*	Vol	Activity (Peak *Z* score)				Correlations (Pearson's *r*)			Robust correlations			Network affiliation		
Region						Go/no-go	Flanker	SRC	Saccade	Go/no-go	Flanker	SRC	Go/no-go	Flanker	SRC	Go/no-go	Flanker	SRC
Common regions (FDR corrected in each task)																		
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	0.64	0.90
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		
Common regions ($P < 0.05$ uncorrected in each task)																		
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	0.75	0.85
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	0.90	0.85
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	0.73	0.78
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	0.76	0.83
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		0.88
R superior	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	0.71	0.73
frontal sulcus	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	0.82
R premotor cortex	6	26	−11	60	70	1.86*	1.70*	3.89*	0.95	−0.09	0.53*	−0.46**	−0.08	0.78*	−0.78*			
R inferior parietal cortex	40	49	−34	45	70	2.49*	1.75*	3.15*	0.65	0.17	0.35	0.39	0.17	0.72*	0.39		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	0.68	

P values are based on a one-tailed test for activation and a two-tailed test for correlations. The critical Pearson's *r* with Bonferroni correction for the 10 common regions is 0.69, and for correction across regions * tasks is 0.74. Common regions at the lower, uncorrected threshold met the additional restriction of corrected significance in at least one task. Vol indicates the number of cubic mm of brain tissue activated in common. 70 mm of brain tissue equals one 3.75 × 3.75 × 5 mm voxel (used in this study), or 8.75 standard 2 × 2 × 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 (i.e., the component scores); a correlation of $r > 0.53$ ($P < 0.05$, two-tailed) was chosen as the cutoff for considering a region to be part of the network for that task.

* $P < 0.05$.

** $P < 0.10$.

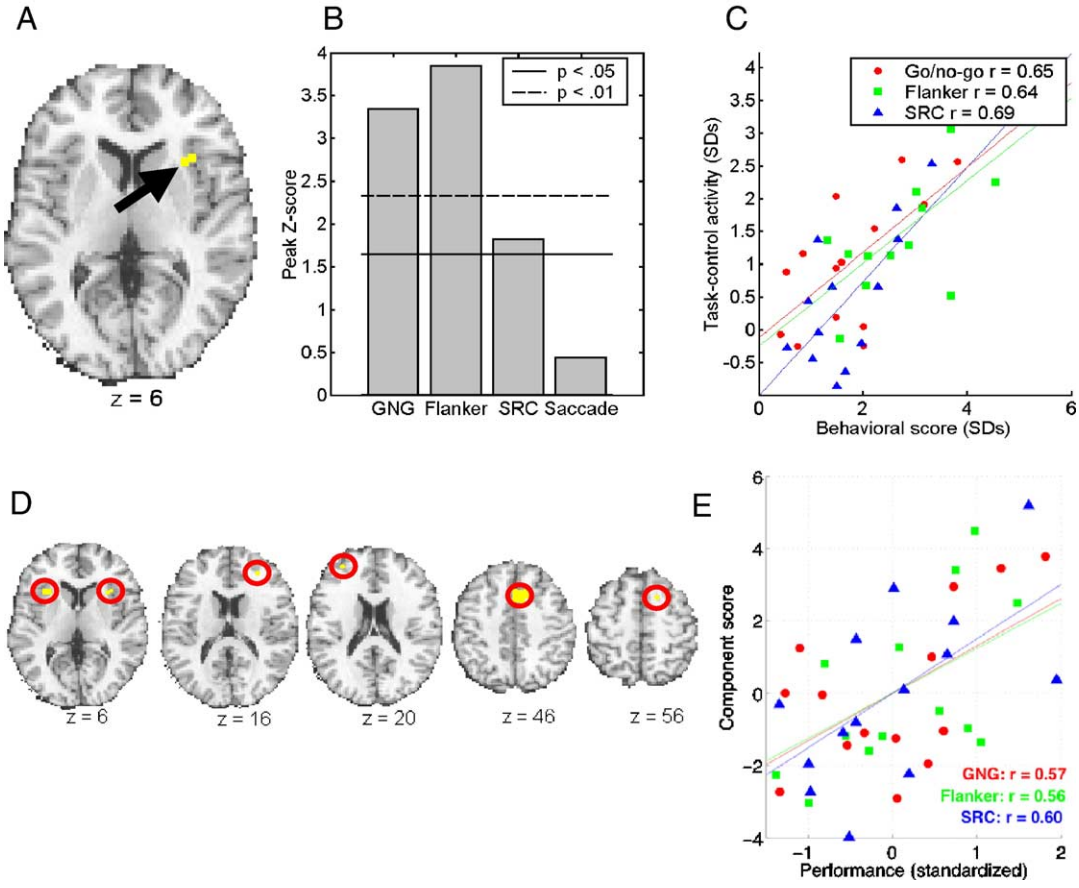

Fig. 3. Right anterior insula and correlations with task performance. Left anterior insula (not shown) also showed consistent positive correlations between task performance and brain activity across the three inhibition tasks. (A) Slice showing the location of the common anterior insula region. (B) Bar plot 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.

insula activation in SRC and GNG). 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 0.006 and 0.10, 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 and Requin, 1995). We return to this point in the Discussion section.

Meta-analysis regions

A previous meta-analysis of reported peak brain activations from these same inhibitory tasks has isolated a set of regions that the tasks may share in common (Nee et al., 2004). This was accomplished using a density analysis method that is described in detail elsewhere (Wager et al., 2003, 2004). This meta-analysis revealed regions consistently activated by interference tasks in previous studies, summarized in Table 4. Using sets of contiguous voxels in these regions, we created regions of interest (ROIs). Analysis of the average activity in these ROIs corresponded closely with results from the analyses presented above. Two of the meta-analysis regions – right inferior frontal gyrus and anterior cingulate – were active in all three tasks, paralleling findings from the common-region analyses.

We also performed separate meta-analyses of reported peak activations from previously published studies of each task. We did this because it is possible that reported activations are highly consistent in one task, but not in others, diluting the statistical significance in the overall across-task meta-analysis. Table 4 shows regions activated in each separate-task meta-analysis. Although most of these were also identified in the overall analysis, regions in the intraparietal sulcus were identified in the SRC meta-analysis only.

Table 3
Regions activated by only one task

Region	BA	x	y	z	Vol	Activity (Peak Z score)				Min Z diff	Correlations (Pearson's r)			Robust correlations		
						Go/no-go	Flanker	SRC	Saccade		Go/no-go	Flanker	SRC	Go/no-go	Flanker	SRC
Go/no-go unique regions																
Thalamus	–	8	−23	0	2602	4.52*	1.06	1.58	0.27	2.08*	0.65*	−0.35	−0.07	0.76*	0.11	0.07
R anterior PFC	10	30	45	30	352	3.98*	1.15	0.23	−1.16	2.00*	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	2.02*	0.03	−0.27	0.01	−0.35	−0.86*	0.43
Mid-cingulate	23	4	−19	30	563	3.53*	1.04	0.28	−2.88	1.76**	0.62*	−0.37	0.62*	0.57*	−0.37	0.61*
R DLPFC	9	45	8	30	2602	3.91*	0.04	1.6	1.42	1.63	0.18	−0.09	0.36	0.18	0.62*	0.50**
L inferior parietal	40	−60	−34	30	281	3.55*	0.63	1.2	1.3	1.59	−0.25	0.16	0.14	0.02	0.59*	−0.42
R SFS	6	15	19	55	703	3.71*	1.47	1.49	1.26	1.57	0.11	0.15	0.70*	0.11	−0.03	0.70*
R posterior parietal	7/40	34	−64	40	1125	3.31*	0	1.3	1	1.42	0.51**	−0.39	0.18	0.51**	−0.11	0.14
R DLPFC	46/9	45	30	25	422	3.43*	1.39	1.56	−2.85	1.32	0.41	0.23	0.47**	0.41	−0.11	0.2
R temporal cortex	21/41	49	−45	5	352	3.43*	0.53	−0.14	1.56	1.32	−0.08	0.21	0.01	−0.07	0.03	0.37
L DLPFC	9	−30	41	30	211	3.29*	1.58	1.03	−2.38	1.21	0.35	−0.2	0.08	−0.13	0.15	0.56*
R sensorimotor cortex	40/2	45	−38	55	211	3.25*	1.35	1.59	−0.15	1.17	0.11	0.50**	−0.06	0.11	0.24	−0.12
Flanker unique regions																
Anterior cingulate	24/32	0	8	40	1688	1.63	4.19*	1.53	0.09	1.81**	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	1.53	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	1.46	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	1.44	0.29	0.1	0.01	0.29	−0.73*	0.01
L Caudate/putamen	–	−23	23	10	773	1.47	3.39*	1.64	1.23	1.24	0.19	0.53*	0.47**	0.19	0.53*	0.47**
SRC unique regions																
R premotor cortex	6	23	−19	55	2602	1.05	1.59	4.45*	1.56	2.02*	0.05	0.49**	0.11	0.05	0.49**	−0.38
R sensorimotor	40/7	26	−45	50	2883	1.59	1.4	4.03*	1.6	1.72**	0	0.38	0.14	0	0.38	−0.19
Thalamus/caudate	–	−15	−15	15	3867	1.27	1.63	3.78*	1.27	1.52	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	1.19	−0.24	0.14	0.41	−0.57*	0.14	0.80*
White matter (near inferior parietal)	40	−34	−45	30	211	−0.42	0.18	3.16*	1.48	1.19	−0.15	−0.1	0.44	−0.15	0.39	0.74*

Regions with significant Min Z diff values, which compare the reliability of activation among tasks directly, meet the criteria for uniquely activated regions. Min Z diff values are the difference between z scores for the most activated task and the second most activated task, divided by $\sqrt{2}$ so that they are normally distributed. Other column labels are as in Table 2.

* $P < 0.05$.

** $P < 0.10$, 2-tailed.

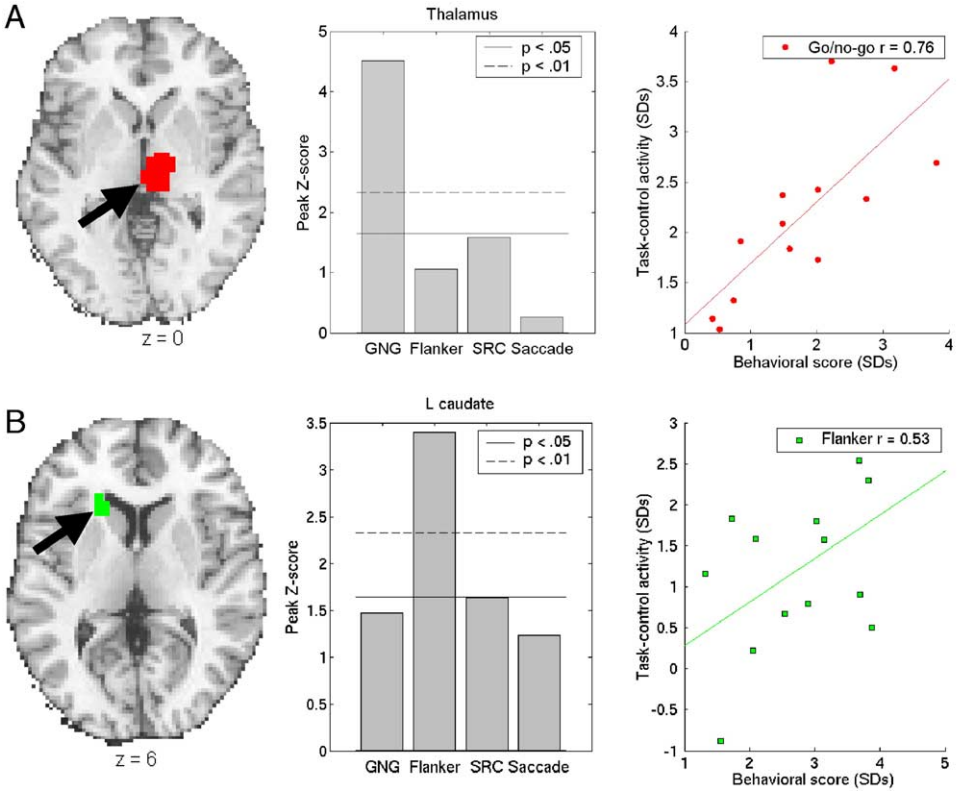

Fig. 4. Selected unique regions showing correlations between activation and task performance. (A) Shows thalamus (left panel) with corresponding activations for the four tasks (center panel) and the correlation scatterplot for z scores of false alarm rate against activation in the go/no-go task (right panel). (B) Left caudate (left panel), activations in each task within this region (center panel), and the scatterplot showing z scores of flanker interference cost against activation in the flanker task (right panel).

The left IFG/insular meta-analysis region was 7 mm rostral and 10 mm superior to the common left insular region reported above, and was uniquely activated and correlated with performance in the flanker task, as shown in Figs. 5F–H. This result parallels the anterior spread of activation only in the flanker task (Fig. 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 in the present study, and thus paralleled results from the previous unique region analysis, as shown in Figs. 5C–E. The results of the meta-analysis thereby support the conclusions that (1) most common regions in the meta-analysis are commonly activated in each task in the present study; (2) GNG may activate a separable region of the right angular gyrus; and (3) The flanker task may produce greater activation (in magnitude or extent) in left IFG.

Discussion

This study demonstrates that diverse inhibitory tasks – one that required withholding of a response (GNG), 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 across participants in each task, suggesting that the network is a functional unit of analysis for individual differences. Activation in an inter-correlated network of regions for each task (which always included, but were not always limited to, the regions listed above), and in the bilateral insula individually, tracked behavioral performance in each task. These findings suggest that the common 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 common regions found in this study corresponded well with those reported as common activations in previous studies (Fan et al., 2003) and in a 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 et al., 1998; Fan et al., 2003; Miyake et al., 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

Table 4
Summary of results from the inhibition meta-analysis regions

Meta-analysis regions	BA	*x*	*y*	*z*	Vol	Activity (peak *Z* score)				Min *Z* diff	Correlations (Pearson's *r*)			Robust correlations		
Region						Go/no-go	Flanker	SRC	Saccade		Go/no-go	Flanker	SRC	Go/no-go	Flanker	SRC
Meta-analysis collapsing across tasks																
R DLPFC	9/46	41	19	25	8930	3.55*	3.41*	1.88*	0.31	0.10	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	1.20	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	0.86	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	1.17	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	2.69	0.51**	−0.34	0.25	0.51	−0.02	0.25
GNG-only meta-analysis																
R DLPFC	9/46	41	23	25	15258	6.69*	4.86*	2.37*	0.31	1.29	0.23	0.15	0.26	0.23	0.45	0.00
R angular gyrus	39	41	−64	35	1688	3.18*	−0.85	0.13	−1.14	2.16	−0.11	−0.46**	−0.18	−0.11	−0.19	−0.34
Flanker-only meta-analysis																
R DLPFC	9/46	41	15	30	4781	3.48*	2.30*	1.88*	−0.09	0.83	0.14	−0.07	0.34	0.44	−0.07	0.34
SRC-only meta-analysis																
L posterior IPS	7	−11	−71	40	563	1.54	1.73*	−0.37	−0.52	1.44	0.19	−0.42	−0.06	−0.29	−0.42	−0.06
R posterior IPS	7	19	−60	45	281	1.96*	0.64	2.60*	2.93*	1.39	−0.01	−0.19	0.15	0.07	0.00	0.15

Meta-analysis identified regions, listed at the left of the table, in which previous study peaks were sufficiently dense to be considered non-randomly distributed throughout the brain. The top rows show significant regions collapsing across tasks, which may miss clusters of peaks that appear only for one task. The rows below show regions significant in a meta-analysis of only peaks from a single task-type. R DLPFC—Right Dorsolateral Prefrontal Cortex; MPFC—Medial Prefrontal Cortex; IFG—Inferior Frontal Gyrus. Min *Z* diff: see Table 3 legend.

* $P < 0.05$.

** $P < 0.10$, 2-tailed.

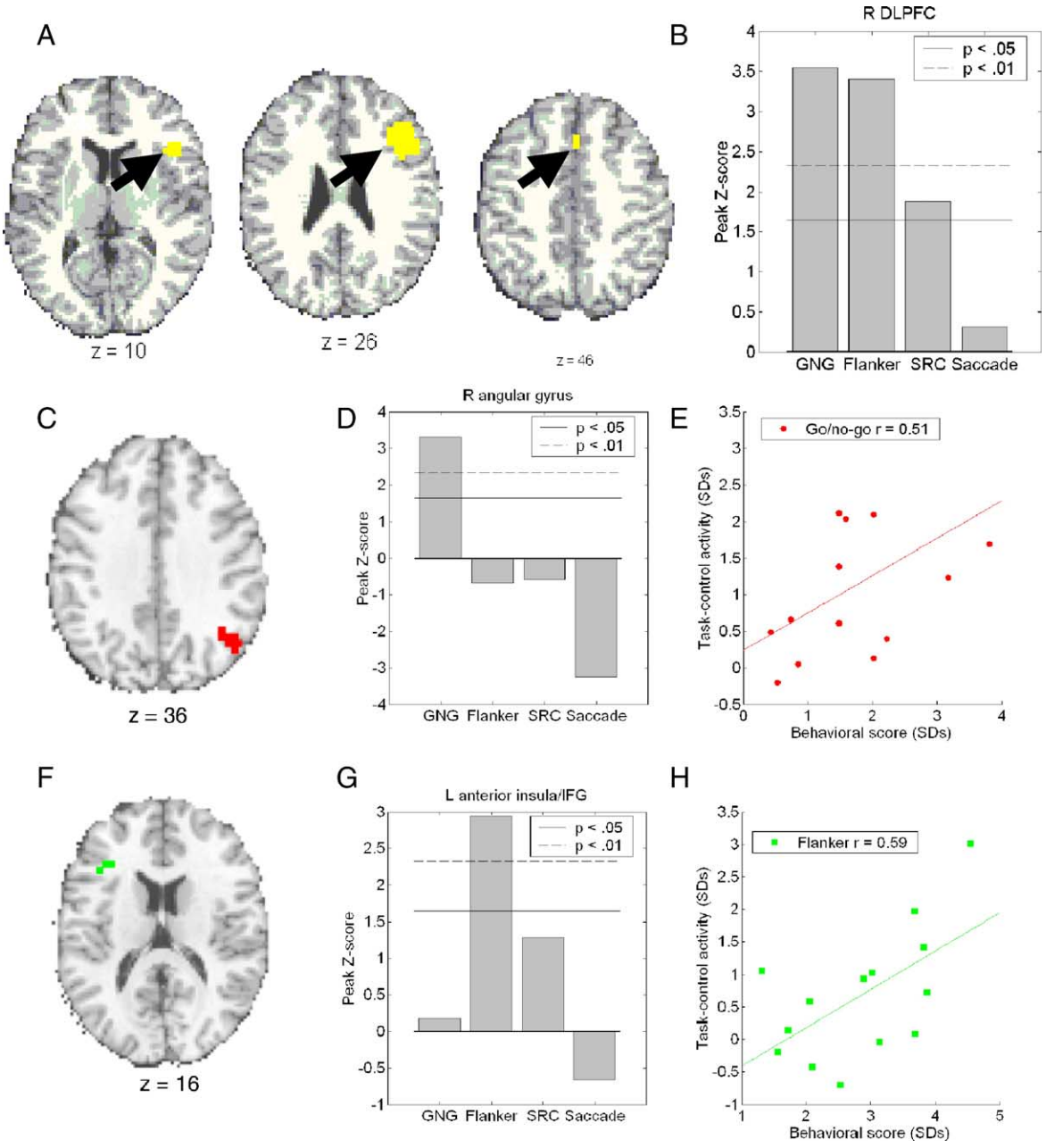

Fig. 5. Analyses of selected regions derived from meta-analysis. (A) Meta-analysis regions in right anterior insula/frontal operculum (left panel), right dorsolateral prefrontal (DLPFC, middle frontal gyrus) and inferior frontal gyrus (center panel), and anterior cingulate (right panel). (B) Activations for the DLPFC/inferior frontal gyrus region in all four tasks. (C) Right posterior parietal meta-analysis region. (D) Activations for all tasks in the posterior parietal region. Activation was significant only for go-no-go. (E) Correlation plot for z scores of false alarm rate in the go-no-go task against activation in the go-no-go task. No other correlations were significant. (F) Left anterior insula/inferior frontal gyrus meta-analysis region. (G) Activations in this region for all tasks, significant only for the flanker task. (H) Correlation plot for z scores of interference in the flanker task against activation in the flanker task. No other correlations were significant.

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.

Another highly relevant previous study was conducted by Bunge et al. (2002a,b), who found that activation of the inferior frontal gyrus and anterior insula were negatively, rather than positively, correlated with susceptibility to interference. While this result reinforces the idea that activation in these regions are functionally related to performance, it raises the question of why the present study and Bunge et al. reported correlations of opposite sign. Perhaps the most obvious alternative is that the present study used a block design, and so activation may reflect both trial-specific processes and sustained control-related processes (Braver et al., 2003). Bunge et al., by contrast, used a rapid event-related

design, which preferentially detects trial-related processing but also requires more assumptions about the shape of the hemodynamic response. An alternative explanation is that Bunge et al. scaled individuals' brain activation scores to have the same global value—thus, the correlations reflect residuals from the global activity, which may itself partially reflect activation of a broad network of brain regions. The present study used raw scores. Future studies must disentangle both transient/sustained activation and global (widespread) vs. local contributions to measures of regional activity.

Interpreting positive correlations

A subset of regions activated in common across the three inhibition tasks used in our study – the bilateral anterior prefrontal cortex, anterior cingulate, and bilateral anterior insula – shows a consistent pattern of activation that is positively correlated with behavioral interference in all three inhibition tasks. This finding suggests that these brain regions play some common functional roles in cognitive control.

How, though, should we interpret the fact that these correlations are positive? After all, it seems most natural to suppose that a region that is involved in the resolution of conflict should show increases in activation when the behavioral manifestations of conflict are reduced. Take the flanker task as an example. If we believe that the activated regions in this task are involved in the resolution of interference, then greater activation might be expected to be associated with reduced interference. For instance, suppose that as these regions are engaged, they reduce the effect of the surrounding flankers so that the subject's response can be governed by the central target item. Therefore, increased recruitment of these areas would lead to reduced interference. The positive correlations we have found belie this simple account.

One explanation for positive brain–behavior correlations is that positively correlated regions implement a monitoring process that increases with greater input conflict. It has been suggested that the anterior cingulate serves a monitoring function, integrating activity from relevant and irrelevant processing streams and, by virtue of the high level of crosstalk created by conflicting inputs, triggering engagement of lateral prefrontal control mechanisms (Cohen et al., 1990; MacDonald et al., 2000; Miller and Cohen, 2001). The frontal network activated in our study that correlated positively with behavioral interference includes the anterior cingulate. Perhaps, then, the cingulate is only part of a larger circuit that is involved in the detection of conflict, augmented by the additional structures that we have found to be positively correlated with behavioral interference effects. By this account, what this circuit does is to detect the presence of conflicting input, signaling downstream mechanisms that further processing will be needed before a response can be programmed.

However, a positive correlation need not imply monitoring rather than implementation of control. Because interference-resolution mechanisms are activated by putative conflict detection mechanisms, both are expected to be more active in participants who have more interference to contend with in the first place. It is only when the amount of conflict experienced is equated across participants, equating input into interference-resolution mechanisms, that negative correlations between activation of these mechanisms and behavioral performance costs are expected to emerge. As we describe below, there is ample evidence that conflict may not be equated across tasks. In fact, the finding of positive correlations in putative "interference-resolution" regions suggests that the major determinant of a person's behavioral cost is not how efficient their interference-resolution mechanisms are, but how difficult a particular task is for them in the first place.

How is the level of conflicting input to detection/resolution mechanisms determined? In current computational accounts of interference tasks, conflict results from strong activity in irrelevant processing pathways as compared to relevant processing pathways (Cohen et al., 1990; Jones et al., 2002; Kornblum et al., 1990; MacLeod and Dunbar, 1988; Zhang et al., 1999). Thus, the level of conflict experienced by a subject may vary from trial to trial, based on attentional set and recent task experience, and from task to task, based on efficiency of processing pathways specific to each task. In the flanker task, the variation in conflict is caused by stochastic differences in the relative intrusiveness of the flankers and/or the efficiency of spatial attention. In the GNG task, it is caused by variations in confusability of the "go" and "no-go" cues and/or variations in tradeoffs between being fast and withholding appropriately. In the SRC task, it is caused by variation in the strength of the prepotent compatible response tendency.

Behavioral correlations and implications for behavioral individual differences studies

If there are common mechanisms involved in resolving interference, why is behavioral performance uncorrelated across the various tasks that measure this interference resolution? This is a critical question. We suppose that there are several reasons why common inhibitory mechanisms may not be detectable in analysis of behavioral scores alone.

The typical model underlying behavioral individual-differences studies is that the behavioral scores (e.g., RT cost for the interference minus the control condition) reflect a single cognitive measure: the underlying inhibitory ability for each participant. When no correlations are found between behavioral measures, the typical inference is that measurement noise and unique factors precluded finding correlations, or there are no detectable common mechanisms across tasks. This is plausible if, for example, different control mechanisms mediate perceptual filtering, rule selection, and response selection, and these are used differentially in various interference tasks. However, there are two basic ways in which common mechanisms may exist but be obscured in behavioral analyses. First, additional task-specific mechanisms may be recruited in addition to common mechanisms. Second, individual differences in the automaticity of task-unique relevant and irrelevant processes may cause differential recruitment of common mechanisms in different tasks. We provide examples of each below.

First, consider the flanker and GNG tasks. Imagine a participant with poor response selection efficiency compared with the rest of the group, but a subject who has a strong focus of spatial attention. In the flanker task, interference can be resolved during stimulus encoding or during response selection. In the GNG task, conflict can only be resolved during response selection. Our example subject may show a reduced flanker interference effect (compared to the group) due to his ability to selectively attend to the target stimulus. The subject experiences less conflict than the rest of the group because she has recruited an additional task-specific mechanism. However, spatial attention will not help this subject during the GNG task, which relies on resolution during response processing. The subject may therefore show a large degree of interference in the GNG task (compared to the group). When

behavioral scores for the two tasks are correlated across participants, the correlation will be low. Although both tasks require conflict-detection and response selection (common mechanisms), they are uncorrelated because different subjects may engage the common response-selection mechanism to different degrees across the two tasks. Thus, though there may be common mechanisms, the existence of additional task-specific processes makes it difficult to uncover them.

Now we turn to the second reason common mechanisms may be obscured in behavioral correlations: individual differences in automaticity of relevant and irrelevant tasks. Suppose that another individual participant has particularly strong automatic response to arrows in the SRC task—that is, the left-arrow/left-button response is particularly automatic. In contrast, the strength of the opposite rule, left-arrow/right button, is relatively weak. That participant will experience more interference and engage interference-resolution mechanisms more than other participants—but only on the SRC task. Those associations between arrows and buttons do not apply to the other tasks. Thus, as in the previous example, the individual's behavioral score will not reflect a constant inhibitory "ability" across all tasks but will instead vary across tasks, and correlations across participants will be low.

The key to both accounts is that different participants experience different amounts of conflict on different tasks (task × participant interactions), independent of their inhibitory ability. Experimental evidence suggests that individual differences in experienced conflict are real, and that they are independent of inhibitory ability. Behavioral evidence from similar tasks suggests that the degree of interference depends on the relative degree of expertise (Ehri and Wilce, 1979), speed (Dyer, 1973), or automaticity (MacLeod and Dunbar, 1988) of each component task. Interference in task shifting may be determined by task automaticity (Allport et al., 1994) and relative speed (Yeung and Monsell, 2003) as well. As various interference tasks involve different relevant and irrelevant processing pathways, differences across individuals in relative speed or automaticity will cause task × participant interactions in experienced conflict. Indeed, individual differences in basic processing speed on particular component tasks do predict behavioral interference (Dyer, 1973) and switch costs (Wager, 2003). In addition, the uniquely activated regions in each task studied here, and those in previous papers (Fan et al., 2003; Rubia et al., 2001), provide some evidence for task-specific mechanisms that may obscure behavioral correlations.

Interpreting unique regions

Paralleling the low correlations in performance among the tasks, we found brain areas uniquely involved in performance of each task we studied. However, it must be stressed that the findings of uniquely activated and correlated regions are preliminary, as this study lacks the experimental power necessary to ensure that false negatives do not occur. Therefore, we interpret these findings cautiously and await further evidence to clarify their roles in cognitive control.

One important role of the meta-analysis is to provide more precise a priori expectations for regions that may be uniquely activated in one task. In the meta-analysis, previous studies showed more consistent activation of the right posterior parietal cortex (angular gyrus) in the GNG task than other tasks. The consistently activated region across previous studies provides a well-defined region in which to test for unique effects. Our analyses of the current study confirm that GNG activity in this region meets our qualifications for a GNG-specific region, in that (a) activations for GNG are more reliable than for other tasks, (b) we found no evidence for activation in other tasks, and (c) activation is correlated with lower performance in the GNG task, but not other tasks.

Thalamic and right parietal activations unique to the GNG task may reflect motor inhibition, through thalamocortical (Guillery and Sherman, 2002) and thalamostriatal loops (Alexander et al., 1986), and control of response execution timing and readiness, though these speculations await support from converging evidence. The GNG task involves only one stimulus and a single, simple response, and so places high demand on control at the response execution stage.

Though we also found limited evidence for flanker-task specificity in left inferior frontal gyrus and underlying insula and striatum, and for SRC-task specificity in the posterior IPS, these results were much weaker. For instance, greater peak density in the meta-analysis in these regions was not paralleled by greater activation in our study. In addition, these regions adjoined commonly activated regions. Thus, at the level of spatial resolution afforded by the fMRI techniques we employed, the commonalities in activation among the tasks seem to outweigh the differences.

Conclusions

We found evidence for common activation of a number of brain structures in three interference tasks that share a requirement for response selection. The regions include bilateral insula/frontal operculum, caudate, lateral prefrontal cortices, anterior cingulate, and right premotor and parietal cortices. A subset of these (most consistently the insula) were positively correlated with poorer behavioral performance in each task. The common locations of performance-correlated activations suggests common mechanisms of interference detection and/or resolution across tasks. However, neither activation nor behavioral scores were correlated across tasks. Together, these findings suggest that these commonly activated brain structures are markers of conflict experienced on a task, and that low behavioral correlations among performance scores reflect systematic effects of task-specific mechanisms and biases rather than a lack of common mechanisms. That is, the brain mechanisms for interference resolution may be common, but a participant may recruit them to different degrees in different tasks, depending on individual differences in ability on different tasks.

References

Alexander, G.E., DeLong, M.R., Strick, P.L., 1986. Parallel organization of functionally segregated circuits linking basal ganglia and cortex. Annu. Rev. Neurosci. 9, 357–381.

Allport, A., Styles, E.A., Hsieh, S.L., 1994. Shifting intentional set-exploring the dynamic control of tasks. Atten. Perform. Xv 15, 421–452.

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. Arch. Clin. Neuropsychol. 13 (7), 585–595.

Braver, T.S., Reynolds, J.R., Donaldson, D.I., 2003. Neural mechanisms of transient and sustained cognitive control during task switching. Neuron 39 (4), 713–726.

Brett, M., Wager, T., Poline, J.-B., Nichols, T., 2004. When is a Conjunction not a Conjunction. Paper presented at the annual meeting of the organization for Hum. Brain Mapp.

Broadbent, D.E., 1977. The hidden preattentive processes. Am. Psychol. 32 (2), 109–118.

Bunge, S.A., Dudukovic, N.M., Thomason, M.E., Vaidya, C.J., Gabrieli, J.D., 2002a. Immature frontal lobe contributions to cognitive control in children: evidence from fMRI. Neuron 33 (2), 301–311.

Bunge, S.A., Hazeltine, E., Scanlon, M.D., Rosen, A.C., Gabrieli, J.D.E., 2002b. 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. J. Int. Neuropsychol. Soc. 4 (6), 547–558.

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. J. Cogn. Neurosci. 9, 835–847.

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. Proc. Natl. Acad. Sci. 97, 8728–8733.

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.

Corbetta, M., Akbudak, E., Conturo, T.E., Snyder, A.Z., Ollinger, J.M., Drury, H.A., et al., 1998. A common network of functional areas for attention and eye movements. Neuron 21 (4), 761–773.

Dagenbach, D., Carr, T.H., 1994. Inhibitory processes in attention, memory, and language. Academic Press, Inc., San Diego, CA.

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.

DeJong, R., Coles, M.G.H., Logan, G.D., 1995. Startegies and mechanisms in nonselective and selective inhibitory motor control. J. Exp. Psychol.: Human Percept. Perform. 21, 498–511.

Duncan, J., Johnson, R., Swales, M., Freer, C., 1997. Frontal lobe deficits after head injury: unity and diversity of function. Cogn. Neuropsychol. 14 (5), 713–741.

Dyer, F., 1973. The Stroop phenomenon and its use in the study of perceptual, cognitive, and response. Mem. Cogn. 1 (2), 106–120.

Ehri, L., Wilce, L., 1979. Does word training increase or decrease interference in a Stroop task? J. Exp. Child Psychol. 27 (2), 352–364.

Eriksen, B.A., Eriksen, C.W., 1974. Effects of noise letters upon the identification of a target letter in a nonsearch task. Percept. Psychophys. 16 (1), 143–149.

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.

Guillery, R.W., Sherman, S.M., 2002. Thalamic relay functions and their role in corticocortical communication: generalizations from the visual system. Neuron 33 (2), 163–175.

Hazeltine, E., Poldrack, R., Gabrieli, J.D.E., 2000. Neural activation during response competition. J. Cogn. Neurosci. 12, 118–129.

Hubert, M., 2001. Multivariate outlier detection and robust covariance matrix estimation—Discussion. Technometrics 43 (3), 303–306.

Iacoboni, M., Woods, R.P., Mazziotta, J.C., 1996. Brain–behavior relationships: evidence from practice effects in spatial stimulus–response compatibility. J. Neurophysiol. 76 (1), 321–331.

Jones, A.D., Cho, R.Y., Nystrom, L.E., Cohen, J.D., Braver, T.S., 2002. A computational model of anterior cingulate function in speeded response tasks: effects of frequency, sequence, and conflict. Cogn. Affect. Behav. Neurosci. 2 (4), 300–317.

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., 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.

Kornblum, S., Hasbroucq, T., Osman, A., 1990. Dimensional overlap: cognitive basis for stimulus–response compatibility: a model and taxonomy. Psychol. Rev. 97 (2), 253–270.

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. Hum. Brain Mapp. 12 (2), 100–109.

Lin, F.H., McIntosh, A.R., Agnew, J.A., Eden, G.F., Zeffiro, T.A., Belliveau, J.W., 2003. Multivariate analysis of neuronal interactions in the generalized partial least squares framework: simulations and empirical studies. NeuroImage 20 (2), 625–642.

Liu, T.T., 2004. Efficiency, power, and entropy in event-related fMRI with multiple trial types: Part II. Design of experiments. NeuroImage 21 (1), 401–413.

Liu, T.T., Frank, L.R., Wong, E.C., Buxton, R.B., 2001. Detection power, estimation efficiency, and predictability in event-related fMRI. NeuroImage 13 (4), 759–773.

Logan, G.D., 1985. Executive control of thought and action. Acta Psychol. 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. Mem. Cogn. 7, 382–389.

MacDonald, A.W., Cohen, J.D., Stenger, V.A., Carter, C.S., 2000. Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. Science 288, 1835–1838.

MacLeod, C.M., Dunbar, K., 1988. Training and Stroop-like interference: evidence for a continuum of automaticity. J. Exper. Psychol., Learn., Mem., Cogn. 14, 126–135.

Mangun, G.R., 1995. Neural mechanisms of visual selective attention. Psychophysiology 32 (1), 4–18.

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. Hum. Brain Mapp. 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.

Miller, E.K., Cohen, J.D., 2001. An integrative theory of prefrontal cortex function. Annu. Rev. Neurosci. 24, 167–202.

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. Cogn. Psychol. 41, 49–100.

Monsell, S., Yeung, N., Azuma, R., 2000. Reconfiguration of task-set: is it easier to switch to the weaker task? Psychol. Res./Psychol. Forsch.: Spec. Issue: Executive Control 63 (3–4), 250–264.

Nee, D., Jonides, J., Wager, T.D., 2004. A meta-analysis of inhibitory tasks in neuroimaging. Manuscript in preparation (unpublished manuscript).

Nichols, T.E., Brett, M., Anderson, J., Wager, T.D., Poline, J.B., in press. Valid conjunction inference with the minimum statistic. Neuroimage.

Oppenheim, A.V., Schafer, R.W., Buck, J.R., 1999. Discrete-time signal processing, 2nd ed. Prentice Hall, Upper Saddle River, NJ.

Peterson, B.S., Kane, M.J., Alexander, G.M., Lacadie, C., Skudlarski, P., Leung, H.C., 2002. An event-related functional MRI study comparing

interference effects in the Simon and Stroop tasks. Cogn. Brain Res. 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. J. Exp. Psychol. Hum. Percept. Perform. 27, 763–797.
Rubia, K., Russell, T., Overmeyer, S., Brammer, M.J., Bullmore, E.T., Sharma, T., 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. Hum. Brain Mapp. 17 (3), 193–201.
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.
Talairach, J., Tournoux, P., 1988. A Coplanar Stereotactic Atlas of the Human Brain. Theime, Stuttgart.
van Veen, V., Cohen, J.D., Botnich, M.M., Stenger, V.A., Carter, C.C., 2001. Anterior cingulate cortex conflict monitoring and levels of processing. NeuroImage 14, 1302–1308.
Wager, T.D., 2003. Brain and Behavioral Mechanisms of Switching Attention. Unpublished Dissertation, University of Michigan, Ann Arbor.
Wager, T.D., Nichols, T.E., 2003. Optimization of experimental design in fMRI: a general framework using a genetic algorithm. NeuroImage 18 (2), 293–309.
Wager, T.D., Phan, K.L., Liberzon, I., Taylor, S.F., 2003. Valence, gender, and lateralization of functional brain anatomy in emotion: a meta-analysis of findings from neuroimaging. NeuroImage 19 (3), 513–531.
Wager, T.D., Reading, S., Jonides, J., 2004. Neuroimaging studies of shifting attention: a meta-analysis. NeuroImage 22 (4), 1679–1693.
Wager, T.D., Vasquez, A., Hernandez, L., Noll, D.C., 2005. Accounting for nonlinear BOLD effects in fMRI: parameter estimates and a model for prediction in rapid event-related studies. NeuroImage 25 (1), 206–218.
Woods, R.P., Grafton, S.T., Holmes, C.J., Cherry, S.R., Mazziotta, J.C., 1998. Automated image registration: I. General methods and intra-subject, intramodality validation. J. Comput. Assist. Tomogr. 22, 141–154.
Yeung, N., Monsell, S., 2003. Switching between tasks of unequal familiarity: the role of stimulus-attribute and response-set selection. J. Exp. Psychol. Hum. Percept. Perform. 29 (2), 455–469.
Zhang, H.H., Zhang, J., Kornblum, S., 1999. A parallel distributed processing model of stimulus–stimulus and stimulus–response compatibility. Cogn. Psychol. 38 (3), 386–432.