Published in final edited form as:

Psychol Sci. 2008 May ; 19(5): 490–500. doi:10.1111/j.1467-9280.2008.02114.x.

Dissociable Interference-Control Processes in Perception and Memory

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Abstract

Control over interference is a pervasive feature of cognitive life. Central to research on interference control has been the identification of its underlying mechanisms. Investigations have focused on processes that filter out distracting perceptual information, leading to negative priming, and processes that discard intruding memories that cause proactive interference. Theories differ regarding whether or not a single process during episodic retrieval underlies both negative priming and the resolution of proactive interference. Using functional magnetic resonance imaging, we combined both phenomena into a single paradigm and found that occipital cortex shows activation uniquely related to negative priming, whereas activation increases in left lateral prefrontal cortex are uniquely associated with proactive interference. This pattern of results contradicts theories that rely on a single process to account for both phenomena. However, results also showed common recruitment of right dorsolateral prefrontal cortex and parietal regions and therefore suggest that some control processes are shared.

Successful cognition depends on performing goal-directed actions in the face of interference. Most tasks require selective attention to some inputs and filtering out of others. In addition, most tasks require holding certain relevant thoughts in mind while shielding these from potential intrusion by irrelevant thoughts. Goal-directed actions therefore require the selection of relevant information, the deselection of irrelevant information, or both. Understanding how people are able to perform such selection and deselection is central to understanding cognition.

For more than a century, inhibition has been a popular account of how people are able to filter out intrusive information (for reviews, see R. Smith, 1992, and MacLeod, Dodd, Sheard, Wilson, & Bibi, 2003). The idea is that inhibition can attenuate the representation of distracting information so that it poses a reduced threat to ongoing cognition. Failures of inhibition have been associated with various disorders, including schizophrenia (MacQueen, Galway, Goldberg, & Tipper, 2003), attention-deficit/hyperactivity disorder (Nigg, 2001), depression (Joormann, 2005), and obsessive-compulsive disorder (Enright & Beech, 1993). Moreover, improved inhibition has been used to explain cognitive advances during development (Diamond & Gilbert, 1989), and declining inhibition has been linked to cognitive deficits associated with aging (Hasher & Zacks, 1988). These examples demonstrate the central importance of the concept of inhibition in accounts of cognitive functioning.

Although central to many cognitive models, inhibition remains poorly understood. There is contention regarding whether a single process (Hasher & Zacks, 1988; Kane, Bleckley,

Conway, & Engle, 2001) or a family of processes (Harnishfeger, 1995) downregulates distracting information, and theories that posit multiple inhibition-related functions differ regarding the appropriate taxonomy of these functions (Dempster, 1995; Friedman & Miyake, 2004; Harnishfeger, 1995; Kornblum, Hasbroucq, & Osman, 1990). Moreover, some theorists doubt whether inhibition exists at all and instead posit that performance costs thought to be related to inhibition are actually products of conflict resolution resulting from memory retrieval (MacLeod et al., 2003).

Negative priming (NP) has long been taken as a hallmark of inhibitory function (for reviews, see Fox, 1995; May, Kane, & Hasher, 1995; Tipper, 2001). In a typical NP task, subjects are required to attend to a target while ignoring an irrelevant distractor. Subjects generally demonstrate slowed and less accurate responding when the target of the current trial was a distractor on a previous trial, relative to when the target was not previously a distractor. Initial accounts posited that distracting items are inhibited in order to shield processing from interference (Tipper, 2001); then, when an inhibited item later becomes a target, additional processes have to be recruited to overcome the inhibition, and this leads to slowed, more inaccurate performance.

A contrasting position is that inhibition need not be invoked to explain these findings (MacLeod et al., 2003). The claim is that, instead, the costs associated with NP are a result of episodic retrieval processes. According to this position, presentation of an item automatically retrieves prior episodes associated with that item. These episodes include various pieces of contextual information, including identity information, location, and the status of the item (e.g., relevant or irrelevant), as well as responses that have been associated to the item (e.g., “respond” or “do not respond”). Hence, when a previous distractor becomes a target, current goals will clash with some retrieved details (e.g., information about relevance and responses). It is the resolution of this episodic-retrieval-related conflict that causes the observed reduction in performance, according to this account.

MacLeod et al. (2003) supposed that conflict in episodic retrieval underlies performance costs not only in NP, but also in Stroop, task-switching, and directed-forgetting situations. This model is parsimonious in explaining a variety of data, providing a single account for many interference effects previously associated with inhibition. The potential impact of this model on theories of cognitive control, development, aging, and various disorders has generated a great deal of debate regarding whether or not inhibition exists at the psychological level (see Gorfein & MacLeod, 2007, for a summary of conference proceedings on this matter). However, there is now accruing evidence that response-related processes do, in fact, enlist inhibitory control (see Aron, 2007, for a review). Moreover, there is some evidence that functions that inhibit responses are dissociable from functions that resolve interference in memory (Bissett, Nee, & Jonides, 2008; Friedman & Miyake, 2004). Therefore, although some processes of interference control may act upon conflicting episodic details, those that act upon responses may be inhibitory. Is it possible that other effects of interference control are also due to inhibitory mechanisms that are distinguishable from episodic retrieval?

The present study examined the control of interference to see whether control involves a single process or multiple processes. In a single experimental paradigm, we combined NP and a directed-forgetting procedure that induces proactive interference (PI). It has been claimed that interference in both cases may require control processes that resolve conflict during episodic retrieval (Jonides & Nee, 2006; MacLeod et al., 2003; Nee, Jonides, & Berman, 2007). Using event-related functional magnetic resonance imaging (fMRI), we looked for common and dissociable neural patterns underlying NP and the resolution of PI.¹ This method allowed us to determine (a) whether interference-control processes related to

distracting information and intruding memories are common or dissociable and (b) whether NP is related to inhibition, episodic retrieval, or both.

METHOD

Participants

Sixteen right-handed adults (12 females, 4 males; ages 19–26) participated in this study. One subject was removed from analyses of the imaging data because of motion artifacts, leaving 16 subjects for behavioral analyses and 15 subjects for imaging analyses.

Materials and Procedure

Subjects performed two tasks, illustrated in Figure 1. Trials of the two tasks were randomly intermixed.

In the ignore task, each trial began with a red fixation cue, presented for 1 s. This cue alerted the subject that the trial was about to begin. The following 1-s cue instructed subjects to ignore words presented in a particular color (either “ignore teal” or “ignore blue”). After 1.5 s of fixation, six words, three in blue and three in teal, were presented for 3 s. Displays were arranged such that words of the same color formed either a “V” or an upside-down “V” shape. Subjects were required to commit to memory the three words they were *not* told to ignore, and to ignore the other three words. After a 3-s retention interval, a probe was presented for 1.5 s. Fifty percent of the probes were members of the target set (positive-ignore probes), 25% were words subjects had been told to ignore (interference-ignore probes), and 25% were words that had not appeared in the previous two trials (control-ignore probes). All responses were recorded on an MR-compatible 10-button response unit included with the IFIS 9.0 system (MRI Devices Corp., Latham, NY). Subjects were told to respond affirmatively to positive-ignore probes by pressing the button corresponding to their left index finger, and to respond negatively to all other probes by pressing the button corresponding to their right index finger. Trials were separated by a 4-s interval. NP was measured by contrasting responses to interference-ignore probes with responses to control-ignore probes.

In the forget task, each trial began with a red fixation cue, presented for 1 s. Again, this cue alerted the subject that the trial was about to begin. After an additional 2.5 s of fixation, six words, three in blue and three in teal, were presented for 3 s. Subjects were required to commit all six words to memory. Displays were arranged such that words of the same color formed a “V” or upside-down “V” shape. After a 1-s retention interval, a 1-s cue instructed subjects to remove words of a particular color from memory (e.g., “forget teal”). After another 1-s retention interval, a probe was presented for 1.5 s. Fifty percent of the probes were members of the target set (positive-forget probes), 25% were words subjects had been told to forget (interference-forget probes), and 25% were words that had not appeared in the previous two trials (control-forget probes). Subjects were told to respond affirmatively to positive-forget probes by pressing the button corresponding to their left index finger, and to respond negatively to all other probes by pressing the button corresponding to their right index finger. Trials were separated by a 4-s interval. PI was measured by contrasting responses to interference-forget probes with responses to control-forget probes.

¹We use the term “resolution of PI,” rather than “PI,” to distinguish the interference (PI) and the interference-control processes acting upon that interference (resolution). Of interest are these resolution processes, which are hypothesized to involve left lateral prefrontal cortex (see Jonides & Nee, 2006, for a recent review). By contrast, we use the term “NP,” rather than “resolution of NP,” because the interpretation of the behavioral phenomenon depends on the particular theoretical stance one takes, which is part of what is explored here. Hence, we use the term “NP” to describe the phenomenon, which can be either a product of prior inhibition or interference that necessitates episodic-retrieval control processes.

Subjects performed eight runs of 24 trials, for a total of 96 trials of each task. On each trial, all words were drawn randomly from a list of 80 four-letter nouns, with the restriction that no word had appeared in the previous 2 trials.

Image Acquisition and Preprocessing

Images were acquired on a GE Signa 3-T scanner equipped with a standard quadrature head coil. Head movement was minimized using foam padding and a cloth restraint strapped across participants' foreheads. Experimental tasks were presented using E-Prime software (Psychology Software Tools, Inc., Pittsburgh, PA) and the IFIS 9.0 system, with its 10-button response unit (MRI Devices Corp., Latham, NY).

Functional T2*-weighted images were acquired using a spiral sequence with 40 contiguous slices with voxels of size $3.44 \times 3.44 \times 3$ mm (repetition time, or TR = 2,000 ms; echo time, or TE = 30 ms; flip angle = 90° ; field of view, or FOV = 22 mm^2). A T1-weighted gradient-echo anatomical overlay was acquired using the same FOV and slices (TR = 250 ms, TE = 5.7 ms, flip angle = 90°). Additionally, a 106-slice high-resolution T1-weighted anatomical image was collected using spoiled-gradient-recalled acquisition (SPGR) in steady-state imaging (TR = 10.5 ms, TE = 3.4 ms, flip angle = 25° , FOV = 24 mm^2 , slice thickness = 1.5 mm).

Each SPGR anatomical image was corrected for signal inhomogeneity and skull-stripped using FSL's Brain Extraction Tool (S.M. Smith et al., 2004). These images were then normalized to the Montreal Neurological Institute (MNI) template using SPM2 (Wellcome Department of Cognitive Neurology, London). Functional images were corrected for differences in slice timing using 4-point sinc interpolation (Oppenheim, Schafer, & Buck, 1999) and were corrected for head movement using MCFLIRT (Jenkinson, Bannister, Brady, & Smith, 2002). To reduce the impact of spike artifacts, we winsorized functional images on a voxel-by-voxel basis so that no voxel had a signal greater than 3.5 standard deviations from the mean of the run (Lazar, Eddy, Genovese, & Welling, 2001). Spatial normalization transformations and 8-mm full-width/half-maximum isotropic Gaussian smoothing were applied to all functional images prior to analysis using SPM2. All analyses included a temporal high-pass filter (128 s), and each image was scaled to have a global mean intensity of 100.

Image Analysis

Whole-brain analyses were conducted using the General Linear Model implemented in SPM2. Probe-locked predictors were convolved with a canonical hemodynamic response function, as well as time and dispersion derivatives. To account for artifacts produced by head motion, we calculated linear, quadratic, differential, and quadratic differential motion regressors from the realignment parameters and included these regressors in the model (Lund, Norgaard, Rostrup, Rowe, & Paulson, 2005). Contrast images for each participant were subjected to a random-effects group analysis. Trials with incorrect responses were excluded from analysis.

To examine neural correlates of NP, we contrasted responses to interference-ignore probes with responses to control-ignore probes. To examine neural correlates of PI resolution, we contrasted responses to interference-forget probes with responses to control-forget probes. The threshold for both of these contrasts was $p < .001$, uncorrected, and the contrasts were restricted to regions demonstrating at least five contiguous suprathreshold voxels (Forman et al., 1995; Poline, Worsley, Evans, & Friston, 1997).

To assess regions showing dissociable responses to NP versus PI, we examined regions showing Task (ignore vs. forget) $\times$ Probe (interference vs. control) interactions. Interactions were assessed using a separate whole-brain random-effects analysis. Interaction regions were defined as regions showing both significant ($p < .001$) interference-related activation increases in one task and significantly greater ($p < .01$) interference-related activation increases for one task than the other; for both criteria, regions were required to include at least five contiguous voxels.

To assess regions showing common responses to NP and PI, we performed a conjunction analysis on the contrasts of both tasks. For each task, the threshold used was $p < .01$; thus, the conjoint threshold was $p < .0001$. Again, regions were required to include at least five contiguous voxels.

RESULTS

Behavioral Results

Reaction times (RTs) were calculated for trials with correct responses. Separate 2 (task) $\times$ 3 (probe) repeated measures analyses of variance were performed on error rates and RTs. We found significant NP and PI in the RTs. Table 1 presents the means and standard deviations for the behavioral dependent variables.

The main effect of task was significant for error rates, $F(1, 15) = 28.442$, $p < .001$, and marginally significant for RTs, $F(1, 15) = 4.19$, $p < .06$. Inspection of the data revealed that the effect of task was due to worse performance on the forget task than on the ignore task. The main effect of probe was significant for both error rates, $F(2, 14) = 5.651$, $p < .05$, and RTs, $F(2, 14) = 15.467$, $p < .001$. The Task $\times$ Probe interaction was also significant for both error rates, $F(2, 14) = 7.823$, $p < .01$, and RTs, $F(2, 14) = 5.48$, $p < .05$.

Planned contrasts revealed a significant NP effect in the RTs, with responses to interference-ignore probes being slower than responses to control-ignore probes (difference = 23.1 ms), $t(15) = 2.392$, $p < .05$. Error rates did not show a comparable effect, $t(15) = -1.112$, $p > .25$. There was also a significant PI effect in the RTs, with responses to interference-forget probes being slower than responses to control-forget probes (difference = 79.5 ms), $t(15) = 4.545$, $p < .001$. The error rates showed an effect in the same direction, but it did not reach significance (difference = 4.6%), $t(15) = 1.808$, $p < .1$.

Imaging Results

Activation increases associated with NP were most notable in occipital cortex, in the left calcarine sulcus and right lingual gyrus. There were also significant activation increases in right inferior temporal gyrus, right premotor cortex, left paracentral gyrus, and right intraparietal sulcus (see Table 2 and Fig. 2).

PI-related activation was most prominent in left lateral prefrontal cortex, largely in dorsolateral prefrontal cortex, but also reaching ventrolateral prefrontal cortex. Additionally, activation increases were found in bilateral precuneus, right intraparietal sulcus, right premotor cortex, right dorsolateral prefrontal cortex, and right cerebellum (see Table 2 and Fig. 2).

To examine whether any regions demonstrated unique interference-specific activation, we looked for regions demonstrating a Task $\times$ Probe interaction. Complete results are listed in Table 3, but we focus on the most critical regions of interest here. Whereas left occipital cortex in the calcarine sulcus demonstrated unique NP-related activation, left lateral prefrontal cortex demonstrated unique PI-related activation (see Table 3 and Fig. 3). To

assess whether these dissociable regions were related to performance, we looked for activation-behavior correlations in left occipital and left lateral prefrontal cortex (see Fig. 4). A region in left occipital cortex (MNI center: $x = -14$, $y = -88$, $z = 10$; Brodmann's area 17; 17 voxels) correlated with NP ($r = .6031$, $p < .05$), but not PI ($r = -.0657$, $p > .8$). By contrast, a region in left lateral prefrontal cortex (MNI center: $x = -42$, $y = 20$, $z = 34$; Brodmann's area 9/44; 33 voxels) correlated with PI ($r = -.6439$, $p < .01$), but not NP ($r = -.1925$, $p > .4$).

Finally, we used a conjunction analysis to assess whether NP and PI produced any common neural correlates. Three regions emerged from this analysis: right dorsolateral prefrontal cortex, right intraparietal sulcus, and left precuneus (see Table 3 and Fig. 5).

DISCUSSION

Theories of interference control disagree about whether NP and PI involve a single process acting during episodic retrieval (MacLeod et al., 2003) or distinct forms of control (e.g., Friedman & Miyake, 2004). We found dissociable neural recruitment for NP and PI, with occipital cortex demonstrating unique involvement in NP and left lateral prefrontal cortex demonstrating unique activation related to PI. These results support the notion that NP and PI involve at least partially distinct control mechanisms. Using confirmatory factor analysis in a correlational study, Friedman and Miyake (2004) proposed that resistance to interference from distractors and resistance to PI were distinguishable factors. However, ours is the first study to combine both forms of interference control in a single experimental paradigm. Our results, together with those of Friedman and Miyake, provide strong support for the position that interference-control processes for filtering perceptual material are distinct from interference-control processes for filtering intrusive memories.

Accounting for Negative Priming

Our results showed activation increases in primary visual cortex that were unique to NP. Moreover, this occipital region demonstrated a strong correlation with behavioral indices of NP. Why was primary visual cortex associated with NP? One possible explanation is that the NP trials (interference-ignore probes) yielded longer RTs than the NP control trials (control-ignore probes), and hence more time on task. However, if this portion of cortex were simply responding to time on task, it also should have yielded greater activation on the PI trials (interference-forget probes) than on the PI control trials (control-forget probes). In fact, this region demonstrated decreased activation in the face of PI. Therefore, time on task was not the mediating factor.

A second possibility is that the increases in occipital activation somehow represent difficulty in episodic retrieval. This seems implausible in that activation of primary visual cortex should precede any memory-related processes. Furthermore, any episodic conflict should have been present for both NP conflict and PI conflict, yet the occipital activation was present only for NP. It is possible that NP and PI elicited the retrieval of different episodic details. For example, subjects may have retrieved visual details in responding to interference-ignore probes and phonological details (e.g., placement in the rehearsal loop) in responding to interference-forget probes, and this could have caused the observed dissociation between occipital and left frontal activation. However, both color and phonology distinguished both ignore and forget items from relevant material. Hence, this account would likely predict a quantitative distinction between interference-ignore and interference-forget probes, whereas the observed data indicate a qualitative distinction.

We believe that the observed pattern of activations for NP is best accounted for by an inhibitory mechanism. Some models of NP have lodged the effect of inhibition at the level

of semantic representations (e.g., Tipper, 2001). Better suited to our task, however, is an account in which the inhibitory processes occur earlier in the processing stream, with the visual representations themselves. The task we used requires subjects to filter out three distractors, while making saccades to encode three relevant items. The likelihood of encoding a wrong item in this situation may therefore call for recruitment of early selection processes. Consequently, the *perceptual* representations of the ignored items may be inhibited. If so, when an ignored item is subsequently presented as a probe, visual processes must overcome this inhibition in order to encode the item. Hence, the increases in primary visual activation associated with NP may be related to overcoming perceptual inhibition.

Accounting for Proactive Interference

A growing body of literature has implicated left lateral prefrontal cortex in the resolution of PI (see Jonides & Nee, 2006, for a review). Our results are consistent with this literature, in that we found unique PI-related activation in lateral prefrontal cortex, and this activation correlated with performance. These activation increases were somewhat more dorsal than in previous reports (e.g., Nee et al., 2007), perhaps because of increased selection difficulty in the task we studied. There is evidence that more dorsal regions of prefrontal cortex are recruited as processing demands increase (e.g., Postle, Berger, & D'Esposito, 1999). Previous studies of directed forgetting in short-term memory used item- or location-based forget cues (Nee et al., 2007; Zhang, Feng, Fox, Gao, & Tan, 2004; Zhang, Leung, & Johnson, 2003). These cues had obvious mappings to the items to be maintained and discarded, and made it relatively easy to distinguish relevant and irrelevant items in short-term memory. However, the color cue we used did not have an obvious mapping to the items to be maintained and forgotten, so selection was potentially more difficult.

Is the left lateral prefrontal area somehow involved in inhibitory processing? Although early accounts claimed it is (Jonides, Smith, Marshuetz, & Koeppe, 1998), more recent models have gravitated toward the notion that this region is involved in selection of contextual details during episodic retrieval (Badre & Wagner, 2005; Jonides & Nee, 2006; Nee et al., 2007). In the face of PI, this region shows increased functional connectivity with the medial temporal lobe and premotor cortex, which suggests that left lateral prefrontal cortex selects episodic details in order to bias decision processes (Nee et al., 2007). Our data suggest that these control processes are distinct from those related to NP.

Common Activations

We also found common recruitment of right dorsolateral prefrontal cortex, right intraparietal sulcus, and left precuneus for NP and PI. In a previous study examining NP in a Stroop task, Egner and Hirsch (2005) also found activation increases in right dorsolateral prefrontal cortex (Egner & Hirsch, 2005). Because this region has been associated with episodic retrieval, these authors took this as evidence supporting the episodic-retrieval account of NP. Moreover, the parietal regions that we found to be related to both NP and PI have also been implicated in the retrieval of specific episodic details (see Wagner, Shannon, Kahn, & Buckner, 2005, for a review). These results suggest that there are episodic components common to NP and PI. These components may reflect contrasting episodic details that are retrieved when an item is a probe ("respond to me") versus when it is ignored or removed from memory ("do not respond to me"). However, none of these regions demonstrated a significant correlation with behavior ($p > .05$), so it is premature to conclude that they reflect episodic components common to NP and PI.

Relation to Other Work

Egner and Hirsch (2005) also reported NP-related activation in the medial dorsal thalamus (MNI peak: $x = 10$, $y = -20$, $z = 14$; 30 voxels). Moreover, they reported that activation in

this region correlated negatively with behavioral indices of NP. In a post hoc analysis, we looked for comparable activity in the medial dorsal thalamus. At a more liberal threshold ($p < .01$), our NP contrast identified a similar region (MNI peak: $x = -10$, $y = -18$, $z = 16$; 10 voxels) that demonstrated a marginally significant correlation with behavioral indices of NP ($r = -.4831$, $p = .07$). This region did not show comparable activation related to PI ($p > .9$). Egner and Hirsch noted that this region is altered in schizophrenia. Schizophrenics demonstrate reduced NP (MacQueen et al., 2003) and a concomitant decrease in medial dorsal thalamus volumes (e.g., Kemether et al., 2003). Schizophrenics also show decreased metabolic activity in the medial dorsal thalamus (Buchsbaum et al., 1996). Egner and Hirsch interpreted NP-related activity in the medial dorsal thalamus to indicate that episodic retrieval, and not inhibition, is deficient in schizophrenia. However, our results suggest the contrary, in that activation increases in this region were associated only with NP, not with PI. This evidence is more consistent with models that associate schizophrenia with deficient inhibition (MacQueen et al., 2003).

CONCLUSIONS

In summary, our data suggest some dissociable interference-control processes related to NP and PI. They indicate that in addition to interference costs in responding, other interference costs cannot be cast as problems in episodic retrieval. Resisting perceptual interference and resolving PI appear to be dissociable functions. Moreover, our data suggest that there are inhibitory components to NP, acting as early as primary visual cortex, and perhaps involving the medial dorsal thalamus as well. However, NP may have some components related to conflict during episodic retrieval, as retrieval-related regions are recruited in both NP and PI.

Understanding how control over perceptual and memorial representations is achieved is central to understanding cognition. Our data highlight the importance of distinguishing different forms of interference control that are overcome by different mechanisms. Our data also highlight the value of neuroimaging as a way to parse different psychological mechanisms that are critical to cognitive processing. Such parsing of psychological mechanisms can be applied to studying deficits in cognitive processing as well (e.g., Jonides & Nee, 2005).

Acknowledgments

This research was supported in part by the National Science Foundation (Grant 0520992), by the National Institute of Mental Health (Grant MH60655), and by a National Science Foundation Graduate Research Fellowship awarded to the first author.

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Fig. 1.

A schematic of the tasks. On each trial, half of the words were presented in teal (shown here in gray) and half in blue (shown here in black). Trials of the two tasks were randomly intermixed. Hemodynamic effects were assessed when the probes were presented. See the text for details. Fixation crosses depicted here in gray were presented in red; fixation crosses depicted here in black were presented in black. ITI = intertrial interval.

Fig. 2.

Results of whole-brain analyses demonstrating regions responsive to interference. The results are plotted on lateral (first and third illustrations from the left) and medial (second and fourth illustrations from the left) surfaces of a canonical SPM2 brain. The upper panel depicts the contrast of interference-ignore probes and control-ignore probes (negative priming), and the bottom panel depicts the contrast of interference-forget probes and control-forget probes (proactive interference). Regions showing activation increases for interference relative to control probes are highlighted in color. The color scale represents t values ranging from 2.0 (red) to 5.0 and above (yellow). L = left hemisphere; R = right hemisphere.

Fig. 3.

Activation increases unique to negative priming and proactive interference. Regions demonstrating Task (ignore vs. forget) $\times$ Probe (interference vs. control) interactions are plotted on axial slices of a canonical SPM2 brain; z coordinates are from Montreal Neurological Institute space. The results for these regions, in left occipital cortex (top) and left lateral prefrontal cortex (PFC; bottom), are shown in the bar graphs, which plot contrast estimates (beta values derived from the general linear model) as a function of contrast: interference-ignore – control-ignore for negative priming and interference-forget – control-forget for proactive interference.

Fig. 4.

Correlations between behavioral indices of interference control (negative priming in the graphs on the left and proactive interference in the graphs on the right) and neural activation in left occipital cortex (top graphs) and left lateral prefrontal cortex (bottom graphs). PFC = prefrontal cortex.

Fig. 5.

Regions demonstrating common interference-related activity for negative priming and proactive interference. These regions are plotted on axial slices of a canonical SPM2 brain; z coordinates are from Montreal Neurological Institute space. The results for these regions, in right dorsolateral prefrontal cortex (DLPFC; top), left precuneus (bottom left), and right intraparietal sulcus (IPS; bottom right), are shown in the bar graphs, which plot contrast estimates (beta values derived from the general linear model) as a function of contrast: interference-ignore – control-ignore for negative priming and interference-forget – control-forget for proactive interference.

TABLE 1

Mean Reaction Times and Error Rates in the Two Tasks

Measure	Ignore task			Forget task		
	Interference- ignore probe	Control- ignore probe	Positive- ignore probe	Interference- forget probe	Control- forget probe	Positive- forget probe
Reaction time (ms)	642.89 (31.14)	619.82 (23.65)	619.88 (25.50)	698.89 (32.59)	619.43 (29.87)	614.18 (33.29)
Error rate (%)	1.8 (2.5)	3.3 (5.3)	3.5 (3.4)	9.8 (7.0)	5.2 (6.5)	14.8 (10.9)

Note. Standard deviations are given in parentheses.

TABLE 2

Neural Correlates of Negative Priming and Proactive Interference

Peak coordinates			Number of voxels	<i>t</i>	Brodmann's area	Region
<i>x</i>	<i>y</i>	<i>z</i>				
Greater activation for interference-ignore probes than for control-ignore probes (negative priming)						
38	18	−32	7	4.83	38	Right inferior temporal gyrus
2	−64	0	14	4.79	18	Right lingual gyrus
−10	−84	6	42	4.54	17	Left occipital cortex (calcarine sulcus)
36	−4	60	13	4.38	6	Right premotor cortex
−8	−34	54	15	4.29	5	Left paracentral gyrus
36	−46	56	8	4.2	7/40	Right intraparietal sulcus
Greater activation for interference-forget probes than for control-forget probes (proactive interference)						
−38	22	40	133	6.16	9/8	Left dorsolateral prefrontal cortex
−48	18	30		4.05	44/9/46	Left ventrolateral, dorsolateral prefrontal cortex
0	−66	42	45	5.01	7	Precuneus
14	−54	54	59	4.89	7	Right precuneus
−6	−52	48	21	4.41	7	Left precuneus
42	−68	50	38	4.4	7	Right intraparietal sulcus
44	−74	38		4.33	19/39	Right intraparietal sulcus
48	−68	42		4.31	7	Right intraparietal sulcus
8	−68	−46	6	4.36	—	Right cerebellum
30	12	54	26	4.36	6/8	Right premotor cortex
32	34	46	7	4.16	9/8	Right dorsolateral prefrontal cortex

Note. The table presents results of a whole-brain analysis with thresholds of $p < .001$ and 5 contiguous voxels. Peak coordinates are in Montreal Neurological Institute space.

TABLE 3

Common and Distinct Regions of Interference Control

Coordinates of center voxel			Number of voxels	Brodmann's area	Region
x	y	z			
Unique activation associated with negative priming					
-10	-86	6	16	17	Left calcarine sulcus
Unique activation associated with proactive interference					
-40	26	38	40	9	Left dorsolateral prefrontal cortex
46	-70	40	26	39	Right intraparietal sulcus
2	-64	44	9	7	Right precuneus
14	-54	52	36		
32	36	44	6	9/8	Right dorsolateral prefrontal cortex
30	10	54	17	6/8	Right premotor cortex
4	-66	-48	6	—	Right cerebellum
Common neural correlates					
32	32	42	9	9/8	Right dorsolateral prefrontal cortex
38	-50	50	26	40	Right intraparietal sulcus
-8	-54	50	7	7	Left precuneus

Note. Coordinates are in Montreal Neurological Institute space. The criteria for activation unique to negative priming were that activation differed significantly, $p < .001$, between interference-ignore and control-ignore probes and that this difference was significantly greater, $p < .01$, than the difference between activation for interference-forget and control-forget probes (i.e., interference-ignore – control-ignore > interference-forget – control-forget). The criteria for activation unique to proactive interference were that activation differed significantly, $p < .001$, between interference-forget and control-forget probes and that this difference was significantly greater, $p < .01$, than the difference between interference-ignore and control-ignore probes (i.e., interference-forget – control-forget > interference-ignore – control-ignore). Regions of common activation are those that showed both greater activation for interference-ignore than for control-ignore probes and greater activation for interference-forget than for control-forget probes (interference-ignore > control-ignore and interference-forget > control-forget), $p < .01$.