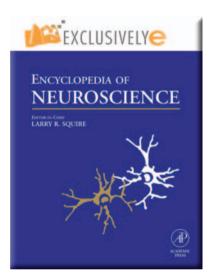
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# **Strategic Control of Memory**

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## Introduction

Cognitive control mechanisms permit memory to be accessed strategically and so aid in bringing knowledge to mind that is relevant to current decisions and actions. A fundamental component of the strategic control of memory is the resolution of interference from competing, irrelevant representations. This article considers how the ventrolateral prefrontal cortex (VLPFC) regulates mnemonic competition in multiple memory systems. We initially discuss how damage to lateral prefrontal cortex impacts mnemonic function and then consider recent neuroimaging and focal lesion findings that highlight the distinct roles that subregions of the VLPFC play in the control of memory.

## **Lateral Prefrontal Cortex**

The lateral prefrontal cortex (PFC) consists of ventral, dorsal, and frontopolar subregions. In this article, we primarily focus on the function of VLPFC. In the human (Figure 1(a)), VLPFC corresponds to the inferior frontal gyrus, which includes (moving caudally to rostrally) the inferior frontal pars opercularis (Brodmann area (BA) 44), inferior frontal pars triangularis (BA 45), and inferior frontal pars orbitalis (an area that Petrides and Pandya term area 47/12, which corresponds to the lateral portion of BA 47). Whereas Petrides and Pandya refer to area 47/12 and BA 45 collectively as the mid-VLPFC, distinguishing these regions from the caudally situated BA 44, in this article we functionally distinguish area 47/12 from BA 45. Thus, we use anterior VLPFC to refer to the inferior frontal pars orbitalis (area 47/12), mid-VLPFC to refer to the pars triangularis (BA 45), and posterior VLPFC to refer to the pars opercularis (BA 44). We note that the caudal portion of area 47/12 actually lies ventral (rather than rostral) to BA 45 (Figure 1(a)). The inferior frontal sulcus in humans (and the principal sulcus in monkeys) marks the approximate boundary between the VLPFC and the dorsolateral PFC (DLPFC). In terms of anatomical connectivity, the VLPFC is strongly connected with cortical areas in the lateral temporal lobe and medial temporal lobe (MTL), including (but not limited to) the inferotemporal cortex, superior temporal cortex, and, more medially, perirhinal and parahippocampal cortices.

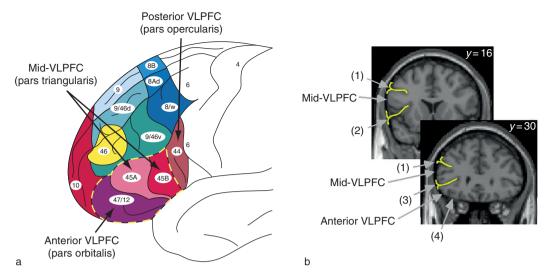
## Mnemonic Deficits Following Lateral Prefrontal Damage

Neuropsychological studies in humans and lesion studies in animals indicate that insult to the lateral PFC can produce memory impairments that are qualitatively distinct from those observed following MTL damage. Whereas MTL damage results in an amnesic condition that reflects the inability to encode and retrieve new declarative memories long-term memories for events (episodic memory) and facts (semantic memory) - lateral PFC damage impairs the strategic regulation of multiple forms of memory, including declarative memory and working memory. Significantly, impairments in episodic memory, semantic memory, and working memory following lateral PFC damage are often most apparent when performance requires the resolution of interference, which led Moscovitch, Shimamura, and others to propose that lateral PFC subserves cognitive control mechanisms that regulate how we work with or dynamically filter memory.

## **Episodic Memory Deficits**

Patients with lateral PFC damage show modest impairments in their ability to encode and retrieve episodic memories, with these deficits being particularly apparent when memory for target information is required in the face of distraction. Here we illustrate these deficits by highlighting a number of welldocumented paradigms in which PFC patients are impaired.

First, PFC patients show disproportionate deficits on tests of free recall compared to item recognition. Theorists have argued that the unconstrained nature of free recall increases the likelihood of mnemonic competition or interference. Second, even within recognition tests, PFC patients show impairments when distracter items at test (foils) are similar to items from the study list. In other words, PFC patients suffer interference from items that are similar to those they studied, as evidenced by false claims of having studied these items. Third, in tests of source memory, in which memories for individual items must be attributed to a particular learning context - a discrimination that presents high interference because irrelevant contexts are often highly salient - PFC patients exhibit disproportionate deficits relative to tests of item recognition. Fourth, the use of AB-AC learning paradigms reveals that learning an AC association is disproportionately impaired in PFC patients (i.e., patients show a heightened sensitivity to proactive interference). In such paradigms, the patient's



**Figure 1** Anatomical divisions of the ventrolateral prefrontal cortex (VLPFC) in the human: (a) representation of the cytoarchitectonic subdivisions of the lateral prefrontal cortex (PFC); (b) coronal slices through the PFC depicting the anatomical boundaries that define the mid-VLPFC and anterior VLPFC. The subregions of the VLPFC include the pars orbitalis, pars triangularis, and pars opercularis, corresponding to the anterior VLPFC (area 47/12), mid-VLPFC (area 45), and posterior VLPFC (area 44), respectively. (Note that we use the term mid-VLPFC to refer to area 45 only, adopting anterior VLPFC to refer to area 47/12.) As shown in (b), both the anterior and mid-VLPFC lie ventral to the inferior frontal sulcus (1). In caudal slices, the mid-VLPFC is bounded ventrally by the insular sulcus (2), and in rostral slices it is bounded by the horizontal ramus of the lateral fissure (3). The anterior VLPFC is bounded ventrally and medially by the orbital gyrus (4). (a) Adapted from Petrides M and Pandya DN (2002) Comparative cytoarchitectonic analysis of the human and the macaque ventrolateral prefrontal cortex and corticocortical connection patterns in the monkey. *European Journal of Neuoscience* 16: 291–310. (b) From Badre D and Wagner AD (2007) Left ventrolateral prefrontal cortex and the cognitive control of memory. *Neuropsy-chologia* 45(13): 2883–2901.

memory for AC pairs is largely compromised by increased intrusions of the earlier learned AB associations. Moreover, even when sequentially studying two unrelated lists of items (i.e., the lists do not share common associates), PFC patients show disproportionate list 1 interference when trying to learn list 2. Finally, when PFC patients are provided with a subset of items from a previously studied list as retrieval cues (i.e., part-list cueing), the high salience of the provided items results in increased impairment in the patients' ability to recall the remaining (unpresented) items from the list. Collectively, these and other observations indicate that the ability to resolve interference in episodic memory is compromised following lateral PFC damage.

### **Semantic Memory Deficits**

Just as PFC patients show impairments in unconstrained episodic memory tests, they also show deficits in unconstrained tests of semantic retrieval. For example, patients show reduced total output on verbal fluency tasks, which involve freely generating as many unique exemplars as possible in response to a semantic or orthographic cue. This impairment is thought to partially reflect patients' heightened sensitivity to output interference stemming from the initially retrieved exemplars. Significantly, this interference-dependent deficit during semantic or lexical retrieval stands in contrast to the typically unimpaired ability of PFC patients to recognize semantic structure or evaluate semantic relationships. Beyond verbal fluency tasks, patients with damage to the left lateral PFC fail to show normal semantic priming when the meaning of a semantic cue (the prime) is a context-dependent homograph, compared to words with less ambiguous meanings. This impairment may be due to PFC patients failing to retrieve the appropriate stimulus meaning when the prime is ambiguous, thereby preventing priming from occurring. Both these deficits are consistent with the perspective that the lateral PFC is recruited during controlled semantic retrieval, as well as when interference between competing semantic or lexical representations must be resolved.

### **Working Memory Deficits**

In tests of working memory, PFC patients are often unimpaired, as measured by simple tests of verbal span. However, in tests in which maintenance is complicated by interference from irrelevant information, lateral PFC damage tends to result in impairment. For example, in delayed recognition tests, PFC patients are particularly sensitive to the presence of distracters during the delay. When compared to patients with damage to the MTL, PFC patients show impairments at all delay intervals as long as

distracters are present, whereas patients with MTL damage show impairments principally at long delays. A similar observation comes from studies of PFClesioned monkeys. Whereas it was initially believed that PFC-lesioned monkeys were unable to remember the location of a food item that was hidden for only a short delay, it was later shown that this apparent memory deficit could be eliminated if the delay period was held in total darkness. In other words, the monkey's memory deficit was a function of the interfering effects of visual input during the delay period. Strikingly, not only do PFC patients show impaired working memory performance due to distracters, but PFC patients also show exaggerated electrophysiological responses in cortical areas that process the modality of the distracting information, suggesting that when distraction is present, the PFC is responsible for gating activity in regions that would otherwise process the distracting information. Further evidence that lateral PFC damage results in an inability to filter or gate irrelevant distracting information comes from studies of negative priming. In such studies, healthy controls typically showed increased reaction times when processing a stimulus that was previously a distracter (i.e., these previous distracters showed negative priming). By contrast, frontal patients showed the reverse effect - facilitated processing of stimuli that previously served as distracters – which suggests that the patients had difficulty preventing the processing of the distracters.

# Functional Specificity within Lateral Prefrontal Cortex

Although data from frontal patients provide compelling evidence that one contribution of the lateral PFC to the strategic control of memory is to regulate interference, the lack of anatomical specificity that typically accompanies naturally occurring PFC lesions in humans often precludes the determination of the specific functional contributions of particular PFC subregions. In an attempt to specify the mechanistic contributions of the subregions of the VLPFC to the control of memory, we consider in the next section evidence from functional neuroimaging studies and from recent reports of patients with lesions focused on subregions of VLPFC. As we discuss, extant data implicate the mid-VLPFC as a key component of the neural circuitry that resolves mnemonic interference, be it within working memory, semantic memory, or episodic memory.

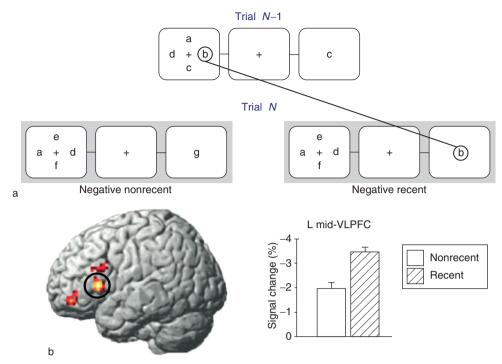
## Memory and the VLPFC

Focal lesion studies with monkeys have shown that damage to the inferior convexity (the monkey homolog of the human inferior frontal gyrus), but not to the DLPFC, causes perseverative tendencies in contexts in which the prior response learning must be reversed. Similar perseverative errors are a classic hallmark of human frontal lobe damage, as is often revealed through errors on the Wisconsin Card Sorting Task, when subjects must override a previously established, but now irrelevant, response set. As already noted, the strategic control of working memory, semantic memory, and episodic memory often requires conceptually similar mechanisms that permit the overriding of interference from irrelevant representations. Functional neuroimaging data in humans, complemented by focal lesion evidence, now indicate that the left mid-VLPFC regulates mnemonic interference by enabling the selection of relevant representations in the face of mnemonic conflict, whereas the left anterior VLPFC controls access to, and guides the retrieval of, long-term semantic knowledge. Response override or inhibition, by contrast, appears to differentially depend on regions in the human right VLPFC.

## Left Mid-VLPFC and Interference in Working Memory

Extensive evidence for the role of the left mid-VLPFC (BA 45) in resolving mnemonic interference comes from a variant of the Sternberg working memory paradigm (Figure 2(a)). In this task, participants encode a target set of stimuli (e.g., four letters), which they then attempt to maintain in working memory across a brief delay. Following the delay, a probe (e.g., a letter) is presented and participants make a yes/no decision as to whether the probe is in the currently maintained memory set. Critically, some negative probes (i.e., probes to which the subject should respond no) come from the immediately preceding memory set (negative recent probes), whereas other negative probes have not appeared in either the present or the preceding memory sets (negative nonrecent probes) (Figure 2(a)). Given this structure, negative recent probes are associated with interference because subjects must attribute the familiarity of the probe to its having been in the preceding memory set to correctly reject the probe as not being a member of the current memory set. Thus, comparisons of negative recent to negative nonrecent trials provide leverage on the neural mechanisms that are engaged in the face of this mnemonic interference.

Jonides and colleagues were the first to demonstrate that functional activation in the left mid-VLPFC is greater during negative recent relative to negative nonrecent trials, a pattern that has been replicated and extended by others (Figure 2(b)). Subsequent data indicated that (1) the response in the left mid-VLPFC during negative recent trials is



**Figure 2** Interference resolution in the Sternberg working memory task: (a) a representative version of the task; (b) representative fMRI data. As shown in (a), during the task subjects maintain a set of four letters in working memory until a probe letter appears, at which point subjects indicate whether the probe is a member of the currently maintained set (positive probe) or is not a member of the current set (negative probe). Interference occurs when a probe is not a member of the current set, but is a member of the immediately preceding set (negative recent probe), relative to situations in which the probe is a member neither of the current set nor of the immediately preceding set (negative nonrecent probe). In (b), the fMRI data reveal greater activation during negative recent vs. negative nonrecent trials in the left mid-VLPFC (circled), reflecting that the presence of interference in working memory is accompanied by greater recruitment of the left mid-VLPFC. fMRI, functional magnetic resonance imaging; L, left; VLPFC, ventrolateral prefrontal cortex. Adapted from Badre D and Wagner AD (2005) Frontal lobe mechanisms that resolve proactive interference. *Cerebral Cortex* 15: 2003–2012.

restricted to the probe period of the trial, which is consistent with the fact that interference is a function of the probe's familiarity, and (2) when directly interrogating the DLPFC, a similar difference between negative recent and nonrecent trials is not typically observed, suggesting a specific role of the left mid-VLPFC in resolving interference in this task. Moreover, in a compelling study motivated by the aforementioned neuroimaging data, Thompson-Schill and colleagues showed that PFC patients with damage that spared the left VLPFC demonstrated interference effects that were comparable in magnitude to those in healthy controls, whereas one patient, R.C., with a focal lesion that damaged almost the entire extent of the left BA 45, exhibited an exacerbated interference effect despite relatively normal working memory performance when interference was minimal. Complementing these data, Postle and colleagues observed that when repetitive transcranial magnetic stimulation (TMS) – which transiently disrupts cortical function – was applied to the left VLPFC during the probe period in healthy humans there was a similar increase in susceptibility to interference to that seen in patient R.C.

By contrast, TMS disruption of the primary motor, primary somatosensory, or supplemental motor area did not affect the ability to resolve interference in this task. Collectively, these data demonstrate that the left mid-VLPFC makes necessary contributions to resolving interference from currently irrelevant information in working memory. A leading hypothesis is that this region enables the selection of relevant active information over irrelevant active representations.

#### Left VLPFC and Declarative Memory Retrieval

Complementing the working memory literature implicating the left mid-VLPFC in resolving interference from recently activated but now irrelevant mnemonic representations are patient and imaging data that suggest that the left mid-VLPFC also supports selection during retrieval from declarative memory (i.e., semantic and episodic memory). For example, as already discussed, demands on interference resolution during semantic retrieval may be greater when available retrieval cues are unconstrained, and it is under these conditions that left PFC patients show impairments in generating semantic knowledge. Such impairments may reflect an inability to select relevant representations from among competing alternatives.

Functional neuroimaging and focal lesion data converge on the left mid-VLPFC as being central for mediating the selection of relevant semantic representations. For example, in an influential functional magnetic resonance imaging (fMRI) study, Thompson-Schill and colleagues compared semantic processing under conditions that varied the demands placed on selection. Across several tasks, low-selection conditions were constructed such that they involved retrieving dominant, or prepotent, semantic information, whereas high-selection conditions involved retrieving nondominant semantic information from among competing representations. Significantly, the imaging data revealed that the left VLPFC (BAs 44 and 45) was consistently more active during the high-selection (vs. low-selection) conditions, demonstrating that this is a replicable characteristic of left mid-and/or posterior VLPFC function. Subsequent neuropsychological work by Thompson-Schill and colleagues revealed that it is the proportion of damage to this left VLPFC region, but not the gross lesion size, that strongly predicts the magnitude of behavioral impairment on high-selection semantic retrieval tasks.

Related data have increased the precision of our understanding of left mid-VLPFC function and its relation to the functions of the surrounding anterior and posterior VLPFC subregions. In particular, Badre and colleagues demonstrated that selection demands during semantic processing tasks are specifically associated with the same left mid-VLPFC subregion (BA 45; pars triangularis) that resolves interference in working memory tasks (Figure 3(a)). Moreover, these researchers observed a functional dissociation within the left VLPFC, indicating that the left mid-VLPFC supports selection from among active representations, whereas the more rostrally and ventrally situated left anterior VLPFC subregion (area 47/12; pars orbitalis) controls the retrieval of semantic representations stored in the lateral temporal cortical areas. This functional distinction between the left mid-VLPFC and anterior VLPFC appears replicable and generalizeable; Gold and colleagues reported a similar functional pattern using a lexical decision task - namely that the left anterior VLPFC was associated with controlled semantic retrieval and the left mid-VLPFC was associated with resolving interference from irrelevant active representations.

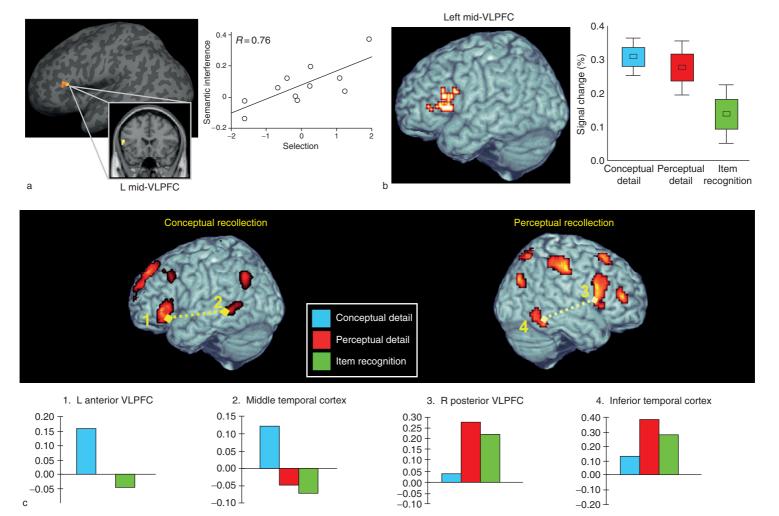
Evidence for functional segregation within the left VLPFC has also come from studies comparing semantic versus phonological control. For example, neuroimaging studies of phonological rehearsal and

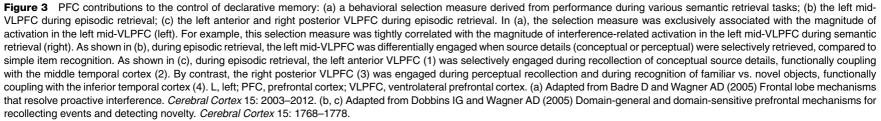
phonological analysis of stimuli have implicated the left posterior VLPFC (BA 44; pars opercularis), suggesting that this VLPFC subregion is critical for representing and maintaining phonological codes. On the other hand, neuroimaging studies of semantic retrieval and analysis of stimuli have implicated the left anterior VLPFC. Significantly, these dissociations between the left anterior and posterior VLPFC have been observed in studies that directly contrasted tasks that differentially depend on semantic and phonological control. Moreover, Devlin and colleagues have shown that TMS to the left anterior VLPFC differentially disrupts concurrent semantic processing, whereas TMS to the left posterior VLPFC differentially disrupts concurrent phonological processing, demonstrating that these subregions are necessary for distinct forms of control.

Functional distinctions between the left VLPFC subregions are also apparent during tasks that probe episodic memory. For example, Dobbins and colleagues observed that the left mid-VLPFC is associated with selecting between episodic details in the course of making a source judgment (Figure 3(b)), whereas the left anterior VLPFC is associated with semantically elaborating on the cues used to probe episodic memory. Further, during episodic encoding, Dolan, Fletcher, and colleagues demonstrated that left mid-VLPFC activity increases when prior learning interferes with to-be-encoded information (i.e., when the resolution of proactive interference is required). Collectively, these data suggest that the left mid-VLPFC contributes to the resolution of interference during all forms of declarative memory, whereas the left anterior VLPFC controls the retrieval of semantic knowledge that is not retrieved in an automatic (bottom-up) manner. Moreover, when considered along with the evidence that the left mid-VLPFC resolves interference within working memory, extant data favor the hypothesis that the left mid-VLPFC serves a domain-general role of resolving mnemonic interference.

### **Right VLPFC and the Control of Memory**

We have thus far focused on how the left VLPFC contributes to interference resolution and other forms of cognitive control; however, it should be emphasized that the right VLPFC is sometimes coactive with the left VLPFC during tasks that require overcoming mnemonic conflict. Nevertheless, although the right VLPFC may support control functions that are conceptually analogous to those supported by the left VLPFC, it appears that (in the human) the right VLPFC at least partially differs from the left VLPFC in the domain of knowledge on which is operates. For example, Aron and colleagues, among others,





reported that functional activation in the posterior aspect of the right VLPFC is consistently implicated in situations in which prepotent motor responses must be inhibited or when well-learned stimulus-response contingencies must be reconfigured. Moreover, these researchers demonstrated that when the right VLPFC is damaged, subjects display increased susceptibility to interference at the response level, highlighting the necessary contribution of this VLPFC subregion to response inhibition.

By contrast, other data indicate that the right VLPFC may contribute to the orienting of visual attention toward task-relevant object representations (and away from distracters). Significantly, with respect to the control of memory, this latter role of the right VLPFC in orienting to visuo-object representations has been observed in a number of mnemonic contexts, including discriminating novel from encountered visual objects, encoding faces and complex visual scenes into episodic memory, recollecting object-object associations, and recollecting perceptual details about previously encountered objects (Figure 3(c)). Thus, the extent to which the right VLPFC (as opposed to the left VLPFC) is engaged at least partially depends on the modality of the mnemonic representations attended, with the right VLPFC preferentially engaged during visuospatial processing and the left VLPFC engaged during phonological and semantic processing (Figure 3(c)). Significantly, although the right VLPFC has been implicated in visuo-object attention and in resolving competition at the response level, functional understanding of the subregions within the right VLPFC is not as well advanced as that of the homologous structures in the left VLPFC.

## **Concluding Comments**

Interference resolution is a fundamental aspect of efficient mnemonic processing and may represent a principal way in which cognitive control interacts with memory. This interaction is critically enabled by control mechanisms that depend on the lateral PFC for their operation. This article focuses on the delineation of the mechanisms supported by the VLPFC, but it should be noted that the VLPFC must dynamically interact with the posterior neocortical association areas and with the MTL to implement the control of memory. Further, although VLPFC functions are clearly important for the resolution of mnemonic interference, the VLPFC also interacts with the DLPFC and frontopolar cortical areas that subserve other forms of cognitive control. Next we briefly illustrate each of these points.

To regulate memory-relevant processes, PFC control mechanisms must interact with systems that support or

store long-term mnemonic representations. One line of evidence for such frontal-posterior interactions comes in the form of coactivations within functional neuroimaging studies. For example, fMRI studies of episodic remembering indicate that the left anterior VLPFC coactivates with the left middle temporal cortical areas that represent conceptual knowledge during controlled attempts to recollect such details about the past (Figure 3(c)). Analogously, the right VLPFC coactives with bilateral occipito-temporal areas that represent visuo-object form during attempts to recollect such details (Figure 3(c)). Moreover, as previously alluded to, lesions to the lateral PFC result in the failure to regulate processing in such posterior neocortical areas, demonstrating that the lateral PFC is necessary for gating the processing of distracting or interfering stimuli. Collectively, these data illustrate the top-down role of PFC control mechanisms in regulating perception, memory, and action.

With respect to cross-regional interactions within the PFC, it is important to emphasize that other structures in the DLPFC and frontopolar cortex are also often engaged in situations in which control must be implemented to accomplish a mnemonic goal. For example, DLPFC activation is frequently observed during complex working memory tasks, during attempts to remember past events, and during many tasks that require response selection or the resolution of response conflict. Although the specific nature of interactions between the DLPFC and VLPFC have yet to be well characterized, it is hypothesized that the DLPFC may operate at a higher stage in the processing hierarchy relative to the VLPFC, serving to perform operations on the products of VLPFC processing.

Thus, it is clear that the strategic control of memory is multifaceted. Future research promises to further illuminate how cognitive control emerges through lateral PFC function, allowing us to strategically wrest control of our memories, bringing them in line with current goals.

See also: Cognition: An Overview of Neuroimaging Techniques; Event-Related Potentials (ERPs); Inhibitory Control over Action and Memory; Neuroimaging; Prefrontal Cortex: Structure and Anatomy; Prefrontal Cortex; Short Term and Working Memory; Spatial Cognition and Executive Function; Working Memory: Capacity Limitations.

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