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Capacity limits of information processing in the brain

René Marois and Jason Ivanoff

Department of Psychology, Center for Integrative and Cognitive Neurosciences, Vanderbilt Vision Research Center, Vanderbilt University, Nashville, TN, USA

Despite the impressive complexity and processing power of the human brain, it is severely capacity limited. Behavioral research has highlighted three major bottlenecks of information processing that can cripple our ability to consciously perceive, hold in mind, and act upon the visual world, illustrated by the attentional blink (AB), visual short-term memory (VSTM), and psychological refractory period (PRP) phenomena, respectively. A review of the neurobiological literature suggests that the capacity limit of VSTM storage is primarily localized to the posterior parietal and occipital cortex, whereas the AB and PRP are associated with partly overlapping fronto-parietal networks. The convergence of these two networks in the lateral frontal cortex points to this brain region as a putative neural locus of a common processing bottleneck for perception and action.

The human brain is heralded for its staggering complexity and processing capacity: its hundred billion (10^{11}) neurons and several hundred trillion synaptic connections can process and exchange prodigious amounts of information over a distributed neural network in the matter of milliseconds. Such massive parallel processing capacity permits our visual system to successfully decode complex images in 100 ms [1], and our brain to store upwards of 10^9 bits of information over our lifetime [2], more than 50 000 times the text contained in the US Library of Congress. Yet, for all our neurocomputational sophistication and processing power, we can barely attend to more than one object at a time, and we can hardly perform two tasks at once.

A rich history of cognitive research has highlighted three major processing limitations during the flow of information from sensation to action, each exemplified by a specific experimental paradigm. The first limitation concerns the time it takes to consciously identify and consolidate a visual stimulus in visual short-term memory (VSTM), as revealed by the attentional blink paradigm. This process can take more than half a second before it is free to identify a second stimulus. A second, severely limited capacity is the restricted number of stimuli that can be held in VSTM, as exemplified by the change detection paradigm. Finally, a third bottleneck arises when one must choose an appropriate response to each stimulus. Selecting an appropriate response for one stimulus delays by several hundred milliseconds the ability to select a response for a second stimulus (the 'psychological refractory period').

To be sure, these are not the only processes exhibiting capacity limitations. Indeed, it can be safely argued that *all* processing stages are capacity limited. However, these three bottlenecks are arguably the most severe ones that can impair our ability to be aware of, hold in mind, and act upon visual information. A recent flurry of neuroimaging studies, together with earlier brain lesion and electrophysiological work, have begun to unravel the neural underpinnings of these bottlenecks, and several recent behavioral studies have made great strides in isolating the underlying cognitive processes. The purpose of this article is to review our current understanding of the neurobiology of these bottlenecks of human information processing in the context of their extant cognitive models.

Capacity limit in explicit visual event detection: the attentional blink

Virtually all models of visual cognition distinguish between capacity–unlimited and capacity-limited stages of information processing (e.g. [3,4]). In these two-stage models, an early stage permits the rapid, initial evaluation of the visual world, whereas later attention-demanding, capacity-limited stages are necessary for the conscious report of the stimuli. The dual nature of visual cognition is well illustrated by the attentional blink (AB) paradigm: when subjects attempt to identify two targets in a rapid, serial visual presentation (RSVP) of distractors, they are severely impaired at detecting the second of the two targets when it is presented within 500 ms of the first target (Figure 1) [5]. Importantly, the deficit with the second target (T2) is a result of attending to the first target (T1): subjects have no difficulty in reporting T2 when only it is required to be detected.

What exactly is the capacity limited process revealed by the AB? Some models suggest that the AB bottleneck is inherently attentional, revealing either the necessary time an item must be attended before other items can be attended [6], or the time required to switch attention from the first to the second target [7], whereas others propose that the AB is related to the capacity-limited stage of encoding targets in VSTM [3,8,9] (Box 1). Another important issue pertains to the location of this bottleneck along the visual information processing pathway. Some

Corresponding author: Marois, R. (rene.marois@vanderbilt.edu).
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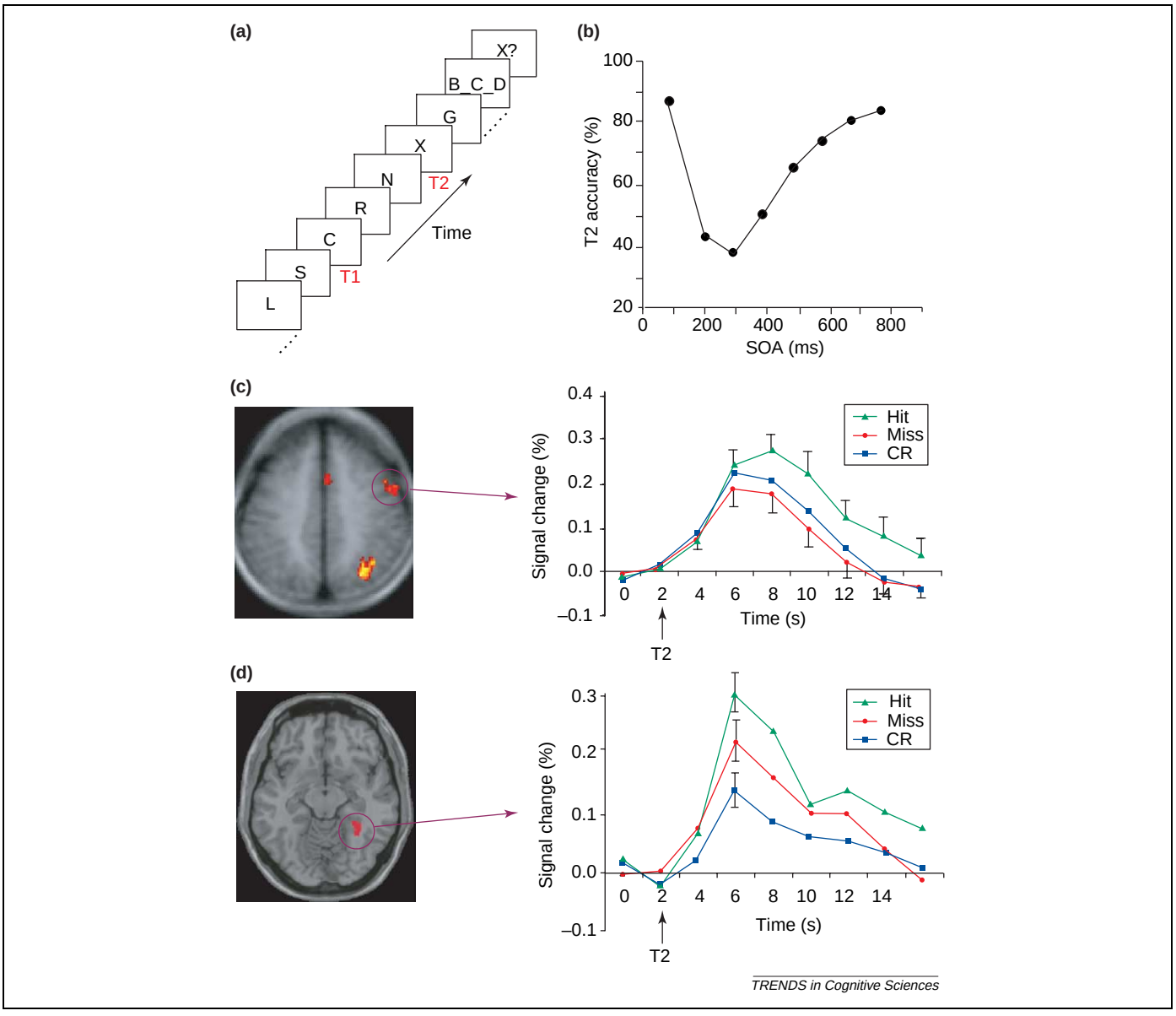

Figure 1. Neural correlates of the attentional blink (AB). **(a)** The attentional blink paradigm. Subjects search for two target letters (T1 and T2)) presented among distractors, and respond at the end of each trial. **(b)** Standard T2 accuracy performance by stimulus onset asynchrony (SOA) between T1 and T2 for trials with correctly identified T1. **(c)** Left: brain activations in frontal and parietal cortex associated with T1 manipulations that affect the magnitude of the AB [16]. Right: activation time course in the lateral frontal cortex with Hit, Miss, and Correct Rejection (CR) T2 trials for the Scene-AB experiment of Marois *et al.* [17]. **(d)** Left: location of the scene-selective region in the parahippocampal gyrus. Right: activation time course in this region with Hit, Miss, and Correct Rejection T2 trials for the Scene-AB experiment [17].

studies suggest that the AB occurs primarily in modality-specific information processing stages [10], before central amodal stages of processing. Consistent with this notion, little or no AB is obtained when the two targets originate from different modalities (e.g. visual and auditory), provided subjects are simultaneously monitoring both streams and all targets belong to the same stimulus category [11,12]. By contrast, other results suggest that the AB bottleneck is affected by late, response-related stages of processing, because increasing the response demands of T1 exacerbates the AB [8,13,14]. Thus, the AB is not only affected by modality-specific factors, it is at least partly affected by a late amodal stage of information processing corresponding to response selection (see below).

The neural basis of the AB bottleneck

Two-stage models of the AB have received considerable support from event-related potential (ERP) studies. T2 target words that are not explicitly perceived in an AB paradigm nonetheless elicit an N400, an electrophysiological marker of semantic processing [15], whereas successful detection of T2 elicits a P300 [9], an electrophysiological marker of working memory updating. Thus, T2 targets that fail to reach working memory are nevertheless processed up to the semantic level.

Functional magnetic resonance imaging (fMRI) studies complement the ERP studies by pinpointing the neural substrates underlying the AB bottleneck. T1 manipulations known to modulate the attentional blink (e.g. distractor interference [16]) recruit the lateral frontal, intra-parietal and anterior cingulate cortex

Box 1. Capacity limits of attention or information processing?

It is generally accepted that our brain cannot process all the information with which it is bombarded, and that attention is the process that selects which stimuli/actions get access to these capacity-limited processes [83,84]. In this view, attention can selectively act at multiple stages of information processing [84] and operate differently in each stage according to the processing characteristics of that stage [84]. Such view implies that capacity limits of information processing reside primarily in content-specific information channels. However, attention has also been conceived as a capacity-limited resource allocator [85] with dedicated neural substrates [86,87]. As a result, some cognitive phenomena have been alternatively interpreted as revealing a capacity limit of attention and of a content-specific information channel. For instance, whereas some see the attentional blink as revealing the temporal capacity limits of attention [6,88], others view it as the capacity limit of visual short-term memory consolidation [3,62,77]. This issue is not merely semantic. Performance limitations may result from an interaction between capacity-limited content-specific information channels and capacity-limited attentional processes. Such a view has been put forward to account for VSTM storage capacity [37,38]. Thus, one important challenge in studying bottlenecks of information processing is to disentangle attentional capacity limits from the capacity limits of content-specific information channels.

(Figure 1c). This fronto-parietal network has therefore been proposed to represent the neural locus of the capacity-limited process underlying the AB deficit [16]. The ERP studies and the fronto-parietal locus for the AB bottleneck also predict that T2 items that go undetected should nonetheless activate the visual cortex, whereas the fronto-parietal cortex should be recruited primarily when the T2 target is consciously reported. These predictions were confirmed in a recent AB fMRI experiment [17] (see also [18]). Undetected T2 targets still activated a region of visual cortex supporting high-level scene categorization (Figure 1d). This activation was further amplified when T2 was correctly identified. By contrast, the lateral frontal cortex was activated only when T2 was successfully reported [17]. These results are consistent with the idea that visual stimuli suffering from an attentional blink are nonetheless deeply processed by the brain [15], and that visual cortex activation can occur without awareness [19,20]. In addition, the amplification of activation with conscious T2 detection also suggests that visual cortical areas may contribute to the AB bottleneck as well, and could in principle account for lower-level perceptual factors that affect the AB [21]. The results also further implicate the lateral frontal [17,18] and parietal cortex [18] as likely neural substrates of the AB bottleneck.

Several additional studies with different methodological approaches converge in implicating a fronto-parietal network in the AB bottleneck. Brain lesions of the right inferior parietal cortex and lateral frontal cortex exacerbate the attentional blink [22,23], and trans-cranial magnetic stimulation (TMS) over the posterior parietal cortex lessens the AB [24]. Furthermore, a recent fMRI study using a stimulus-driven form of the AB in which T1 is replaced by an unexpected novel stimulus revealed activation in inferior parietal and lateral frontal cortex that correlated with the magnitude of the AB (Marois, R., Snyder, A., Gilbert, C., and Todd, J.J., unpublished). Finally, a magneto-encephalography study indicates that successful target detection in an AB paradigm is associated with increased neural synchronization over a fronto-parietal network nearly 300 ms after target presentation [25], a time frame that is consistent with the P300 signature of the AB [9].

This body of work is strikingly consistent with two-stage models of the AB, which propose that stimuli are initially characterized at an early stage of visual information processing, followed by a second, capacity-limited attention-demanding stage required for conscious report [3]. The visual cortex might subserve the initial stage of stimulus categorization whereas the lateral frontal and parietal cortex might reflect the neural locus of the attention-demanding, capacity-limited stage of information processing [26]. If conscious perception arises from an interaction between sensory/perceptual areas and attentional/working memory networks [20,27], then it is possible that the second stage may require visual cortex involvement as well.

Capacity limits of visual short term memory (VSTM)

Our visual cognition is not only limited by the *rate* at which information can be attended or consolidated into VSTM, it is also limited by the *amount* of information that can be stored in VSTM. Although the capacity of VSTM is generally estimated to be about 4 items [28–30], it is set not only by the number of objects but also by the complexity of each object [31]. This capacity limit has significant behavioral consequences. For instance, VSTM storage capacity is thought to be at least partly responsible for our inability in detecting a gross change between two scenes that are otherwise identical ('change blindness') [32,33].

Just as dual-task interference and brain lesion studies indicate that short-term memory can be parceled out into largely distinct visual and auditory buffers [34], VSTM can in turn be subdivided into at least two separate stores, one for object-based information and one for location-based information [35,36]. There may even be independent stores for each distinct visual features (e.g. color, stimulus orientation) [37,38].

Attention is likely to be an important component of VSTM storage capacity. Diverting attention away from the location of an item maintained in spatial VSTM interferes with memory performance [39]. Such result has led to the proposal that, just as verbal working memory consists of an 'articulatory loop' that includes a subvocal rehearsal system and phonological stores [34], VSTM may also involve both an attention-based maintenance process and visual stores [39]. Thus, VSTM may not only be limited by the independent capacity of feature stores, but also by demands on attention to integrate this distributed information into unified objects [37,38]. Another link between VSTM and attention is that they share roughly similar monitoring capacity limits. The ability to attentively track several randomly moving targets among distractors is limited to about 4–5 targets (multiple-object tracking [MOT] task [40]), a number that is strikingly similar to VSTM's storage capacity limit [28–30]. The close relationship between attention and working memory is

also reflected in several theories of VSTM storage capacity that propose that attention is the underlying limiting factor [28,32,33].

In summary, recent work paints a fragmented view of VSTM, with distinct stores and storage capacities for different material, and with the capacity limit of VSTM resulting from an interaction between the capacities of these individual stores and attention.

The neural basis of VSTM storage capacity limits

Visual working memory is mediated by a network of brain regions distributed across the cerebral cortex [41–43]. This network can be loosely divided into regions that contribute to Baddeley's [34] central executive system, and regions involved in maintaining the information in working memory (i.e. VSTM). Executive processes, such as encoding, manipulation and retrieval of information from working memory, have been primarily associated with the frontal/prefrontal cortex [44,45]. By contrast, maintenance of sensory information in working memory is more regionally distributed, with different types of working memory maintenance (e.g. verbal, visual) associated with different cortical substrates [46]. However, even the simple maintenance of information in working memory recruits a distributed network of brain regions [41–43], and one may therefore also expect that the limit of VSTM capacity to be a widely distributed property as well.

In contrast to these expectations, two complementary studies suggest that the capacity limit of VSTM storage may be relatively localized to the posterior parietal and occipital cortex [47,48]. Both studies used the change detection paradigm [29,30], in which participants detect if there is a change in object identity and/or location between two briefly presented visual arrays separated by a short retention interval (Figure 2a). The fMRI study revealed that activity of the posterior parietal/superior occipital cortex strongly correlated with the number of objects stored in VSTM [47] (Figure 2b,c), whereas the electrophysiological study showed a striking correlation between amplitude of ERPs and individual differences in VSTM capacity at parietal and occipital electrode sites [48].

Taken together, these results suggest that VSTM storage capacity is primarily localized to the posterior parietal and occipital cortex. Importantly, these findings were obtained when the task involved encoding both object identity (color) and location [47]. By virtue of its central role in visuo-spatial attention and working memory [49] and in visual feature integration [50,51], the posterior parietal cortex is well positioned to build an integrated mental representation of the visual scene. Thus, the posterior parietal/superior occipital cortex might contribute an attention-based component to VSTM [28,32,37,38]. This notion is further supported by the partial overlap in parietal cortex activation between VSTM load and attentional load, as studied with the MOT task [52,53]. Nevertheless, this brain region might not be solely involved in spatial working memory, as it can also be recruited for object-based VSTM [42].

Although the localization of VSTM storage capacity limitations to posterior cortical regions is consistent with recent models suggesting that VSTM stores are not located in frontal/prefrontal cortex [44,45], it is also not inconsistent with the involvement of these anterior brain regions in visual working memory. Frontal/prefrontal cortex may serve to maintain task-specific goals [44,54] or assist VSTM-related processes at high loads and/or long duration intervals [42]. In addition, given the behavioral and neuro-anatomical dissociations between object and spatial working memory systems, different forms of VSTM storage capacity limit could very well be associated with partly distinct neural substrates. Furthermore, if VSTM storage capacity is a product of the interaction between attentional mechanisms and the independent capacity of distinct feature stores [37,38], feature-processing regions of visual cortex may also contribute to VSTM capacity (Figure 2). Thus, as more types of VSTM storage capacities become the focus of neuroimaging investigations, distinct foci of the visual cortex may turn out to be associated with VSTM.

The limits of response selection: the psychological refractory period

A third major bottleneck of information processing takes place when an appropriate action has to be selected in response to a stimulus. This is evidenced by the psychological refractory period (PRP) paradigm [55]: when subjects are required to perform two sensorimotor tasks in rapid succession, the response to the second task is increasingly delayed as the stimulus onset asynchrony (SOA) between the two tasks decrease (Figure 3a). This occurs even when sensory and motor modalities are distinct for the two tasks (e.g. audio-manual Task 1 and visual-vocal Task 2), suggesting a central, amodal origin to the dual-task cost.

Several lines of evidence suggest that the PRP is the result of a bottleneck occurring at the stage of response selection (RS) (Figure 3b) [55]. However, theories differ as to the cause of this bottleneck. A primary source of contention concerns the degree to which the bottleneck reflects an inherent structural inability to concurrently select two responses [56], versus a cognitive strategy controlled by an executive system to optimize performance in dual-task conditions (Figure 3c) [57,58]. The cognitive strategy hypothesis has drawn support from the finding that dual-task costs are not immutable: they can be significantly reduced provided the component tasks are given equal priority and are highly practiced [59] (but see [60]). However, the effect of practice on the PRP can be explained both by cognitive strategy and structural bottleneck accounts, as they might reflect learning by the executive system to perform two tasks concurrently without interference [59], or the shortening of the response-selection stage with practice [61]. If the PRP results from an inability to simultaneously retrieve two sets of response selection rules from working memory [62], and if practice automatizes retrieval [63], then it should reduce the PRP. Elucidating how over-learning stimulus–response associations reduces the PRP is likely to provide key insights into the nature of this bottleneck.

Another source of contention concerns how the PRP bottleneck is implemented. Structural and strategic models presume that multiple response selections are

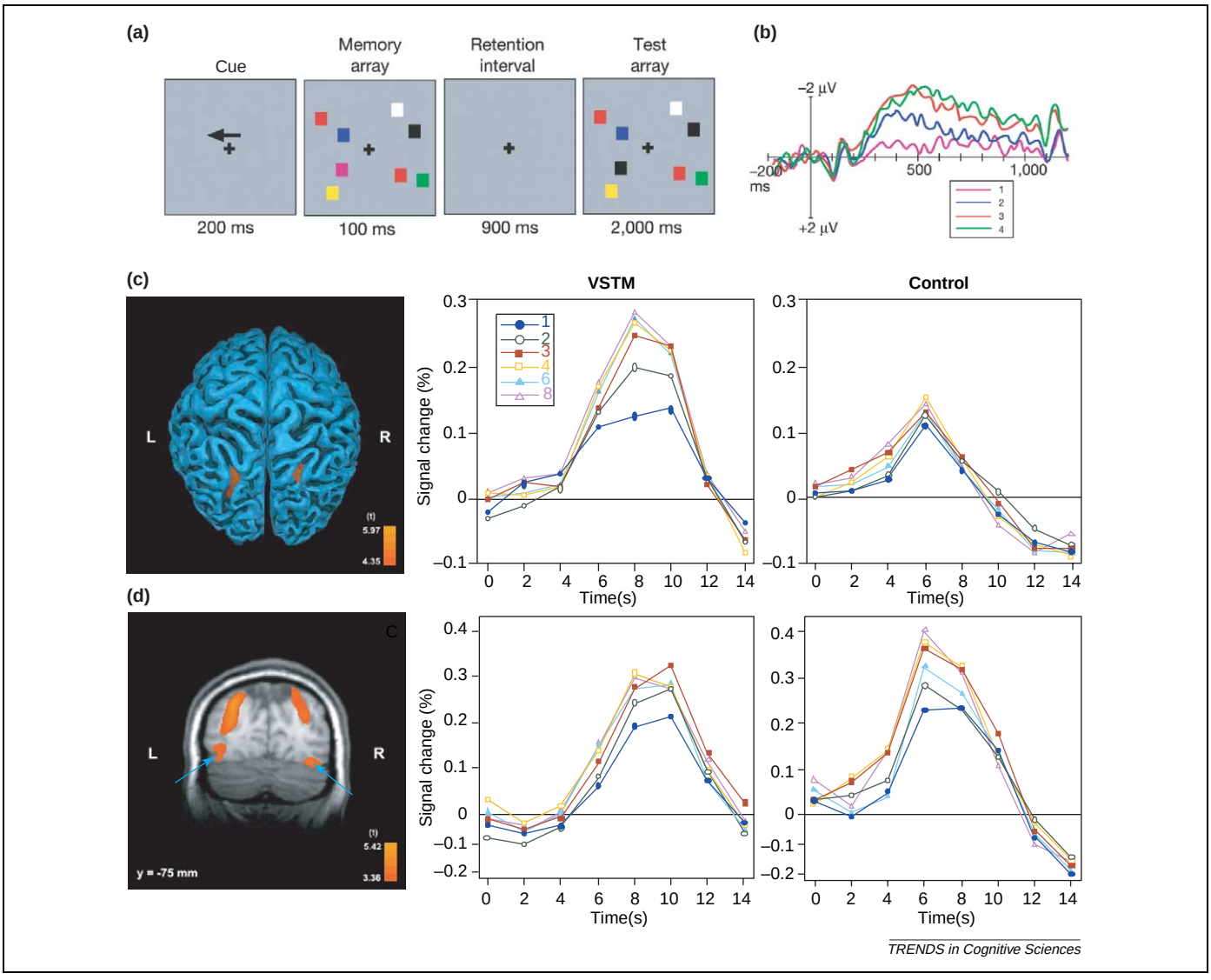

Figure 2. Neural correlates of visual short-term memory (VSTM) storage capacity. **(a)** Change detection paradigm from the VSTM load study of Vogel and Machizawa [48]. Subjects compared a memory array with a test array shown after a retention interval for an item change between the two arrays (here, cerise to black square). The cue instructed which side of the memory array subjects were to encode. The set size of the arrays varied between trials. **(b)** ERP difference waves (Cued side–Uncued side) at lateral occipital and posterior parietal electrode sites showing amplitude increases between memory loads of one to three items, but not between three and four items [48]. **(c)** Left: using a similar task, Todd and Marois (2004) [47] observed that posterior parietal/superior occipital activation correlated with VSTM storage capacity. Middle and Right: activation time courses in posterior parietal cortex by set size (1–8) shows a load effect in the VSTM task (Middle) but not in a perceptual control task (Right) using the same display. **(d)** By contrast, an extrastriate color area (arrows) responds similarly to the VSTM and perceptual tasks. However, the response also shows evidence of saturation at higher loads, which might correspond to the capacity limit of a feature-specific store (see text).

carried out serially. By contrast, shared resource models argue that response selections occur simultaneously for both tasks, but with processing resources differentially weighted for one task over the other, resulting in a lag between Task 1 and Task 2 reaction times (RT) (Figure 3d) [64,65]. Thus, models of the PRP may be regarded as falling along two continuums. The first considers the extent to which the cost is the result of purely structural or purely strategic processes, and the second concerns the extent to which the performance cost is the result of sharing a limited capacity resource or whether it reflects a serial bottleneck of information processing.

Neural correlates of the psychological refractory period

Electrophysiological studies of the PRP have established the temporal boundary of the PRP bottleneck to a stage between stimulus consolidation in working memory and motor preparation [66,67], and split-brain patient studies implicate a sub-cortical component to the PRP [68,69]. By contrast, imaging studies have primarily aimed at localizing the cortical substrates of the PRP. Although the neural basis of dual-task interference has received considerable attention over the past 10 years [70], only imaging studies of the PRP are considered below.

One experimental approach to the PRP consists by contrasting dual-task with single-task conditions. This approach is well-suited to addressing whether dual-tasking recruit brain regions beyond those activated under single-task conditions [70]. Importantly, it is conceptually biased towards the central executive/cognitive strategy models of the PRP as they assume that executive demands (e.g. task prioritization) should be primarily, if not exclusively, recruited under dual-task conditions. Correspondingly, several studies [71–73] have

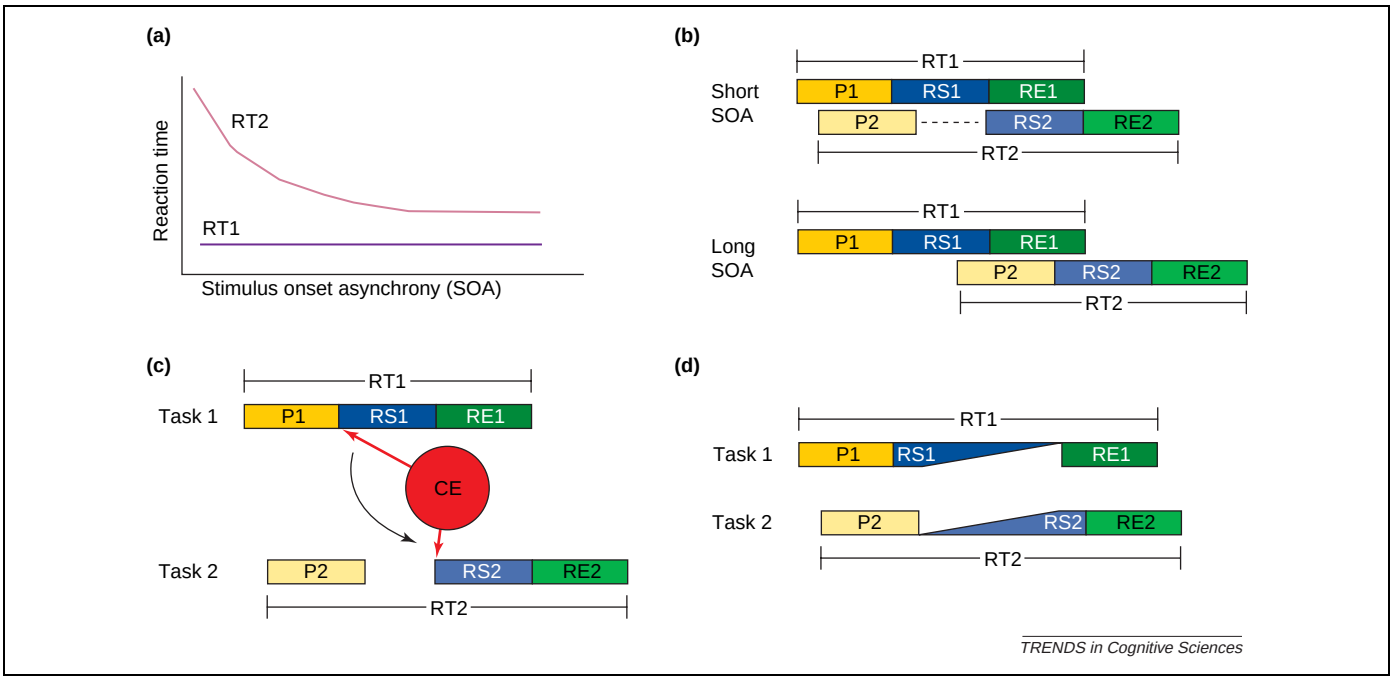

Figure 3. Models of the Psychological Refractory Period (PRP). **(a)** The PRP refers to the increase in reaction time to the second target (RT2) when the stimulus onset asynchrony (SOA) between the two tasks is shortened. **(b)** The classical interpretation of the PRP effect [55]. Perceptual processing of stimulus 1 (P1) and stimulus 2 (P2) occur in parallel, but response selection for task 2 (RS2) is deferred (dotted line) at short SOAs until response selection for task 1 (RS1) is completed. Response execution (RE) follows RS. Although each process box is presented in a serial fashion, information flow between boxes can be discrete or continuous [57]. The classical model assumes the PRP results from a structural bottleneck: RS1 and RS2 occur serially because they require activation of a common neuroanatomical hub that can only select one response at a time, although this hub may take the form of a subcortical/cortical network [89]. **(c)** Central executive (CE) models assert that RS1 and RS2 occur serially because this is the optimal strategy to avoid poor performance [57,58]. **(d)** Capacity sharing models presume that RS1 and RS2 can co-occur, but that an initial asymmetric allocation of resources to RS1 leads to its completion earlier than RS2 [64,65].

reported frontal and parietal cortical activations under dual-task conditions. These activations might reflect the executive control needed to co-ordinate dual-task performance, but they could also be related to executive processes (e.g. task-switching) that are not directly responsible to the dual-task slowing observed in the PRP.

Another fMRI experimental approach consists by contrasting activations obtained at short and long SOAs. This approach is directly comparable to the behavioral method for assessing the PRP (i.e. RT difference between short and long SOA). However, such contrast is relatively insensitive to response selection bottleneck and graded resource accounts of the PRP because the total duration of response selection processing should be identical for both short and long SOAs. Instead, the SOA contrast is suitable for detecting brain regions whose activity correlate with the magnitude of dual-task interference, either for detecting and/or resolving the interference. One such brain region might be the right inferior frontal area, as two studies reported greater activation of this area at shorter SOAs [71,74]. However, these studies conflicted with regard to the relationship between right inferior frontal activation and dual-task costs, and yet another study suggests that this brain region might be more involved in resolving interference in visuo-spatial attention than in response selection [75], casting further doubt on its involvement in the PRP bottleneck per se.

A final fMRI approach assessed whether activity in brain regions involved in response selection correlates with the magnitude of the PRP. This approach specifically tests the serial bottleneck hypothesis, for the duration of the PRP is believed to be directly proportionate to the duration of response selection in Task 1 [55]. On the other hand, this approach does not strongly distinguish between executive/strategic and structural bottleneck/graded resource accounts of the PRP, and is insensitive to models that assume the specific recruitment of brain regions under dual-task conditions. In one such study, regions of the lateral frontal, medial frontal, premotor, and parietal cortex involved in response selection were first isolated, and then probed in a dual-task study that manipulated the magnitude of the PRP by varying the number of response selection alternatives [76]. Activity in the lateral frontal and medial frontal cortex correlated with the magnitude of the PRP [76]. These results not only pinpoint a putative neural locus of the PRP, but also provide neurobiological support for the serial bottleneck account of the PRP [55].

It should be clear from this review that not a single fMRI experimental approach may be sufficient to uncover the neural basis of dual-task limitations, and that each approach is biased in assessing specific models of the PRP. In addition, despite the fact that the critical measure of the PRP is reaction time, these imaging studies have relied on activation levels. Nevertheless, it is remarkable that several of these fMRI studies recruited comparable regions of lateral frontal and dorsal premotor cortex (Figure 4), although it is currently unclear what, if any, role(s) these areas play in the PRP (see also below).

Convergence of processing limitations?

This review has so far discussed capacity limits in encoding visual information in working memory (AB), in

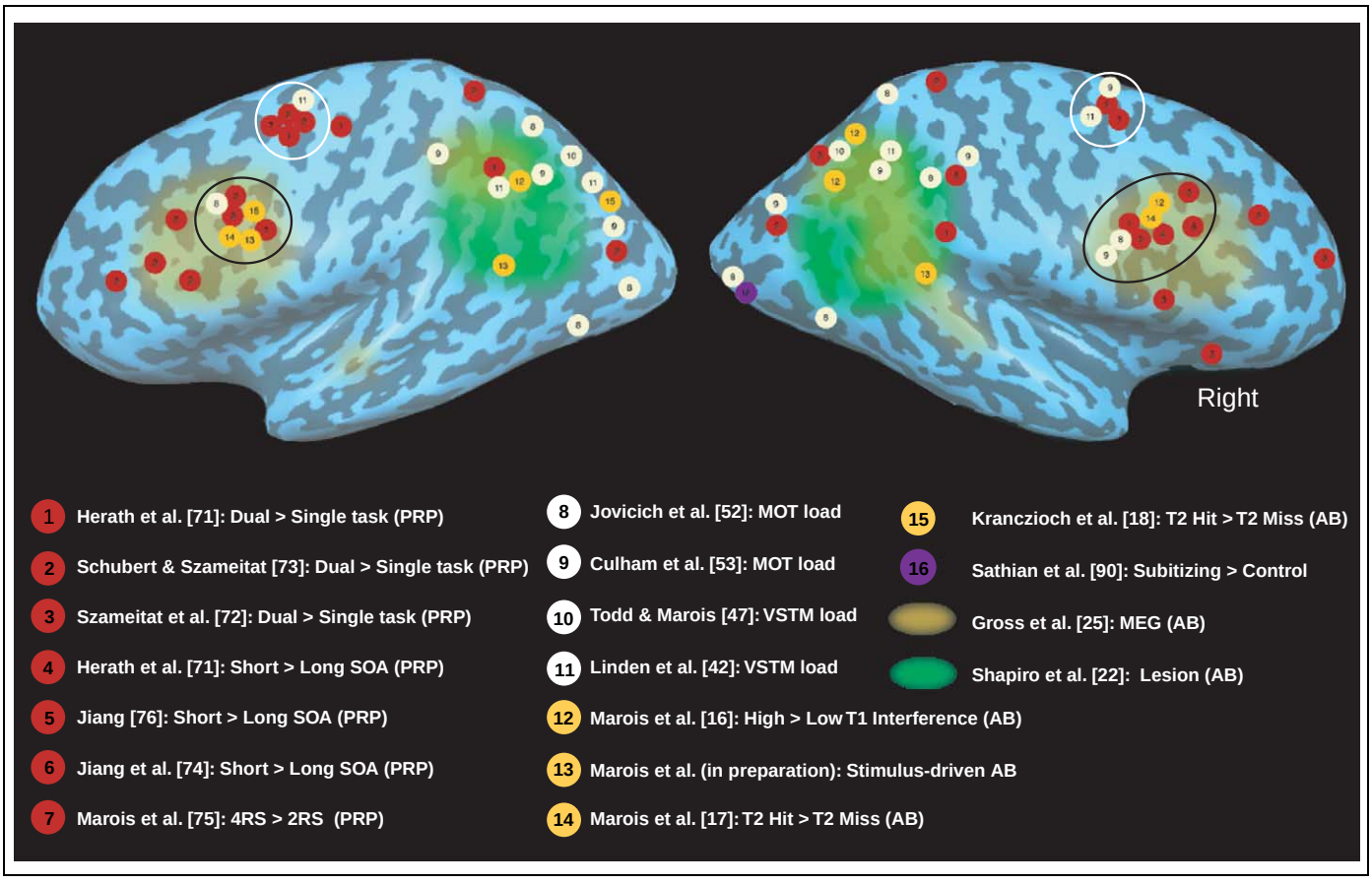

Figure 4. Localizing the sites of processing bottlenecks in human cerebral cortex. Activation foci in fMRI studies of the PRP, MOT, VSTM, and AB on lateral views of an inflated brain. Also included are the approximated extents of a lesion and an MEG study of the AB (light- and dark-green shaded regions, respectively). Several foci from AB and PRP studies cluster in lateral frontal cortex (black ellipse). By contrast, the postero-lateral parietal cortex is primarily dotted with perceptual activations (AB, MOT, VSTM), whereas a dorsal premotor cortex area (white ellipse) includes PRP and load studies, but no AB. Red-, white-, and gold-colored circles refer to PRP, VSTM/MOT, and AB fMRI studies respectively, with each study source and fMRI contrast of interest listed in the key below. Localization of the activated foci was primarily based on Talairach coordinates. The VSTM studies only include those that examined capacity limits of VSTM storage for non-linguistic visual material. Scattered activation foci in lateral temporal and anterior cingulate cortex are not shown. As a comparison, we also show the activation focus (blue circle, 16) for subitizing, the capacity-limited process of object enumeration thought to be 'pre-attentive' [90].

maintaining and monitoring that information (VSTM and MOT), and in selecting an appropriate response for it (PRP), as if these tapped into strictly independent stages of information processing. Indeed, perceptual and response limitations have traditionally been viewed as such [55]. However, this view has been challenged by findings that the AB and PRP partly share a common capacity-limited stage of information processing [8,14,77,78]. In hybrid AB–PRP experimental designs, the AB is increased when response to Task 1 is speeded [13,14], and a PRP to Task 2 can be elicited simply by making a perceptual decision to Task 1 [14,77]. What might be the bottleneck of information processing commonly tapped by both the AB and PRP paradigms? One possibility is that both target consolidation and response selection compete for short-term memory processing [62,77]: explicit target detection involves short-term memory consolidation [3,8], and response selection may require retrieval of stimulus–response associations from short-term memory [62]. Alternatively, target consolidation and response selection might competitively interact. Although the latency of the P300 to the Task 2 target is not affected by Task1–Task2 SOA in a PRP paradigm, its amplitude is diminished [67,79], raising the possibility that Task 1 response selection suppresses stimulus consolidation in working memory for Task 2.

Although it is likely that the AB and PRP tap into a common capacity-limited stage of information processing, it remains to be seen whether that stage corresponds to the central, amodal bottleneck believed to be at the heart of the PRP. For one, it is unclear whether the second stage of the two-stage AB model corresponds to the central bottleneck of information processing revealed by the PRP [10]. Second, crossmodal conditions that yield large PRP costs do not necessarily produce AB deficits [10–12], raising the possibility that the AB and PRP deficits are dissociable. Furthermore, the AB and the PRP are associated with largely distinct electrophysiological measures. The AB affects the P300 [9,79] whereas the PRP affects the lateralized readiness potential (LRP), an ERP signature of motor preparation, but leaves P300 onset intact [67,79]. These and other findings have led to the conclusion that there are at least two major sources of dual-task costs: a processing resource that is common to both the AB and PRP, and a modality-specific capacity-limited process unique to the AB [10,14,78].

Finally, although the AB and PRP show processing convergence, there is little evidence for such convergence

between the AB and VSTM maintenance, as the AB is largely unaffected by VSTM load [80]. These results are consistent with the view that short-term memory consolidation – the capacity limited process considered to be at the root of the AB [3,62,77] – is dissociable from short-term memory storage [81].

The functional neuroanatomy of processing bottlenecks: a synthesis

Another approach to assessing the relationship between processing bottlenecks is to determine whether they engage similar neural substrates. Although demonstrating that two cognitive processes activate similar brain regions does not necessarily imply that these processes overlap at the cellular level, the failure to find such common foci of activation would rule out the possibility that these processes affect each other through a common neural substrate. Figure 4 shows the brain regions implicated in the various capacity-limited processes examined in this review. There is a striking convergence of activation for AB and PRP studies in the lateral frontal/prefontal cortex (Figure 4, black circles). This brain region might therefore represent a neural locus of the common processing bottleneck revealed by hybrid AB–PRP studies. Consistent with it exerting a role in both target consolidation and response selection, the lateral frontal cortex has been proposed to use a flexible coding system that allows it to process that information which is relevant for current behavior [82].

In contrast to the lateral frontal cortex, other brain regions may be preferentially involved in specific processing bottlenecks. In particular, the lateral parietal cortex is generally more responsive to AB tasks than to PRP paradigms (Figure 4). This brain region, perhaps along with the visual cortex [17], may therefore be crucial for the modality-specific component of the AB bottleneck [26]. Conversely, the clustering of PRP-related activations in dorsal pre-motor cortex (Figure 4, white circles) may correspond to a response selection-specific substrate of the PRP. Finally, the finding that VSTM and MOT load studies recruit partially overlapping regions of the posterior parietal cortex is consistent with a role for attention in VSTM, although the data is currently not as conclusive regarding the relationship between the functional neuroanatomy of VSTM load and AB studies.

Conclusion

This review of the literature supports the view that the neural substrates of VSTM storage capacity are primarily localized to posterior cortical regions, and predicts that distinct regions of visual cortex may contribute to separate forms of VSTM storage capacity. By contrast, the AB and PRP bottlenecks are most likely to result from the interaction between foci of a distributed fronto-parietal – and probably visual – cortical network, with individual nodes of the network (e.g. lateral frontal cortex) potentially contributing to more than one processing bottleneck. Indeed, the temporal characteristic of the AB and PRP deficits might originate not from the time required for processing information in one particular neural locus, but rather from the temporal constraints imposed by the reciprocal exchange of information across the neural nodes of the network.

Although this review has raised a series of testable hypotheses regarding the functional neuroanatomy of bottlenecks of human information processing, it speaks little to the underlying causes of these bottlenecks. That is, what physiological properties of our functional neuroarchitecture are responsible for our all-too-humbling limitations in what we can perceive, what we can hold in mind, and in what we can do? It is our hope that isolating the brain substrates associated with these processing bottlenecks will pave the way for future research aimed at elucidating their neurophysiological origins and, ultimately, at informing cognitive models of human information processing. Surely, it will take the best of our minds to understand the worst of our brains.

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