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Review Article

Visual attention: Linking prefrontal sources to neuronal and behavioral correlates

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ABSTRACT

Attention is a means of flexibly selecting and enhancing a subset of sensory input based on the current behavioral goals. Numerous signatures of attention have been identified throughout the brain, and now experimenters are seeking to determine which of these signatures are causally related to the behavioral benefits of attention, and the source of these modulations within the brain. Here, we review the neural signatures of attention throughout the brain, their theoretical benefits for visual processing, and their experimental correlations with behavioral performance. We discuss the importance of measuring cue benefits as a way to distinguish between impairments on an attention task, which may instead be visual or motor impairments, and true attentional deficits. We examine evidence for various areas proposed as sources of attentional modulation within the brain, with a focus on the prefrontal cortex. Lastly, we look at studies that aim to link sources of attention to its neuronal signatures elsewhere in the brain.

Contents

Abbreviations: AC, anterior commissure; ACd, dorsal anterior cingulate area; CC, corpus callosum; D1R, D1-type dopamine receptors; D2R, D2-type dopamine receptors; dlPFC, dorsolateral prefrontal cortex; FEF, frontal eye field; Fr2, frontal cortical area; IT, inferotemporal cortex; LGN, lateral geniculate nucleus; LIP, lateral intraparietal area; LIP/d/v, lateral intraparietal area dorsal/ventral; MEF, medial eye field; MST, medial superior temporal area; MT, middle temporal visual area; PFC, prefrontal cortex; PMC, premotor cortex; RF, response field; SC, superior colliculus; TRN, thalamic reticular nucleus; VIP, ventral intraparietal area.

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Attention is the means by which we focus on behaviorally relevant information, to select and enhance a subset of sensory information for further processing while ignoring the rest. Visual attention alters the processing of visual information (Carrasco et al., 2004; Carrasco, 2011; Yeshurun and Carrasco, 1998), prioritizing a location (spatial attention) or a particular feature (feature-based attention) based either on internally represented goals (top-down attention) or the physical salience of the stimulus (bottom-up attention). The focus of this review is on goal-driven, top-down modulations of visual processing: the changes in visual responses thought to underlie the behavioral benefits of attention, and the network of areas thought to drive these changes based on attentional cues. Specifically, our focus will be on the role of prefrontal cortex (PFC) in the top-down control of attention, as this area is critical for coordinating our goal-driven behavior. We will first address the attention-driven changes in visual processing and how they could benefit behavior (Section 1), then turn to some methodological concerns regarding measuring attention at a behavioral level (Section 2), review the evidence for several areas as sources of attentional modulation (Section 3), and finally examine experiments seeking to link attention in these source areas to the signatures previously discussed (Section 4).

1. Attention-driven changes in visual processing

1.1. Neuronal signatures of attention

Attention has long been known to modulate visual cortical responses (Moran and Desimone, 1985); since this initial report, attentional modulation has been reported in a wide range of visual cortical areas (Buffalo et al., 2010; Herrero et al., 2008; Sharma et al., 2014; Spitzer et al., 1988; McAdams and Reid, 2005; McAdams and Maunsell, 1999; Boudreau et al., 2006; Gregoriou et al., 2009b; Treue and Maunsell, 1996; Treue and Martínez Trujillo, 1999; Sheinberg and Logothetis, 2001), and as early in the visual hierarchy as the lateral geniculate nucleus (McAlonan et al., 2008). Attentional modulation has also been reported in a number of oculomotor structures with visual responses, including the lateral intraparietal area (LIP), superior colliculus (SC), frontal eye field (FEF), and dorsolateral PFC (dlPFC) (Goldberg and Wurtz, 1972; Bisley and Goldberg, 2003; Thompson et al., 2005; Buschman and Miller, 2007). Since attention is the means by which we select and enhance a subset of sensory information for further processing, a natural assumption is that this improvement in perception should be the result of an increase in the strength of sensory signals within visual areas. The first and most frequently reported effect of attention is an increase in visual responses when attention is directed toward the response field (RF) of a neuron (Moran and Desimone, 1985); however, a variety of other measures are increasingly included in attention studies (reviewed in Noudoost et al., 2010). In addition to increased visual responses, other reported signatures of attention include shrinkage of RFs containing the attended location (Womelsdorf et al., 2008; Anton-Erxleben et al., 2009), shifts of visual RFs toward the attended location (Womelsdorf et al., 2006a, 2008; Connor et al., 1997), decreases in trial to trial variability of individual neuron's responses (Mitchell et al., 2007; Cohen and Maunsell, 2009), enhanced contrast sensitivity (Reynolds et al., 2000), increased synaptic efficacy (Briggs et al., 2013), changes in noise correlations between neurons (Mitchell et al., 2009; Ruff and Cohen, 2014; Cohen and Maunsell, 2009), decreases in low-frequency LFP power and coherence (Fries et al., 2001, 2008; Mitchell et al., 2009), increases in gamma-band LFP power (Fries et al., 2008; Gregoriou et al., 2009a; Taylor et al., 2005), increases in gamma-band coherence both within and between areas (Fries et al., 2001, 2008; Gregoriou et al., 2009b; Womelsdorf et al., 2006b; Saalmann et al., 2007), decreases in response latency (Sundberg et al., 2012; Galashan et al., 2013), decreases in bursting activity (Anderson et al., 2013), and reductions in action potential height (Anderson et al., 2013). The question then becomes which of these changes in neuronal responses are functionally relevant for producing the behavioral benefits of attention.

1.2. Potential benefits of known signatures of attention

An enhanced neuronal representation of the target stimulus could underlie the behavioral benefits of attention, including greater sensitivity (Sridharan et al., 2014), greater spatial resolution (Carrasco, 2011), and faster response times (Posner, 1980). Here we summarize the potential representational benefits of the reported signatures of attention.

1.2.1. Attention enhances the visual representation at the level of single neurons

First, we consider the effects of attention on the responses of individual neurons: attention increases response magnitude, reduces neuronal latency, alters RFs, reduces burstiness, and reduces the variability of visual responses. From a signal processing perspective an increase in the difference in a neuron's response to its preferred vs. non-preferred stimuli can result in more reliable stimulus discrimination (Green and Swets, 1966). Computational models confirm that multiplicative-gain type increases in neuronal firing rates can produce a benefit when decoding population activity (Cohen and Maunsell, 2009; Mitchell et al., 2009; Ling et al., 2009). Attention also reduces the latency of neuronal responses (Sundberg et al., 2012; Galashan et al., 2013), which could potentially provide more information in a shorter amount of time, thus facilitating a faster reaction. More intense or higher contrast stimuli evoke shorter latency responses across numerous visual areas (Bell et al., 2006; Raiguel et al., 1999; Albrecht, 1995; Oram et al., 2002); thus attention, by reducing response latencies, effectively makes a stimulus resemble a higher contrast version of itself, similar to its effect on the contrast response function measured in firing rate (Reynolds et al., 2000). The response latencies of neurons in visual cortex are also directly correlated with reaction times on saccade tasks (Lee et al., 2010). Moreover, attention alters neuronal RFs, which could allocate more neuronal resources to the locus of attention. Attention causes a dynamic change in the RFs of neurons in several visual areas, shifting them toward the locus of attention and shrinking RFs at the attended location (Anton-Erxleben et al., 2009; Connor et al., 1996, 1997; Kusunoki and Goldberg, 2003; Womelsdorf et al.,

2006a). These shifts could underlie some of the misperceptions that occur away from the locus of attention (Suzuki and Cavanagh, 1997), while contributing to an enhanced spatial resolution at the location of the attentional target (Anton-Erxleben and Carrasco, 2013). Recently, attention has been shown to reduce the burstiness of neuronal responses in visual area V4 (Anderson et al., 2013). Although previous work in other brain areas had suggested that bursts are better tuned and propagate more reliably than non-burst spikes (Krahe and Gabbiani, 2004; Sherman, 2001; Cattaneo et al., 1981; Samonds and Bonds, 2004; Shih et al., 2011; Reich et al., 2000), if these attentional changes are indeed beneficial to visual processing then in V4 bursts might instead introduce variability into the neuronal response. Attention increases the reliability of neuronal responses, as quantified by the ratio of the mean and variance of the firing rate across trials (Mitchell et al., 2007); such a reduction in trial-to-trial variability is by definition an improvement in signal to noise ratio.

The sum of these attention-driven changes in individual neuronal responses has been shown to improve the population coding of objects in inferotemporal (IT) cortex (Zhang et al., 2011). Using a classifier on the responses of ~200 IT neurons to stimuli presented in isolation or in an array, followed by an attentional cue toward one of the objects, Zhang and colleagues demonstrated that attention-driven changes in IT responses improved the ability of the classifier to identify an object in the presence of other stimuli, causing responses to more strongly resemble those to the attended stimulus presented in isolation. As implemented, this classifier approach does not specify which of the changes in the neural response were critical for the improvement in performance (although in this case at least it must be properties which can be measured in the pseudopopulation of individually recorded neurons; a true simultaneous population recording could potentially provide additional improvements using precise trial to trial correlations or synchronization).

1.2.2. Attention alters inter-neuronal correlations

Another signature of attention whose effects on population encoding have been modeled is a change in the correlated noise of simultaneously recorded pairs of neurons (Cohen and Kohn, 2011). Low-frequency fluctuations in firing rate, correlated across the neuronal population, are often characterized as representing noise due to common inputs; attention reduces the strength of this correlation in V4, and this decorrelation has a greater impact on the signal to noise ratio of the pooled activity than the attentional modulation of firing rates does (Mitchell et al., 2009). A separate study reported that over 80% of the attention-driven improvement in V4 population sensitivity was attributable to reduced trial-to-trial correlations (noise correlations) between neurons (with the remainder reflecting individual neuron's changes in firing rate and reliability) (Cohen and Maunsell, 2009). Correlations between neurons are not fixed based on connectivity, but have been shown to depend on their relationship to task parameters: for example, two direction-selective neurons in the middle temporal area (MT) will show a greater noise correlation in conditions when their preferred directions of motion correspond to the same behavioral response (Cohen and Newsome, 2008). The effect of attention on these correlations is likewise not fixed: attention will decorrelate some pairs of neurons and increase the correlation between others, depending on whether the neurons in question are driving the same or different behavioral responses (Ruff and Cohen, 2014). In the cases of certain potential changes in visual representations, the modeling suggests rather complicated relationships between individual neuronal responses and the quality of the population visual representation; for example, the information content of the neural population can be improved either by sharpening or broadening the feature tuning of neurons, depending on factors such as noise correlations between neurons and the ability of downstream areas to utilize pairwise correlations (Zhang and Sejnowski, 1999; Seriès et al., 2004). Such complexities highlight the need for a more than theoretical measure of the impact of these representational changes on behavior.

1.2.3. Attention synchronizes activity within and between areas

Oscillations may be a means for the brain to synchronize spikes so that their input arrives simultaneously at downstream neurons; such synchronized spikes will have a much greater impact on a downstream neuron than the same number of spikes arriving at different times (Salinas and Sejnowski, 2000, 2001; Fries, 2005). Changes in local synchronization could be reflected both in the overall power in a particular frequency band of the LFP, and in the synchronization (or phase-locking) of neuronal spikes with a particular LFP oscillation frequency. Neurophysiological recordings during working memory demonstrate that information can be encoded in the synchronization of spiking activity with local oscillations: the theta-band synchronization of V4 activity during the delay conveys greater information about the contents of memory than the overall firing rate of these neurons (Lee et al., 2005); similarly, in PFC, spikes occurring at a specific phase relative to a 32 Hz LFP oscillation contain more information about the stimulus held in memory (Siegel et al., 2009). Just as synchrony within an area can theoretically enhance the impact of that area on downstream neurons, synchrony between areas could enhance the influence of one area on another (Salinas and Sejnowski, 2000, 2001; Fries, 2005; Knoblich et al., 2010). For example, the timing of sensory input relative to the gamma cycle has been shown to alter the magnitude of evoked responses in mouse barrel cortex (Cardin et al., 2009). Thus attention-driven changes in synchrony could strengthen the effect of visual activity on other areas in the visual hierarchy, and its ability to drive behavior. The most reliable reported effect of attention on local synchrony has been an increase in gamma-band LFP power and spike-field synchrony in area V4 (Fries et al., 2001, 2008; Gregoriou et al., 2009a; Taylor et al., 2005; Womelsdorf et al., 2006b). However, the effects of attention on gamma power and synchrony in other visual areas are more varied, with some studies reporting that attention decreases V1 gamma power and coherence (Chalk et al., 2010), while others report an increase in gamma coherence within V1 (Buffalo et al., 2011) or between V1 and V4 (Bosman et al., 2012). There have been reports of variations in LFP power spectrum and coherence in different layers of visual cortex (Buffalo et al., 2011), with gamma coherence in superficial layers and lower-frequency coherence in the deeper layers. This finding led to the suggestion that theta and gamma bands may represent feedforward input, while beta frequencies reflect feedback; this hypothesis is supported by the effects of microstimulation in V1 or V4 on LFP activity in the other area (Van Kerkoerle et al., 2014), and by a comparison of measures of frequency-specific influence between areas with anatomical hierarchies (Bastos et al., 2015). In this hypothesis the attentional enhancement of gamma power and coherence would be a secondary consequence of top-down beta-band effects (Lee et al., 2013; Bressler and Richter, 2014), but could still be causally related to the behavioral effects of attention.

1.3. Relationships between neuronal signatures and behavior

In the previous section we reviewed the currently known signatures of attention, and discussed the potential ability of these changes to enhance the representation of attended stimuli. Of course, the theoretical benefit of a particular change in the visual representation for an ideal observer, or for a particular decoding model (Grewe et al., 2007), even if biologically plausible, does not guarantee that such a benefit actually exists. One practical

approach to examine whether these signatures are actually related to perceptual performance, or merely epiphenomenal, is to see whether they covary with some measure of behavioral performance.

Table 1 presents a number of neuronal signatures of attention that are correlated with some aspect of behavioral performance. Attentional modulation of firing rates in visual cortex is greater on correct trials than error trials in the same neurons (Gregoriou et al., 2014); across recording sessions the magnitude of attentional modulation in a given neuron was correlated with overall behavioral performance in that session (McAdams and Maunsell, 1999). In V1, larger attentional modulation of firing rates corresponds with faster reaction times (Sharma et al., 2014), and in V4 the magnitude of firing rate modulation increases with increasing task difficulty (Spitzer et al., 1988; Boudreau et al., 2006). Correlations between firing rate changes and behavioral measures have also been reported in parietal and prefrontal cortex, which are candidates for driving the attentional modulations seen in visual cortex. In LIP, attentional modulation is larger in correct trials (Bisley and Goldberg, 2003), and attentional modulation of FEF activity is present in correct, but not missed, change trials in a change detection task (Armstrong et al., 2009). Attentional modulations of firing rate in FEF and lateral PFC correlate with reaction time in a visual search task (Buschman and Miller, 2007). Attention also shortens the latency of visual responses in area MT, and this decrease in latency is associated with faster overall reaction times (Galashan et al., 2013). Attention-induced changes in synchrony also predict performance in attention tasks. Gamma-band LFP power and spike-field synchronization at the attended location in V4 are both higher in correct trials (Gregoriou et al., 2014), with gamma-band spike-field synchrony predicting reaction times (Womelsdorf et al., 2006b). Consistent with these findings, gamma-band LFP power in response to distractor stimuli is elevated on trials in which the animal incorrectly responds to the distractor (Taylor et al., 2005). Gamma-band power and synchronization increase at the attended location; in contrast, V4 alpha-band LFP activity decreases at the attended location, and the magnitude of this drop in alpha power was larger on correct trials (Gregoriou et al., 2014).

However, not every observed signature of attentional deployment is correlated with correct performance or reaction time. Because the source of errors or determinates of reaction time in a specific task are not always clear, such a lack of correlation between neural signatures and behavior is most compelling when

Table 1
Studies demonstrating relationships between neuronal responses and behavior in attention tasks. Studies are grouped according to the attentional signature being studied (*Signature*), then by the area being recorded from (*Area*). The effect of attending to a neuron's RF on the signature is listed (*Effect of attention*), and the relationship between this modulation and the animal's behavior is noted (*Behavioral correlate*). Each row summarizes an individual study (*Reference*). Studies which report data for more than one area may be listed multiple times. RT, reaction time; Ach, acetylcholine.

Signature		Area	Task	Effect of attention	Behavioral correlate	Reference
Firing rate		V1	Disappearance detection	Increase	Inverse correlation between attentional modulation and RT	Sharma et al. (2014)
		V1	Luminance-change detection	Increase	Ach application decreases RT and increases attentional modulation	Herrero et al. (2008)
		V4	Delayed match-to-sample	Increase	Positive correlation between attentional modulation and animal performance (across sessions)	McAdams and Maunsell (1999)
		V4	Orientation discrimination	Increase	Larger attentional modulation in correct trials vs. error trials	Gregoriou et al. (2014)
		V4	Orientation-change detection	Increase	Larger attentional modulation as task difficulty increases	Boudreau et al. (2006)
		LIP	GO/NOGO cue discrimination	Increase	Larger attentional modulation in correct trials vs. error trials	Bisley and Goldberg (2003)
		FEF	Orientation-change detection	Increase	Larger attentional modulation in hit trials vs. miss trials	Armstrong et al. (2009)
		FEF	Oddball paradigm	Increase	Harder search task increased RT and the time of target selection by visually responsive FEF neurons	Sato et al. (2001)
		FEF	Free gaze visual search	Increase	Inverse correlation between attentional modulation and number of saccades	Zhou and Desimone (2011)
		FEF	Visual search	Increase	Inverse correlation between FEF response and RT	Buschman and Miller (2007)
		LPFC	Visual search	Increase	Inverse correlation between dlPFC response and RT	Buschman and Miller (2007)
Gamma-band synchronization		V4	Orientation discrimination	Increase	Higher Gamma-band synchrony in correct trials vs. error trials	Gregoriou et al. (2014)
		V4	Color-change detection	Increase	Higher Gamma-band synchrony in faster trials	Womelsdorf et al. (2006b)
LFP power	Gamma-band	V4	Orientation discrimination	Increase	Higher Gamma-band LFP power in correct trials vs. error trials	Gregoriou et al. (2014)
		V4	Shape-tracking	Increase	Higher Gamma-band LFP power in distracter related false alarms	Taylor et al. (2005)
	Alpha-band	V4	Orientation discrimination	Decrease	Larger attentional modulation in correct trials vs. error trials	Gregoriou et al. (2014)
Latency		MT	Speed-change detection	Decrease	Decrease of RT in trials with shorter neuronal latency	Galashan et al. (2013)

it is reported alongside demonstrated correlations for other neuronal signatures in the same dataset. For example, in a cued change detection task, Gregoriou and colleagues reported that in V4, attention-induced increases in gamma-band LFP power and gamma-band spike-field synchronization, decreases in alpha-band LFP power, and increases in firing rate were all larger in correct trials; in the same recordings, attention-driven drops in beta-band LFP power, beta-band spike-field synchronization, and noise correlations between pairs of neurons were no different on correct and incorrect trials (Gregoriou et al., 2014). This last finding—that in contrast to changes in firing rate, the magnitude of the change in noise correlations is unrelated to performance—is particularly surprising given the results of models which suggest that attentional changes in noise correlations theoretically improve the visual signal more than the changes in firing rate do (Cohen and Maunsell, 2009; Mitchell et al., 2009). For many other areas or signatures, an examination of their correlation with behavioral performance has not been performed, or not reported; provisionally, however, those signatures which have demonstrated a relationship with performance—enhanced visual responses, faster visual latencies, increased gamma-band LFP power and spike-field synchronization, and decreased alpha-band LFP power—may be considered the most promising candidates for contributing to the performance benefits of attentional cueing.

2. Behavioral measures of attention

2.1. Visual and response confounds of impaired performance on attention tasks

A deficit on an attentional task is not necessarily an attentional deficit. Experimenters have long aimed to design tasks which allow the measurement of attention independent of visual effects on the one hand (Squire et al., 2013), or motor deficits and response biases on the other (Sridharan et al., 2014). While complete blindness at a particular retinotopic location—for example from localized damage to the retina itself—is easily controlled for, more subtle yet still fundamentally visual deficits are possible. One important feature of many models of cortical visual processing is that there are competitive interactions between stimulus representations (Reynolds et al., 1999, 2000; Reynolds and Heeger, 2009; Desimone and Duncan, 1995). Since visual representations are spread across many interconnected cortical areas (Felleman and Van Essen, 1991), weakening the visual representation in any one of these areas could put the representation of that stimulus at a competitive disadvantage when other stimuli also appear. See Squire et al. (2013) for a simulation showing how the effects of visual lesions could depend on the presence of distracting stimuli, using the model developed by Reynolds and Heeger (2009). Thus distractor-dependence alone does not guarantee that a behavioral impairment is attentional.

2.2. Studies reporting behavioral deficits

Neglect is an attentional deficit that describes the inability to perceive, process and/or respond to stimuli within a particular sensory domain, for example a region of visual space such as in spatial hemi-neglect (Mesulam, 1981; Heilman et al., 2000; Corbetta and Shulman, 2011; Milner and McIntosh, 2005). By definition, neglect occurs in the absence of a purely sensory deficit. The classic criteria that rules out a loss of sensation is the phenomenon of extinction (De Haan et al., 2012; Brozzoli et al., 2006; Riddoch et al., 2009). Extinction is the inability to detect or recognize two or more simultaneously presented stimuli, despite intact abilities to detect the same stimuli presented in isolation. For example, the observation that a subject has impaired detection of stimuli within an affected part of space (the scotoma) *only* when competing stimuli are simultaneously presented outside of the scotoma is historically interpreted as indicating that visual processing is intact but attention is disrupted. A large number of studies using a loss-of-function approach in non-human primates have relied on neglect and extinction to rule out simple visual (or motor) deficits in an attempt to identify specific brain regions controlling visual attention (see Table 2). Such studies have employed a variety of behavioral tasks to study vision and attention including neurological testing, detection, search, and sensory discrimination tasks, all of which can be designed to accommodate both endogenous and exogenous attention cues if so desired. Unilateral lesions, either permanent or reversible (e.g., pharmacological inactivation), of many different cortical and subcortical structures have been shown to produce behavioral deficits in at least one of these behavioral tasks, but the interpretation of these deficits has varied across different areas, as well as within a single area across different studies (see Table 2).

Importantly, since neglect and extinction (although considered to be associated with attention function) are not themselves brain processes, but rather behavioral phenotypes, it is worth emphasizing that these phenotypes may be indistinguishable from blindness or other purely visual deficits in many behavioral tasks, such as a detection task. Nonetheless, these terms are often used in the literature to explain the nature of a deficit, without explicitly interpreting the underlying dysfunctional brain process (e.g., vision, attention, etc.). Usually when the behavioral phenotype of neglect and extinction is discussed an attention deficit is implied and sometimes explicitly stated (Lovejoy and Krauzlis, 2010). But this is not always the case; extinction-like deficits following lesions have also been explicitly interpreted as visual deficits and not attention deficits, highlighting the ambiguity of what underlying dysfunction is responsible for producing extinction (Moore et al., 1995; Cowey and Stoerig, 2004). As discussed above, a behavioral deficit on an attention-demanding task does not necessarily imply an attention deficit. In this vein, many lesion and inactivation studies reporting deficits on tasks that likely require attention, such as a detection task, have not interpreted the observed deficits as related to attention, but instead as deficits in vision or oculomotor function (Latto and Cowey, 1971; Sommer and Tehovnik, 1997; Li et al., 1999; Cowey and Stoerig, 1995; Moore et al., 1995; Mohler and Wurtz, 1977; see Table 2). In contrast, many other studies using lesion or inactivation have interpreted the observed deficits as not purely sensory or motor, but as deficits in attention or target selection. Some of these studies explicitly sought to rule out a visual deficit (e.g., Crowne et al., 1981; Schiller and Chou, 1998; Wardak et al., 2004, 2006; Schiller and Lee, 1991; De Weerd et al., 1999; Desimone et al., 1990; Lovejoy and Krauzlis, 2010; Monosov et al., 2011; Song et al., 2011), while others did not (Welch and Stuteville, 1958; Wurtz and Goldberg, 1972; Petersen et al., 1987; Robinson and Kertzman, 1995; Crowne and Mah, 1998; Balan and Gottlieb, 2009; Liu et al., 2010). Of the studies that did explicitly control for the possibility of purely visual deficits, the most common method was to demonstrate that the behavioral deficits were distractor dependent (see Table 2, Column 6).

As discussed above, a weakening in the sensory representation of a target stimulus may only be evident in the presence of competing stimuli. Visual processing of a target when it is presented alone is not the same as when it is among distractors. Thus, a distractor-dependent deficit may not always be indicative of an attentional deficit. This caveat has been raised by previous authors (Desimone et al., 1990; Desimone and Duncan, 1995; Buffalo et al., 2005). In their landmark review describing the "biased-competition" model of attention, Desimone and Duncan noted the ambiguity of distractor-dependent deficits:

Table 2
Unilateral loss of function studies relevant to visual attention. An overview of permanent and reversible lesions in the non-human primate and the interpretations of the observed behavioral deficits. Reports are grouped by brain area, and within each brain area reports are listed chronologically. Each row summarizes an individual study (listed in *Reference*). Studies which report data for more than one area may be listed multiple times. *Structure*, the lesioned brain area. *Loss of function method*, for subjects with permanent lesions that subsequently recovered to normal function, the word "Lesion" is listed and the latest post-lesion time point that a deficit was observed is listed in parentheses. If the effects of the lesion did not recover (DNR), the latest-observed post-lesion time point is listed. For studies using pharmacological inactivation, the drug is listed with the administered dose in parentheses. *Task*, the paradigm(s) for which behavioral data is reported. *Behavioral response*, the action performed by a subject to correctly complete a trial. *Interpretation of deficit*, the function that the author(s) attribute to the lesioned structure to explain the observed changes in behavior. *Evidence consistent with attention deficit*, the control data or argument provided by the author(s) to support the interpretation of an attention deficit and not a purely visual or motor deficit (also, see text). If no control data or argument was provided to rule out a visual or motor deficit, "- - -" is listed. NA (not applicable) indicates that the author(s) interpreted the deficit as purely visual or motor. Throughout the table a "?" indicates that this information was not reported. *Structure abbreviations*: AC, anterior commissure; CC, corpus callosum; FEF, frontal eye field; LGN, lateral geniculate nucleus; LIP/d/v, lateral intraparietal area dorsal/ventral; MEF, medial eye field; MT, middle temporal visual area; dl/PFC, dorsal lateral prefrontal cortex; SC, superior colliculus; VIP ventral intraparietal area.

Structure	Loss of function method	Task	Behavioral response	Interpretation of deficit	Evidence consistent with attention deficit	Reference
Frontal cortex						
FEF (Area 8)	Lesion (5 months)	Free-moving foraging task	Reach	Visual	NA	Kennard (1939)
Peri arcuate sulcus[a]	Lesion (2 weeks)	Neurological testing	Orientation	Neglect	- - -	Welch and Stuteville (1958)
FEF	Lesion (DNR 2 weeks)	Detection	Lever press	Visual	NA	Latto and Cowey (1971)
FEF	Lesion (DNR 3–3.5 months)	Detection	Body withdraw	Attention	Distracter dependent	Crowne et al. (1981)
FEF	Lesion (DNR 1 year)	Search	Reach	Attention	Search dependent	Collin et al. (1982)
Pre-arcuate or post-arcuate[b]	Lesion (DNR 1–3 months)	Detection	Orientation	Neglect	Distracter dependent, distance dependent	Rizzolatti et al. (1983)
FEF and post-arcuate[c] and with SC	Cooling	Detection	Saccades	Neglect/ oculomotor	- - -/NA	Keating and Gooley (1988)
FEF and bilateral posterior cortex[d]	Lesion (1 week)	Neurological testing	Orientation	Neglect	Distracter dependent	Lynch and McLaren (1989)
FEF	Muscimol (1 μL of 5 μg/μL)	Saccade tasks	Saccade	Oculomotor	NA	Dias et al. (1995)
FEF	Lidocaine (18 μL of 2%) or Muscimol (2 μL of 2 μg/μL)	Saccade tasks (detection)	Saccade	Oculomotor	NA	Sommer and Tehovnik (1997)
Peri arcuate (Area 8)	Lesion (3 weeks)	Detection	Reach	Neglect	- - -	Crowne and Mah (1998)
FEF	Muscimol (1 μL of 5 μg/μL)	Saccade tasks	Saccade	Oculomotor	NA	Dias and Segraves (1999)
FEF or MEF	Lesion (DNR 5 months)	Free choice saccade task	Saccade	Target selection/ extinction	Distracter dependent	Schiller and Chou (1998)
FEF or PFC	Lesion (2–8 weeks)	Search (oddities task)	Saccade	Attention/ target selection	Distracter dependent	Schiller and Chou (2000)
dlPFC	Muscimol (1 μL of 5 μg/μL)	Search	Saccade	Attention	Distracter dependent	Iba and Sawaguchi (2003)
FEF	Muscimol (0.5–1.5 μL of 0.5 μg/μL)	Free choice saccade task, search (oddities task)	Saccade	Target selection	Distracter dependent	Schiller and Tehovnik (2003)
FEF	Muscimol (3 separate 0.5 μL injections of 3–8 μg/μL)	Detection	Lever release	Attention	Distracter dependent	Wardak et al. (2006)
PFC and CC and AC	Lesion (DNR 157 weeks)	Search and sensory discrimination	Lever release	Attention – switching	Attention – switching – frequency dependent	Rossi et al. (2007)
FEF	Muscimol (3–6 μL of 5μg/μL)	Search and sensory discrimination	Lever turn	Attention	Cue-type dependent	Monosov and Thompson (2009)
FEF	Muscimol (3–6 μL of 5μg/μL)	Search and sensory discrimination	Lever turn	Resolving competition between stimuli	Distracter dependent	Monosov et al. (2011)
PFC and CC and AC	Lesion (DNR 5 years)	Search and sensory discrimination	Lever release	Attention	- - -	Gregoriou et al. (2014)
Parietal cortex						
Parietal lobe[e]	Lesion (2 weeks–1 month)	Neurological testing	Orientation	Neglect	Distracter dependent	Heilman et al. (1970)
Posterior parietal[f]	Lesion (DNR 7 days)	Detection	Reach	Neglect	Distracter dependent	Deuel and Regan (1985)
Area 7	Lesion (?)	Cued detection	Bar press	Attention	Cue validity effects	Petersen and Robinson (1986)
Posterior cortex[d]	Lesion (DNR 21 days)	Detection	Saccade	Attention	Distracter dependent	Lynch and McLaren (1989)
Area 7a and LIP and VIP	Lesion (DNR 8 weeks)	Detection	Saccade	Neglect	- - -	Crowne and Mah (1998)

Table 2 (*Continued*)

Structure	Loss of function method	Task	Behavioral response	Interpretation of deficit	Evidence consistent with attention deficit	Reference
LIP	Muscimol (1–3 μL of 8 μg/uL)	Saccade tasks	Saccade	Oculomotor	NA	Li et al. (1999)
LIP	Muscimol (4–6 separate 0.5–1 μL injections of 2–6 μg/μL)	Saccade tasks, search	Saccade	Attention/ target selection	Distracter dependent	Wardak et al. (2002)
LIP	Muscimol (0.5–1.5 μL of 0.5 μg/μL)	Free choice saccade task, search (oddities task)	Saccade	Target selection	Distracter dependent	Schiller and Tehovnik (2003)
LIP	Muscimol (6 separate 0.5 μL injections of 8–12 μg/μL)	Detection	Lever release	Attention	Distracter dependent	Wardak et al. (2004)
LIP	Muscimol (6.09 ± 1.53 μL of 8.2 ± .55 μg/μL)	Search and discrimination	Bar release	Attention	- - -	Balan and Gottlieb (2009)
LIPd and/or LIPv	Muscimol (1–4 μL of 8 μg/μL)	Search	Saccades	Oculomotor and/or attention	- - -[g]	Liu et al. (2010)
Occipital and temporal cortex						
V1	Lesion (4 weeks)	Detection	Bar release or saccade	Visual	NA	Mohler and Wurtz (1977)
V1 and SC	Lesion (DNR 15 weeks)	Detection	Bar release or saccade	Visual	NA	Mohler and Wurtz (1977)
V1	Lesion (3 days) or Muscimol (1 μL of 1 μg/μL)	Detection	Saccade	Visual	NA	Newsome et al. (1985)
V4	Lesion (DNR several months)	Search (oddities task)	Saccade	Target selection	Distracter dependent	Schiller and Lee (1991)
V4 and/or MT	Lesion (DNR 28 months)	Search (oddities task)	Saccade	Attention/ target selection	Distracter dependent	Schiller (1993)
V1	Lesion (DNR several years)	Detection	Reach	Visual	NA	Cowey and Stoerig (1995)
V1	Lesion (DNR 24 months)	Detection	Saccade	Visual	NA	Moore et al. (1995)
V4	Lesion (DNR 3 years)	Sensory discrimination	Lever release	Visual	NA	De Weerd et al. (1996)
V1	Lesion (DNR ? years)	Detection	Reach	Visual	NA	Cowey and Stoerig (1997)
V4 and/or TEO	Lesion (DNR several years)	Sensory discrimination	Bar release	Top down attention	Distracter dependent	De Weerd et al. (1999)
V4 and/or TEO	Lesion (DNR 90 months)	Sensory discrimination	Lever release	Attention	Distracter dependent	De Weerd et al. (2003)
V1	Muscimol (0.5–1.5 μL of 0.5 μg/μL)	Free choice saccade task, search (oddities task)	Saccade	Target selection	Distracter dependent	Schiller and Tehovnik (2003)
V1	Lesion (DNR 10 years)	Detection	Reach	Visual	NA	Cowey and Stoerig (2004)
V4 and/or TEO	Lesion (DNR ? years)	Sensory discrimination	Lever release	Attentional resolution	Distracter dependent	Buffalo et al. (2005)
Sub-cortical						
SC	Lesion (1–7 weeks)	Detection	Saccade	Attention	- - -	Wurtz and Goldberg (1972)
SC	Lesion (DNR 3.5 weeks)	Detection	Bar release or saccade	Oculomotor	NA	Mohler and Wurtz (1977)
SC	Lesion (DNR 1 year)	Search	Reach	Attention	Search dependent	Collin et al. (1982)
SC	Lesion (DNR 3.5 weeks)	Detection	Lever press or saccade	Oculomotor	NA	Albano et al. (1982)
SC	Muscimol (0.4–2 μL of 0.2–5 μg/μL)	Saccade tasks (detection)	Saccade	Oculomotor	NA	Hikosaka and Wurtz (1985)
SC	Lidocaine (1–5 μL of 2%)	Saccade tasks (detection)	Saccade	Oculomotor	NA	Hikosaka and Wurtz (1986)
Pulvinar	Muscimol (1–1.5 μL)	Cued detection	Bar press	Attention	- - -	Petersen et al. (1987)
SC with unilateral or bilateral FEF[c]	Cooling	Detection	Saccades	Oculomotor	NA	Keating and Gooley (1988)
SC	Lidocaine (? μL of 2%)	Detection	Saccade	Oculomotor	NA	Lee et al. (1988)
Magno- or parvocellular layers of LGN	Lesion (DNR several months)	Detection and Search (oddities task)	Saccade	Visual	NA	Schiller et al. (1990)
Pulvinar or SC	Muscimol (?)	Sensory discrimination	Lever press	Attention	Distracter dependent	Desimone et al. (1990)
SC	Muscimol (0.3–0.9 μL of 0.2–1.0 μg/μL)	Detection	Lever release	Attention	- - -	Robinson and Kertzman (1995)
SC	Muscimol (0.25–1 μL) or Lidocaine (0.25–1.25 μL)	Search (oddities task)	Saccade	Target selection	Distracter dependent	McPeek and Keller (2004)
Pulvinar	Muscimol (2–4 μL of ?) or THIP (2–4 μL of 6.67 μg/μL)	Detection	Saccade or reach	Target selection	Distracter dependent	Wilke et al. (2010)

Table 2 (*Continued*)

Structure	Loss of function method	Task	Behavioral response	Interpretation of deficit	Evidence consistent with attention deficit	Reference
SC	Muscimol (0.5 μL of 5 μg/μL)	Sensory discrimination	Button press or saccade	Attention/ visual extinction	Distracter dependent	Lovejoy and Krauzlis (2010)
LGN and chronic V1	V1 lesion, LGN THIP (2 μL of 6.67 μg/μL)	Detection	Saccade	Visual	NA	Schmid et al. (2010)
SC	Muscimol (0.5 μL of 5 μg/μL)	Search	Smooth pursuit, saccade, or button press	Multiple effector target selection	Distracter dependent	Nummela and Krauzlis (2010)
SC	Muscimol (0.5 μL of 5 μg/μL)	Multiple possible target smooth pursuit task	Smooth pursuit	Weighted integration of visual signals/ target selection	NA/- - -	Nummela and Krauzlis (2011)
SC	Muscimol (0.5 μL of 0.5 μg/μL) (*sic*)	Centrally cued, peripheral reach task	Reach	Reach target selection	Behavior was centrally cued and empirically independent of target salience	Song et al. (2011)
Pulvinar	Muscimol (0.5 μL of 66.7 mM) or GABA (0.4 μL of 25 mM)	NA (Anesthetized)	NA	Control and gate information outflow from V1	NA	Purushothaman et al. (2012)
SC	Muscimol (0.4–0.6 μL of 5 μg/μL)	Cued Change Detection	Button Press	All-or-none aspects of spatial attention	Lovejoy and Krauzlis (2010)	Zénon and Krauzlis (2012)

[a] "Posterior part of the superior limb of the arcuate sulcus."
[b] "Part of Area 6: the posterior bank of the arcuate sulcus and the immediately adjacent cortex caudal to it."
[c] "The FEF probe cooled both the crown and anterior bank of the arcuate sulcus. In order to position the sulcal portion of the probe, supplementary motor cortex of the posterior bank of the sulcus was first removed by aspiration."
[d] "Most of the inferior parietal lobule [Area 7] and also that portion of prestriate cortex immediately posterior to it." *Note*: LIP was not consistently lesioned (see their Figs. 2–4).
[e] "Inferior parietal lobule and both banks of the caudal portion of the superior temporal sulcus."
[f] "The lateral portion of area PG of Von Bonin and Bailey was removed in all animals. The anterior extent of the lesion varied. Damage in the superior–posterior border and depths of the superior temporal (STS) and interparietal (IPS) and sulci also varied."
[g] Distracter-dependence implied but not explicitly argued, and data not shown.

> "Because many spatially mapped structures contribute to competition, unilateral lesions will often cause neglect and extinction syndromes that do not necessarily imply a specific role in attentional control." (Desimone and Duncan, 1995, p. 217).

Thus, a lesion that produces a distractor-dependent behavioral deficit, in the absence of further data, may have an ambiguous interpretation. Specifically, a distractor-dependent deficit is ambiguous when the lesion disproportionately affects the representation of the target over the representation of the distractors—as is the case in all spatially specific lesions. This ambiguity follows directly from any competition-based view of visual processing.

2.3. Cue benefits as the key measure of attention

How then can one unambiguously identify an attentional deficit? Given that the core feature of attention is improved perceptual sensitivity following an attentional cue, perhaps rather than testing sensitivity to a stimulus with and without distractors, a less ambiguous deficit would be a loss of such sensitivity improvements, measured across attention conditions. For example, one might expect that the perceptual benefits of validly cueing a particular location would be eliminated following damage to a purely attentional mechanism. One example of this approach is to employ valid and invalid cueing, and compare performance between these attention conditions using identical visual stimuli (Posner, 1980; Petersen et al., 1987; Robinson and Kertzman, 1995). If a post-lesion performance deficit is due to an attentional dysfunction, one expects that the benefits of valid cues (or costs of invalid cues) will be reduced or eliminated, although these explicit comparisons are not always examined (Robinson and Kertzman, 1995).

In the case of exogenous attention, even cue-validity effects (i.e., the effect of the validity of a cue on representational enhancement or the behavioral benefits of attention) may not be able to distinguish between an attention deficit and a purely visual one (despite the fact that many of the studies which compare valid and invalid cues use exogenous cuing; Posner, 1980; Petersen et al., 1987; Robinson and Kertzman, 1995). An exogenous cue is defined as a cue that is briefly presented (usually immediately preceding target onset), and transiently attracts the subject's attention to the cue's location due to its inherent salience. Obviously, successful visual processing of an exogenous cue is necessary for the cue to drive attention. In the case that an exogenous cue no longer brings about attention following a lesion, is that because an attention mechanism has been disrupted, or because the visual processing of the cue itself is dysfunctional?

Thus, a dysfunction in either attention or vision may produce similar deficits at the behavioral level, and this can confound the interpretation of a loss-of-function experiment. However, attentional and visual dysfunctions are distinguishable. Attention is the ability to select some aspects of one's sensory world for enhanced processing over others. If, in a constant visual world, a subject is not able to gain the perceptual benefits (and/or costs) associated

with attending to some aspect of that world, this is a deficit in attention. Such a deficit can be measured by holding the properties of the visual stimuli (e.g., the number of distractors) constant, and evaluating the perceptual benefits associated with attentional cueing (e.g., a valid cue vs. an invalid cue, or a valid cue vs. a neutral cue) before and after a lesion. The loss of such cued benefits following a lesion of a brain structure, independent of a possible change in absolute performance, would be strong and unambiguous evidence for the role of that structure in the control of attention. To date, no non-human primate lesion studies of any brain area have reported such a deficit. In the absence of studies examining these cue-dependent impairments, much of the evidence for a contribution of specific brain areas to behavior comes instead in the form of deficits on attentional tasks (Table 2).

3. Searching for the sources of attention

A brain region which is a source of selective attention would be expected to display the following characteristics: (1) activity in this area should reflect the location of spatial attention (or the features of feature-based attention), (2) activity in this area is causally related to performance on an attention task, and (3) activity in this area is causally related to the neural signatures of attention in other brain areas (this last can be assessed directly, via manipulations of activity in the source area, and also indirectly by examining Granger causality, etc.). Attentional modulation of neuronal responses has been shown in multiple brain areas, including the visual cortex (McAdams and Reid, 2005; Motter, 1993; Moran and Desimone, 1985; Sheinberg and Logothetis, 2001), PFC (Kodaka et al., 1997; Buschman and Miller, 2007; Lebedev et al., 2004; Kaping et al., 2011), SC (Goldberg and Wurtz, 1972), basal ganglia (Kermadi and Boussaoud, 1995), supplementary eye field (Bon and Lucchetti, 1997), premotor cortex (Pellegrino and di Wise, 1993), and parietal areas LIP (Colby et al., 1996) and 7a (Steinmetz and Constantinidis, 1995). In this section we review the evidence for causal contributions by a handful of brain areas whose manipulation alters performance on an attention task (second criterion). In Section 4 we will review evidence linking these areas to the neuronal correlates of attention in visual cortex (third criterion).

A connection between attention and gaze control goes back as far as the study of attention itself (Ferrier, 1890; Ribot, 1890). Only in recent decades, however, have scientists possessed the neurophysiological and psychophysical techniques needed to examine this link between eye movements and attention in detail. Eye movements seem to drive shifts in attention to the target of the impending gaze shift, as indicated by improved target detection thresholds measured at the endpoint of upcoming saccades (Hoffman and Subramaniam, 1995; Peterson et al., 2004). If the eyes are maximally rotated in their orbits, such that further movement in one direction is impossible, attentional cues in that hemifield no longer produce their usual behavioral benefits, suggesting that eye movement planning may underlie attentional benefits (Craighero et al., 2004). Covert attention, in turn, impacts eye movements: the location of covert attention changes the latencies (Rizzolatti et al., 1987) and trajectories of voluntary or electrically evoked saccades, and the magnitude of these trajectory changes depends upon the difficulty of the attentional task (Sheliga et al., 1994, 1995; Kustov and Robinson, 1996). These behavioral links between eye movements and attention have directed the search for the sources of attention primarily toward brain regions known to play a role in oculomotor control; more recent electrophysiological findings confirm the similar effects of eye movements and covert attention on activity in visual cortex (Steinmetz and Moore, 2014). Although this review focuses on prefrontal contributions to attention, we will briefly discuss studies of several other brain areas that are candidate sources of attention—the SC, LIP, and thalamic nuclei—as these areas are heavily interconnected (Markov et al., 2014), and play complementary roles in controlling eye movements (Hikosaka and Wurtz, 1985; Schiller et al., 1980; Hanes and Wurtz, 2001; Bisley and Goldberg, 2010). The activity across these areas and the interactions between them are believed to be the basis for the deployment of visuospatial attention.

3.1. Superior colliculus

Probing the neurophysiological basis of the behavioral link between oculomotor control and attention, much research has examined the role of the SC in attentional control. Neurons in the SC have spatially restricted RFs with a spectrum of visual and motor response properties; activity in the SC is closely tied to eye movements, with the firing rates of motor-responsive neurons in the SC predicting the time of saccade initiation (Dorris and Munoz, 1998). The visual and motor properties of neurons in the SC are related to cortical depth, with predominately visual neurons located mostly in the more superficial layers, while primarily motor-driven neurons are located mostly in the deeper layers, which project to the oculomotor nuclei. The activity of visually responsive neurons in the SC also reflects the location of covert attention (Ignashchenkova et al., 2004; Goldberg and Wurtz, 1972). Electrical stimulation of the SC evokes saccadic eye movements (Robinson, 1972); stimulation at lower currents (subthreshold stimulation) does not move the eyes, and can therefore be delivered while the animal performs a behavioral task. (This 'microstimulation', delivered through a microelectrode, is typically on the order of 1–100 μA.) Subthreshold microstimulation of the SC improves performance on attentional tasks: spatially specific enhancements in performance, corresponding to the RF of the stimulated SC site, have been demonstrated both for a change detection task (Cavanaugh et al., 2006), and for a motion-discrimination task (Müller et al., 2005).

Reversible pharmacological inactivation of the SC also impairs performance on attentional tasks. Lovejoy and Krauzlis (2010) trained monkeys to perform a motion discrimination task at a cued location in the presence of distracting stimuli. Inactivation of a portion of the SC impaired discrimination performance at the corresponding spatial location, but only in the presence of potentially competing distractor stimuli. Surprisingly, when further experiments examined the effects of SC inactivation during this task on responses in visual cortex (MT and the medial superior temporal area, MST), which are modulated by attentional deployment, they found that the attentional modulation of these visual responses was unaffected by SC inactivation (Zénon and Krauzlis, 2012). This presents an interesting dissociation between the behavioral and neuronal measures of attention, the interpretation and potential implications of which will be discussed more fully in Section 4.

3.2. Parietal cortex

Another oculomotor area implicated in attention is the LIP, located in the lateral bank of the intraparietal sulcus of the parietal lobe. As in SC, LIP neurons display a spectrum of visual and motor responses (Mazzoni et al., 1996; Gnadt and Andersen, 1988). Activity of neurons in LIP also reflects the location of covert spatial attention (Bisley and Goldberg, 2003). However, no microstimulation studies in LIP have replicated the spatially selective, distractor dependent benefits seen in FEF and SC (Moore and Fallah, 2001, 2004; Müller et al., 2005; Cavanaugh et al., 2006). One study did show a reaction-time benefit in which subthreshold microstimulation provided a benefit similar to a valid cue in a distractor-free

detection task (Cutrell and Marrocco, 2002), along with a spatially generalized speeding of responses on trials with no cue. LIP inactivation studies also suggest that the area causally contributes to attention. Wardak et al. (2002) first reported that LIP inactivation biased target selection and delayed visual search for targets appearing in the affected hemifield. Subsequent studies showed that LIP inactivation produces distractor and load dependent reaction time deficits in a covert attention task (Wardak et al., 2004). More precisely targeted inactivation experiments suggest a further localization of function within LIP, with the ventral portion contributing to attention-dependent search tasks, while inactivation of the more dorsal portion only affects saccades (Liu et al., 2010). One hypothesis about the unique roles of prefrontal and parietal areas in visual attention is suggested by a study dissociating top-down and bottom-up attention. In that study monkeys were trained to perform both bottom-up and top-down search tasks, and electrophysiological recordings were performed in both parietal and prefrontal cortex (Buschman and Miller, 2007). The relative time of target discrimination in the two areas depended on the nature of the search: in the top-down task, prefrontal neurons reflected the location of the target earlier than parietal neurons, while in the bottom-up search the reverse was true. These results suggest that LIP is more involved in bottom-up, or salience-driven attention, while prefrontal areas are more critical for directing top-down or cue-driven attentional deployment. However, Katsuki and Constantinidis (2012) found that neurons in PFC represented the presence of a target stimulus identified by bottom-up salience as early as neurons in LIP, despite PFC having slower visual latencies. These results held both for a match-to-sample version of the task, and a reaction time oddball-detection task, although overall target discrimination times in all areas were ~20 ms faster in the reaction time version of the task; they also showed that the detection times in LIP and dlPFC were similar over a range of target-distractor similarity and stimulus sizes. Although both used a color-oddball detection task, several differences exist between the task used in this and previous studies: the Katsuki and Constantinidis task used a lever release rather than an eye movement, and presented stimuli farther in the periphery. These results challenge the distinction suggested by earlier findings, and suggest that the PFC may also be involved in directing bottom-up attention based on stimulus properties; further studies will be needed to determine exactly how different task demands preferentially recruit prefrontal or parietal circuits.

3.3. Thalamic nuclei

The major components of the visual portion of the thalamus are the lateral geniculate nucleus (LGN), thalamic reticular nucleus (TRN), and pulvinar nucleus; activity in all three of these nuclei is modulated by attention (McAlonan et al., 2008; Petersen et al., 1985). The TRN contains neurons with short-latency visual responses (McAlonan et al., 2008), and sends inhibitory input to the LGN and pulvinar nuclei. It also receives signals from areas implicated in attention and cognitive control, including the PFC and SC (Guillery and Harting, 2003; Zikopoulos and Barbas, 2006). The TRN is thus anatomically positioned to regulate visual processing at the earliest stages, by gating activity in the LGN; it may also be indirectly involved in coordinating activity between areas in the visual hierarchy, via its effects on pulvinar activity. Attention decreases the activity of TRN neurons (McAlonan et al., 2008), thus reducing inhibitory input to the LGN and pulvinar, and potentially contributing to the attention-driven increases in activity in those nuclei. However, only the initial visual response in TRN is affected by attention, whereas in LGN neurons attentional modulation is present in the initial response and re-emerges in the sustained response, suggesting the involvement of additional sources in attentional modulation of LGN activity. This change in LGN activity is usually characterized as a change in visual processing rather than a 'source' of attentional modulation, although changes in LGN signaling presumably contribute to, and are reflected within, the changes in visual cortical areas.

The extensive and rather complicated anatomical connectivity of the pulvinar nucleus has long fueled speculation as to its functional role. The pulvinar receives input from, and projects to, numerous cortical areas; in general, these cortico-pulvinar-cortical pathways serve to link areas which also have direct cortico-cortical connections to one another (Shipp, 2003). A large portion of the pulvinar receives input from V1 and other occipital visual areas, and roughly mirrors their topographical maps of visual space. These cortico-pulvinar-cortical pathways have been proposed as a means of synchronizing activity between cortical areas. Visual responses of pulvinar neurons are modulated by attention (Petersen et al., 1985; Bender and Youakim, 2001), and it is one of the first structures whose pharmacological inactivation was shown to impair performance on an attentional task (Petersen et al., 1987). One recent study examines the role of the pulvinar in coordinating activity between extrastriate visual areas V4 and TEO (Saalmann et al., 2012). Simultaneous recordings of spiking and LFP activity were made from all three areas, in sites with overlapping visual RFs; since the pulvinar regions connected to V4 and TEO are only partially overlapping, the experimenters used diffusion tensor imaging to verify that the pulvinar recording site was connected to both V4 and TEO. Monkeys performed a cued target discrimination in a crowded array; in this task pulvinar neurons responded to a cue appearing in their RF, maintained a slightly elevated rate during the delay between cue and stimulus presentation, and exhibited an elevated visual response to the target in the stimulus array. Alpha-band spike-field coherence within the pulvinar was also elevated during the cue and delay period when attending the neuron's RF. Synchrony between V4 and TEO also increased with attention, as measured by increases in alpha- and gamma-band coherence for LFPs recorded from both areas. Attention also increased synchrony between the pulvinar and both visual areas, as reflected by increases in pulvinar spike-visual LFP and LFP–LFP alpha-band coherence. Granger causality analysis revealed an attentionally modulated influence of the pulvinar on alpha activity in both V4 and TEO during the delay period; in contrast, direct influences of V4 on TEO (and vice versa) were strong during visual stimulation but weak during the delay period, and not altered by attentional state. These results suggest that pulvinar activity drives alpha-band synchrony between V4 and TEO based on the location of covert attention, which in turn could modulate the sensitivity of TEO to V4 input when the target array appears.

Thus the thalamic nuclei may contribute to producing signatures of attention in visual cortex, particularly those involving oscillations and inter-areal synchrony, although their own attentional modulation is attributed to input from other areas.

3.4. Prefrontal cortex: FEF and dlPFC

The PFC has long been considered a potential seat of 'executive control', encompassing a variety of cognitive functions that control behavior in situations that require more than a fixed stimulus–response mapping. Insofar as top-down attention depends upon context, rules of the task, behavioral goals, and maintaining cue information, it seems likely to involve the PFC, where all of these types of activity have been reported (Miller and Cohen, 2001; Knudsen, 2007; Miller and D'Esposito, 2005). The presence of anatomical projections from the PFC to a variety of sensory cortical areas (Yeterian et al., 2012) suggests the ability of these representations of cognitive factors in PFC to influence sensory

signals. The ability of prefrontal activity to influence visual cortical processing has been experimentally verified both in monkeys, where cooling of the PFC alters inferotemporal responses during a memory task (Fuster et al., 1985), and in humans, where prefrontal lesions reduce visually evoked EEG activity during a visual detection task (Barceló et al., 2000).

Among the prefrontal areas, the dlPFC initially drew much attention for its role in spatial working memory (Funahashi et al., 1989, 1993; Williams and Goldman-Rakic, 1995). Holding a location in spatial working memory closely resembles spatial attention, in terms of the brain areas activated (Ikkai and Curtis, 2011), the effects on visual discrimination (Awh et al., 1998; Smyth, 1996), and the effects on visual cortical responses (Awh et al., 2000; Jha, 2002; Postle et al., 2004). In combination with attention deficits in human lesion patients (Knight et al., 1995), these links and the dlPFC's established role in spatial working memory make it a strong candidate as a source of spatial attention. Responses in dlPFC do reflect the location of covert attention (Everling et al., 2002; Lebedev et al., 2004; Buschman and Miller, 2007). To more closely examine the relationship between the neural representations of attention and working memory within dlPFC, monkeys were trained on a task requiring the simultaneous maintenance of memory of one location while attending to another; in this task, activity in the dlPFC reflects both the attended and the remembered location (Lebedev et al., 2004). Individual prefrontal neurons could reflect the attended location, the remembered location, or both. Interestingly, those neurons that represented both locations usually had different spatial tuning for the remembered vs. attended location; these 'multi-tasking' neurons were more strongly tuned than single-variable neurons, and improved the population representation of attended and remembered locations (Messinger et al., 2009). This is one of several recent experiments suggesting that a focus on easily interpretable neural tuning is at odds with the actual nature of neural processing (Rigotti et al., 2013; Mante et al., 2013; Churchland et al., 2012; Raposo et al., 2014).

Another aspect of attention, along with the enhancement of target representations, is the ability to ignore interference from distracting stimuli, which may entail the suppression of neuronal responses to distractors (Bisley and Goldberg, 2006; Pinsk et al., 2004; Hopf et al., 2006; Boehler et al., 2009). One recent set of experiments compared prefrontal and parietal activity during a memory guided saccade task with distractors, as well as the effects of inactivating each area on task performance (Suzuki and Gottlieb, 2013). Distractor activity in dlPFC was more strongly suppressed than in LIP: in dlPFC distractor responses did not surpass the sustained target-related activity, while in LIP responses to distractors were transiently greater than the sustained target response. Prefrontal responses to distractors were also more closely related to task performance than those in LIP: the magnitude of dlPFC responses to distractors decreased as distance and time between the target and distractor increased, mimicking trends in the monkeys' error rates, and producing a correlation between dlPFC distractor responses and the error rate across conditions. In contrast, LIP distractor responses were larger for the distant, most efficiently behaviorally suppressed, distractors, and there was no correlation between distractor responses in LIP and error rate across conditions. Inactivating dlPFC also had a much greater impact on behavioral susceptibility to the distractors, suggesting that prefrontal activity is more critical than the parietal response for ignoring and suppressing distracting stimuli. Consistent with a distinct prefrontal contribution to distractor-resilience, prefrontal but not parietal neurons have been shown to maintain a spatial representation through distractor representations in a memory task (Qi et al., 2010). The current state of the literature now points to LIP playing a primary role in representing salience and priority (Bisley and Goldberg, 2010; Gottlieb et al., 1998), while its activity in the top-down control of attention depends upon PFC.

Originally identified as an oculomotor region within the PFC (Ferrier, 1890; Robinson and Fuchs, 1969), the role of the FEF in covert attention has now been extensively studied. Tight behavioral links between eye movements and attention initially suggested the FEF as a potential source of attention (reviewed in Moore et al., 2003). The FEF is anatomically well positioned to modulate visual responses throughout cortex, via direct feedback projections to a number of visual cortical areas (Markov et al., 2014; Schall et al., 1995; Stanton et al., 1995; Anderson et al., 2011). It is also interconnected with other areas suggested as sources of attention, including LIP (Stanton et al., 1995; Anderson et al., 2011), SC (Stanton et al., 1988; Sommer and Wurtz, 2000), and neighboring prefrontal areas (Stanton et al., 1993). Individual neurons within FEF respond to visual stimuli and/or saccades to a particular region of space (their RF), and may exhibit any combination of visual, motor, and sustained response properties (Bruce and Goldberg, 1985). The sustained activity of FEF neurons in also modulated by the locus of covert attention (Thompson et al., 2005; Armstrong et al., 2009).

FEF has also been causally linked to the deployment of spatial attention. Electrical stimulation of the FEF at sufficiently high currents will produce fast, ballistic eye movements of a repeatable direction and amplitude, depending on the specific site of stimulation (Bruce et al., 1985; Robinson and Fuchs, 1969). Subthreshold microstimulation of the FEF while the animal performs an attention-demanding change detection task improves performance, improving the animal's ability to detect small changes in luminance in the RF of the FEF site being stimulated (Moore and Fallah, 2001). This improvement is specific both in space, corresponding to the RF of the stimulated FEF site, and in time, detection being improved only in the 300 ms following stimulation, with more dramatic effects at shorter stimulation-detection delays (Moore and Fallah, 2004). Microstimulation of the FEF also mimics the ability of attention to increase the guidance of saccades by the visual features of the saccade target (Schafer and Moore, 2007). In humans, transcranial magnetic stimulation of the FEF region alters BOLD responses in early visual areas, providing behavioral benefits and enhanced responses to peripheral visual stimuli (Ruff et al., 2006).

Mirroring the results of the microstimulation studies, pharmacological suppression of FEF activity produces behavioral deficits in attention-demanding tasks. Wardak et al. (2006) demonstrated that reversible inactivation of a portion of FEF impaired both visually guided saccades, and performance on a covert visual search and visual discrimination task. Similarly, Monosov and Thompson (2009) showed spatially selective impairments on a covert visual search; these deficits were most pronounced on invalid-cue trials, when the animal would need to internally generate a shift of attention to the location corresponding to the FEF inactivation.

Electrical or pharmacological manipulation of neuronal activity allows a direct probe of the causal relationship between that activity and behavior; however, the spatiotemporal patterns of activation produced by these manipulations are unlikely to precisely mimic those occurring during natural behavior. One way to experimentally manipulate brain activity in a more naturalistic manner is via operant conditioning—training the animal to voluntarily increase or decrease neuronal activity in a brain area based on feedback. Does a voluntary increase or decrease in FEF activity reproduce the behavioral signatures of attentional deployment? Schafer and Moore (2011) used operant training techniques to assess the effect of voluntary changes in FEF activity on visually guided behavior (Schafer and Moore, 2011).

Monkeys were given real-time auditory feedback indicating the activity at one FEF site, and rewarded for either increasing or decreasing that activity (in alternating 'UP' and 'DOWN' blocks of trials) without moving their eyes. The authors then tested the behavioral and neurophysiological consequences of these voluntary changes in FEF activity by introducing probe trials in which the animal had to perform a visual search. When search targets appeared in the RF of the FEF site being modulated, monkeys were less likely to detect the target when they were suppressing FEF activity (DOWN trials vs. UP trials). At a neuronal level, FEF neurons discriminate visual search targets from distractors (Buschman and Miller, 2009; Schall and Hanes, 1993); this effect was larger when the animal was attempting to increase FEF activity at the site (UP trials vs. DOWN trials), but not dependent on spontaneous fluctuations in firing rate at the site. Thus internally driven, voluntary changes in the activity of FEF neurons reproduce both behavioral and neurophysiological signatures of covert attention, without training on an attention task.

On the whole, there is currently much greater evidence for the role of FEF in attention than there is for dlPFC. It is possible that the stronger topographic organization of FEF, which greatly facilitates spatially selective manipulation of activity via microstimulation or localized drug infusion, is partially responsible for this disparity. However, recordings comparing FEF and dlPFC activity during the same top-down attention task show that FEF reflects the location of attention earlier than the dlPFC (Buschman and Miller, 2007). FEF spiking activity also reflects the location of a covert search target earlier than the local LFP (which putatively reflects input to the area) (Monosov et al., 2008). Although this criterion has not been tested for and applied to other areas, it may implicate FEF as the first area to signal the location of covert attention. The dlPFC and the FEF are reciprocally connected, both directly and indirectly via neighboring ventrolateral PFC (Stanton et al., 1993; Anderson et al., 2011; Markov et al., 2014), and both display sustained activity during spatial working memory tasks (Funahashi et al., 1989; Sommer and Wurtz, 2000). In combination with the distractibility effects of dlPFC inactivation (Suzuki and Gottlieb, 2013), discussed above, these results suggest that FEF and dlPFC work together to maintain task-relevant information and buffer it from the effects of distractors.

3.5. Prefrontal homology across species

Although this review has focused on experiments performed in non-human primates, an increasing number of attention studies are carried out in a rodent model (Sagvolden et al., 2005; Arnsten and Dudley, 2005). The rodent model system allows experimenters to employ genetic and neurophysiological techniques not yet widely applied in primates. Here, we survey the similarities and differences between primate and rodent prefrontal anatomy, which will ultimately determine the extent to which results from these model systems can be transferred and applied to our understanding of human prefrontal function.

The frontal lobe has a long history of study in multiple model organisms, particularly non-human primates (e.g., rhesus monkeys) and rodents (e.g., rats). The homology of PFC in humans and monkeys has been well documented (Pandya and Yeterian, 1996; Petrides and Pandya, 1999; Petrides et al., 2012). In contrast, the similarity between the PFC of rodents and primates has been historically controversial (Preuss, 1995; Uylings et al., 2003; Wise, 2008; Brodmann, 1909; Rose and Woolsey, 1948). Primate PFC is grossly divided into three different cortical areas: orbito frontal cortex, medial PFC, and dlPFC (Preuss, 1995). In the rodent, homologous structures have been proposed for all three of these primate prefrontal areas based on a number of criteria including connectivity with other cortical and sub-cortical brain regions, the distribution of certain neurotransmitters and neurotransmitter receptors, and functional properties (Ongür and Price, 2000; Uylings et al., 2003).

The primate dlPFC, including the FEF, is the area of the PFC most implicated in visual selective attention. Therefore, the question of whether a structure homologous to the primate dlPFC exists in the rodent is of particular interest, and will be emphasized in this review. If such structures exist in the rodent they are likely the rodent frontal areas named frontal cortical area 2 (Fr2, also known as medial precentral area), dorsal anterior cingulate area (ACd), and possibly also the prelimbic cortical area; however whether these rodent structures are more homologous with primate dlPFC or primate premotor cortex (PMC) is not settled (Preuss, 1995; Uylings et al., 2003). Historically, several similarities between primate dlPFC and rodent frontal structures have been used in support of a dlPFC homology. These include (1) innervation from the mediodorsal nuclei of the thalamus, (2) dopaminergic innervation, (3) connections with multimodal association cortex, and (4) the ability to evoke orienting movements with intracortical electrical stimulation (Van Eden et al., 1992; Sinnamon and Galer, 1984; Preuss, 1995; Akert, 1964; Glowinski et al., 1984). Critics argue that these similarities are not satisfactory for identifying a rodent dlPFC homologue because all of these characteristics (i.e., connectivity with thalamic mediodorsal nuclei, dopaminergic innervation, connection with multimodal association cortex, and electrically evoked orienting movements) are not unique features of primate dlPFC but rather are common features of almost all areas in the primate frontal lobe, including non-PFC areas like the PMC (reviewed in Preuss, 1995). Furthermore, unique characteristics of the primate dlPFC, which distinguish it from other frontal lobe structures in the primate, are conspicuously absent from structures of the rodent frontal lobe (Preuss, 1995). For example although rodent frontal structures like Fr2, and primate frontal structures including primate PMC and primate dlPFC all project to intermediate and deep layers of the superior colliculus, primate dlPFC is the only one of these structures that projects to the SC's superficial layers (Beckstead, 1979; Leonard, 1969; Fries, 1984; Preuss, 1995). Nonetheless, advocates for rodent-primate frontal homology have argued that since any single feature uniquely defining primate dlPFC is tenuous, a more informative way to define primate PFC for evaluating rodent-primate homology is in the relative strengths of its connections with multiple cortical and sub-cortical structures (Uylings and van Eden, 1990). In this vein, it has been argued that rodent Fr2 and ACd are homologous with primate dlPFC (and/or the FEF specifically) because as in the primate, rodent Fr2 and ACd exhibit relatively stronger reciprocal connections with the mediodorsal nuclei than with other thalamic nuclei, such as the ventrolateral or ventromedial nuclei (Uylings et al., 2003, but see Condé et al., 1990).

Given this controversy about whether certain rodent frontal structures are more appropriately designated as homologous with primate PFC or primate PMC, it is interesting to consider the characteristics of the primate FEF (the most posterior extent of dlPFC) and its potential rodent homologue. Although classically considered part of PFC due to its defined granular layer, primate FEF also possesses PMC-like features not shared by the rest of PFC (e.g., large Layer V pyramidal neurons), and has sometimes been referred to as a transition structure between PMC and PFC (Stanton et al., 1989; Preuss, 1995). Advocates for a specific homology between primate FEF and rodent Fr2/ACd heavily weigh functional similarities in addition to their anatomical similarities. For example, electrical stimulation of these areas in rats, monkeys, and humans produce eye and head orienting movements (Sinnamon and Galer, 1984; Erlich et al., 2011; Bruce et al., 1985; Monteon et al., 2010; Blanke et al., 2000). Furthermore, recently Erlich et al. (2011) used muscimol to reversibly inactivate

an area of the rat frontal lobe that they estimated to be Fr2 based on stereotactic coordinates and electrical stimulation. Erlich and colleagues refer to this area as the rodent frontal orienting field. They found that muscimol inactivation of the rat frontal orienting field produced contralateral orienting deficits that were significantly exacerbated for memory-guided orienting movements compared to stimulus-guided ones, the latter of which does not require a time delay between the stimulus cue and the initiation of movement (Erlich et al., 2011). In the primate, behaviors that require maintaining information in the absence of any sensory stimuli across delays of seconds to tens of seconds, such as memory-guided orienting tasks, are classically associated with the function of the dlPFC (Fuster and Bauer, 1974; Goldman-Rakic, 1995). Specifically, the inactivation of primate FEF produces severe deficits in memory-guided eye movements with only minor impairments in stimulus-guided orienting, qualitatively resembling the pattern observed by Erlich and colleagues following inactivation of rat frontal orienting field (Sommer and Tehovnik, 1997; Dias and Segraves, 1999).

Thus, although it remains controversial, there are some structural and functional data that suggest the rodent frontal areas Fr2 (Erlich's frontal orienting field), ACd, and possibly the prelimbic cortical area may be homologous to primate dlPFC and/or specifically the primate FEF. This homology promises that both rodents and primates may be informative models for understanding PFC function.

4. Linking source areas to neuronal signatures in other areas

Having discussed the numerous reported neuronal signatures of attention (Section 1.1), and their correlation with behavioral performance (Section 1.3), as well as the areas that are thought to produce both the behavioral effects of attention and these changes in visual cortical signals (Section 3), we now look more closely at the evidence that particular 'source' areas drive attentional signatures in other brain areas. Understanding which signatures are generated by the activity of which candidate sources of attention is critical for our understanding of how attentional benefits are brought about.

4.1. Linking sources to signatures via lesions

If sources of attention operate by enhancing representations in visual cortex, which in turn underlie improved behavioral performance, then we would expect the signatures of attention in visual cortex before and after inactivation of source areas to display these characteristics: (1) signatures are correlated with behavior during normal task performance, (2) inactivation of the source area reduces the signature (correct after < correct before) while (3) preserving the difference between correct and wrong (correct after > wrong after). In fact, the number of attention related studies in which activity in one area is manipulated while activity in another is recorded is quite small (Fig. 1). Only three neurophysiological studies have manipulated potential attentional source areas and recorded from visual cortex during covert attention tasks (Gregoriou et al., 2014; Monosov et al., 2011; Zénon and Krauzlis, 2012); we discuss the results of each in detail here.

Monosov et al. (2011) examined the effect of FEF activity on object responses in IT during a visual search task. They show that the reversible pharmacological inactivation of FEF reduces the response of IT neurons to their preferred stimulus (at the spatial location corresponding to the inactivation); this effect is only evident when the stimulus appears as part of a search array, rather than in isolation. The implied role for FEF in driving IT object selectivity in crowded scenes dovetails nicely with modeling showing that attentional cues make IT neurons' response to a crowded scene more closely resemble their response to the attended object presented in isolation (Zhang et al., 2011). However, although a visual cue indicated the location of the target object on half of the trials, in Monosov et al. (2011) no significant neuronal modulation based on the cue was measured in the responses of the IT neurons, and so the impact of FEF inactivation *on an attentional modulation* cannot be examined.

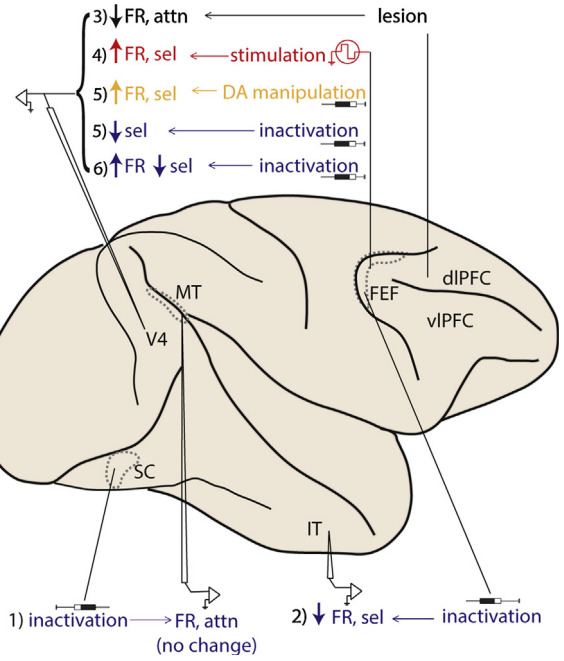

Fig. 1. Manipulations of candidate attention 'sources' and their effects on visual cortical responses. (1) Inactivating SC did not alter firing rate (FR) and attentional modulation (attn) of responses in MT and MST (Zénon and Krauzlis, 2012). (2) Inactivating FEF reduced the magnitude and selectivity (sel) of visual responses in IT (Monosov et al., 2011). (3) Lesioning PFC reduced the magnitude and attentional modulation of V4 responses (Gregoriou et al., 2014). (4) Microstimulation of FEF increases the firing rate and selectivity of V4 responses (Moore and Armstrong, 2003; Armstrong and Moore, 2007). (5) Infusion of a D1R antagonist in FEF increases the firing rate, selectivity, and reliability of V4 responses (Noudoost and Moore, 2011a). In the same study, inactivating FEF reduced the selectivity but not the overall magnitude of V4 responses. (6) Inactivating FEF increases the magnitude but decreases the selectivity of presaccadic modulation in V4 (Noudoost et al., 2014). Pharmacological inactivation experiments in blue; black, permanent lesions; red, microstimulation; orange, D1R antagonist.

Gregoriou et al. (2014) studied the effects of prefrontal lesions on many of the signatures of attention in V4 and their relationship to performance. Unfortunately the orientation discrimination task used does not provide a behavioral measure of attention (cue benefits, as discussed in Section 2), and these prefrontal lesions did not produce robust behavioral effects – although the animals had slower reaction times to stimuli in the lesioned hemifield, and higher rates of incorrectly responding to distractors in the lesioned hemifield, only one animal showed a small decrease in performance in the contralesional visual hemifield (the other had no change). It is not clear whether this mostly preserved behavior is the result of compensation, or because the task was performed with stimuli well above the discrimination threshold. Despite the modest behavioral deficit, the examination of the correlations between various V4 modulations and behavior before and after the PFC lesions provides valuable information. The authors looked at firing rate, gamma- and beta-frequency band coherence and LFP power, as well as alpha power and noise correlation. Among these measures, only cue-driven changes in firing rate, gamma-band LFP power and coherence meet the three criteria listed above: a behavioral correlation before and after inactivation, but with the modulation reduced by inactivation. Ignoring the behavioral

correlation component, PFC lesions also disrupted the suppression of beta band power and coherence, and the reduction in noise correlations usually produced by attention. These results indicate that the PFC plays a role in generating attentional changes in firing rate, gamma band power and synchrony, beta band power and synchrony, and noise correlations within V4—although possibly only the first three of these are related to behavioral performance.

Zénon and Krauzlis (2012) examined the impact of inactivating the SC on attentional modulation of MT and MST neurons. SC inactivation, which significantly impaired the animal's performance on a cued change detection task, had no effect on the cue-directed changes in the firing rate of MT and MST neurons. The behavioral effect of the SC inactivation was to reduce the animals' probability of correctly detecting changes when the target appeared in the affected region, and to slightly increase the likelihood of incorrectly responding to changes at the uncued location. One interpretation of these results is that in fact visual processing, and the cue-driven attentional modulation of visual processing, were unimpaired, but the animal was biased against responding to stimuli appearing at the location of the SC inactivation (Smolyanskaya and Born, 2012). While the implications of this result for the connection between attentional modulations of visual responses and behavior require further examination, the finding does clearly indicate that these attentional modulations do not depend on the activity of neurons in SC.

One other study (Noudoost et al., 2014) has examined the effects of inactivating an attention source area on visual cortical activity—this time in the context of eye movements and the accompanying changes in visual responses, which may represent the shift of attention that occurs just prior to saccades (Moore, 1999; Hoffman and Subramaniam, 1995; Peterson et al., 2004). V4 activity typically increases just prior to a saccade into the V4 neuron's RF (Moore et al., 1998), and this increase in the firing rate is accompanied by enhanced visual guidance, improved feature selectivity and reduced response variability (Steinmetz and Moore, 2010; Moore and Chang, 2009; Moore, 1999). The saccade-related activity in FEF was widely hypothesized as a source of this presaccadic modulation. However, surprisingly, inactivating the FEF actually resulted in an increase in the magnitude of presaccadic activity in V4 (Noudoost et al., 2014;Fig. 2A). The most

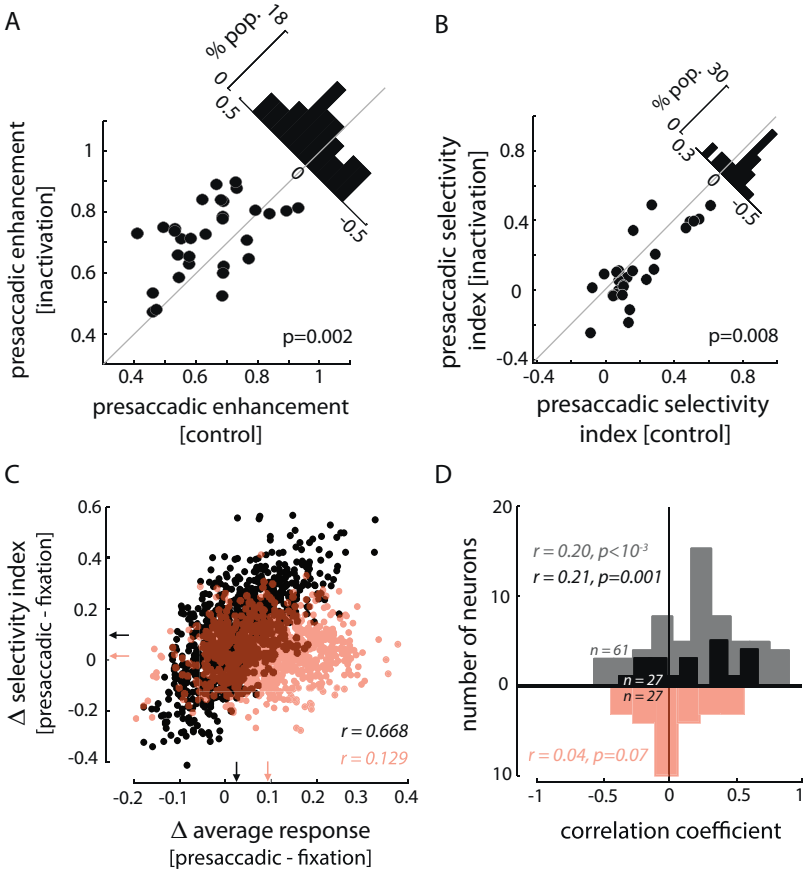

Fig. 2. FEF inactivation has different effects on, and disrupts the relationship between, activity and selectivity in V4. Recordings were made from 33 V4 neurons with RFs overlapping with the RF of an FEF site, before and after pharmacological inactivation of FEF. (A) FEF inactivation increases presaccadic enhancement in V4. The scatter plot shows presaccadic enhancement for saccades made into the RF of 33 V4 neurons, before (*x*-axis) and after (*y*-axis) FEF inactivation. Presaccadic enhancement is a comparison of activity during the presaccadic period with that during fixation, measured as the area under the receiver operating characteristic (ROC) curve. The histogram on the diagonal shows the difference in enhancement. (B) FEF inactivation reduces V4 presaccadic selectivity indices. For 27 V4 neurons with significant orientation selectivity, a comparison of presaccadic stimulus selectivity indexes for saccades made into the RF before (*x*-axis) and after (*y*-axis) FEF inactivation is shown. The histogram on the diagonal shows the difference in selectivity index. (C) FEF inactivation disrupts the relationship between presaccadic enhancement and the stimulus selectivity index of V4 neurons. The scatter plot shows the presaccadic changes in average response (*x*-axis: normalized firing rate during presaccadic period–fixation period) and stimulus selectivity (*y*-axis: selectivity index during presaccadic period–fixation period) for an example V4 neuron before (black) and after (salmon) inactivation. Each point represents changes (Δ: presaccadic vs. fixation) in average response (preferred and nonpreferred stimuli) and stimulus selectivity index (preferred vs. nonpreferred) computed for a randomly selected subset of trials. There is a correlation between presaccadic changes in average response and selectivity prior to FEF inactivation (black), but not following inactivation (salmon). Arrows indicate the marginal means. Pearson correlation coefficients (*r*) computed for data before and after inactivation are shown. (D) Distributions of enhancement-selectivity correlation coefficients for 27 V4 neurons recorded before (black) and after FEF inactivation (salmon), calculated using the sub-sampling method illustrated in C. Presaccadic enhancement and selectivity are significantly correlated prior to, but not following, FEF inactivation. Presaccadic enhancement and selectivity were also correlated in a separate dataset of 61 V4 neurons from a previous study (Moore and Chang, 2009), shown in gray. %pop., percent of population; *p* indicates *p* value in Wilcoxon Signed Rank test.

straightforward interpretation of this result, that FEF exerts a direct inhibitory influence on V4, is rendered unlikely by several previous findings. First, a recent anatomical and histological examination of FEF inputs to V4 found that a vast majority of the FEF input to V4 consists of excitatory synapses onto pyramidal neurons (Anderson et al., 2011). In addition, driving FEF activity via microstimulation rapidly *increases* the magnitude and selectivity of V4 visual responses (Moore and Armstrong, 2003; Armstrong and Moore, 2007). Together these results strongly suggest that the direct projections from the FEF exert an excitatory gain modulation on visually driven inputs to V4. Therefore, the authors suggest that executing an eye movement in the absence of normal FEF function may require greater activity in other parts of the eye movement circuitry (to compensate for the missing FEF drive to oculomotor nuclei), and that this compensatory increase in the activity of other oculomotor areas, such as LIP or SC, may in turn drive the increase in presaccadic activity in V4 (direct evidence of an analogous compensatory effect exists elsewhere in the oculomotor system: SC inactivation, which partially impairs saccades, sometimes produces an increase in presaccadic FEF activity (Berman et al., 2009)). Another possibility is that the increase in response magnitude results from a release of lateral inhibition within V4. If V4 sites interact competitively, as suggested by normalization models of visual processing (Reynolds et al., 1999; Reynolds and Heeger, 2009), then the spread of FEF inactivation to neighboring sites, and a corresponding drop of activity at nearby V4 sites following the loss of excitatory FEF input to those sites, could result in greater activity through the loss of competitive local interactions. (However, this hypothesis implies the existence of other V4 sites or neurons whose presaccadic activity drops following FEF inactivation.).

Importantly, the FEF inactivation had opposite effects on the magnitude vs. the selectivity of V4 presaccadic activity—the absolute firing rate of V4 neurons increased, but their feature selectivity decreased (Fig. 2B)—suggesting that multiple mechanisms are at play. Not only did FEF inactivation differently impact the magnitude and selectivity of presaccadic activity in V4, it also altered the relationship between the two. Normally, the presaccadic increase in activity and presaccadic increase in selectivity are correlated from trial to trial; following FEF inactivation, however, presaccadic changes in firing rate were no longer correlated with changes in selectivity (Fig. 2C and D). This finding suggests that there are multiple sources of presaccadic modulation in V4, one of which increases the magnitude but not the feature selectivity of V4 responses, while the FEF uniquely contributes to increasing the presaccadic selectivity of V4. This contribution of FEF to the feature selectivity of V4 responses in consistent with the effects of microstimulating FEF on visual responses (Armstrong and Moore, 2007), and FEF's suggested role as a source of top-down multiplicative modulation of visual signals during covert attention, i.e., providing a non-selective spatial signal which improves feature selectivity by enhancing the gain of visual signals (Clark and Noudoost, 2014; Squire et al., 2013).

4.2. Linking sources to signatures by driving activity

One potential problem with inactivation and lesion studies is the possibility of compensation by other brain areas, which makes the interpretation of results difficult, especially when unexpectedly enhanced responses are observed (for example, the increase of V4 presaccadic activity after FEF inactivation in Noudoost et al., 2014). Driving activity in a source area, rather than inactivating it, offers another means to examine the relationship between sources and neuronal signatures elsewhere in the brain. Positive results offer a clear interpretation: activity in this area is sufficient to cause the observed signature.

Subthreshold electrical stimulation of FEF, previously shown to improve attentional performance (Moore and Fallah, 2001, 2004), also produces a transient enhancement of visual responses in area V4, for neurons whose RF corresponds with the stimulated FEF RF (Moore and Armstrong, 2003). This enhancement is greater for stimuli matching the preferred features of the V4 neuron, and in the presence of competing distractor stimuli. These changes in response magnitude improve the feature discriminability of the response (Armstrong and Moore, 2007). The effect is quite spatially specific, capable of enhancing the response to one of multiple stimuli appearing within the V4 neuron's RF (Armstrong et al., 2006). A combined microstimulation and fMRI study indicates that the influence of FEF on visual responses is not restricted to V4, but rather enhances visual activity and increases contrast sensitivity in many visual areas (Ekstrom et al., 2008, 2009). These causal findings, all performed during passive viewing by the animal, are supported by correlative measures during attentional tasks: Granger causality measured in both fMRI and neurophysiological studies indicates a top-down influence of the FEF on activity in visual cortex during attention (Bressler et al., 2008; Gregoriou et al., 2009b). These findings, reviewed in greater detail elsewhere (Awh et al., 2006; Squire et al., 2013; Noudoost et al., 2010), figure heavily into our current understanding of attentional modulation (Section 5). To date no studies have activated potential source areas while recording signatures of attention during a covert attention task; such experiments could help answer the crucial question of which source area generates which signatures of attention in visual areas.

4.3. Mechanisms of attention within the PFC

Having identified the FEF as an area meeting many of the criteria for being a source of both the behavioral effects of attention and its correlates in extrastriate visual cortex, it is possible to move one level down in the search for the source of visual attention, from brain regions to the level of individual neurons. As mentioned, response properties within the FEF are highly heterogeneous. Which types of neurons drive the behavioral and neuronal effects of attention? To what extent are these groups of neurons within FEF overlapping with or segregated from those controlling eye movements (or another closely related cognitive function, working memory)?

Noudoost and Moore (2011a) suggest a pharmacological method for dissecting the roles of different populations of FEF neurons in attention (Fig. 3A). They identified a cellular-level mechanism, i.e., dopamine D1-type receptors (D1Rs) within FEF, sufficient to mimic the effects of attention in visual cortex. First, they quantified the animal's tendency to select a target within the RF of the FEF site by parametrically changing the onset asynchrony between the RF target and another stimulus outside the RF (Fig. 3B). They found that infusion of small volumes of the D1R antagonist SCH23390, which has already been shown to enhance the persistent activity within PFC (Williams and Goldman-Rakic, 1995), into the FEF increases the animal's tendency to choose targets appearing within the FEF RF. Infusion of the dopamine D2-type receptor (D2R)-agonist Quinpirole, which has been shown to enhance perisaccadic activity within PFC (Wang et al., 2004), also biased target selection in this task. Dopamine receptors exert a modulatory influence rather than directly driving neural activity, and the effects of manipulating dopamine signaling tend to follow an inverted-U pattern with some optimal level of dopaminergic signaling, which may be different for the two classes of receptors. Based on previous psychophysical, neurophysiological, and iontophoretic studies, a D1R antagonist and D2R agonist were selected as being most likely to enhance persistent and saccade-related activity respectively, as reviewed in Clark and Noudoost

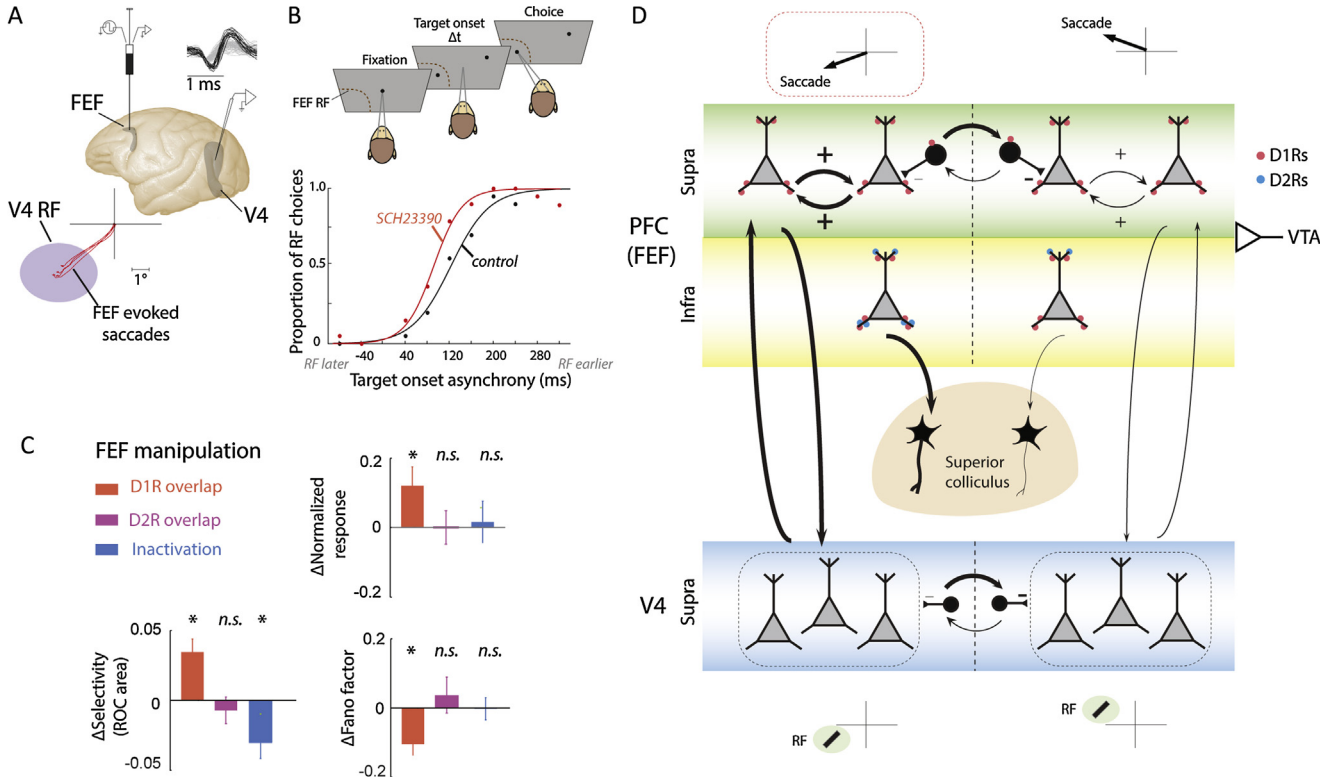

Fig. 3. D1Rs and D2Rs in PFC play different roles in modulating posterior cortical responses and target selection. (A) Noudoost and Moore (2011a) infused a D1R antagonist (SCH23390) into the FEF while recording from V4 neurons with RFs overlapping the area of space represented at the site of drug infusion; the visual responses of the same V4 neurons were recorded before and after infusion of drugs into the FEF. The FEF RF center was estimated based on the endpoints of microstimulation-evoked saccades. The V4 RF was mapped using moving oriented bars in a different task. (B) Manipulating D1R-mediated FEF activity alters saccadic target selection. Monkeys performed a saccadic free-choice task, in which two targets appeared and the monkey could choose to saccade to either target. The two targets appeared at slightly different times (the temporal onset asynchrony), and the monkey's tendency to choose one target depended on the relative time of appearance, as illustrated by the likelihood of choosing the target in the FEF RF as the temporal onset asynchrony varied within a single experimental session (black curve). D1R antagonist administration biases the monkey toward choosing the RF target, as indicated by the leftward shift in the choice probability plot (red). A D2R agonist also biased target choice toward the FEF RF (not shown). (C) Manipulating D1R-mediated FEF activity enhances visual representations in area V4, but D2R-mediated activity does not. Administering a D1R antagonist in FEF caused an increase in orientation selectivity, increase in response magnitude, and decrease in response variability at overlapping V4 sites (orange bars); no effect was seen for non-overlapping V4 sites or saline infusions (not shown). These changes in V4 responses with FEF D1R manipulation mimic those seen during covert attention. Administering a D2R agonist in FEF (purple bars) did not alter V4 response magnitude, variability, or selectivity, despite producing a similar behavioral bias in the monkey's tendency to choose the RF target, resembling D1R effects shown in B. FEF inactivation with GABA agonist muscimol (blue bars) reduced the orientation selectivity of V4 responses, without altering their average magnitude or variability. (D) A schematized model of how D1R-mediated FEF activity could selectively modulate visual cortical responses, for example in area V4 (Noudoost and Moore, 2011b). Shown are two adjacent cortical columns within the FEF, each one representing different retinotopic parts of space. For simplicity, only a couple of pyramidal neurons and one GABA-ergic inhibitory neuron are shown, and only in the supragranular and infragranular layers, where the majority of D1Rs and D2Rs are located. Bold lines indicate pathways driven by a saccade to a target in the RF of the neurons in the left column (red box). D1Rs (red) are located in both the supragranular and infragranular layers; the supragranular layers are where neurons with feedback projections to V4 are located. FEF neurons within the infragranular layers, where D2Rs (blue) are primarily localized, project to the superior colliculus. The colliculus and V4 connections are to the corresponding retinotopically organized saccadic vectors and visual receptive fields. The VTA has diffuse dopaminergic inputs to the FEF. Persistent activity may depend on dopaminergic shaping of reciprocal excitatory connections between pyramidal neurons within the same columns, as well as cross-columnar inhibition. Manipulating D1R-mediated activity may strengthen the reciprocal connections between supragranular FEF neurons and V4 neurons, increasing the gain of visual signals in the retinotopically corresponding visual space. Both D1R and D2R manipulations bias saccadic target selection, possibly via infragranular outputs to SC. This model provides a basis for the increase in target selection by both D1 and D2 mediated activity, while only D1-mediated activity modulates V4 responses. See Clark and Noudoost (2014) for neurophysiological evidence supporting the model.

(2014). The authors also recorded V4 responses before and after manipulating dopamine signaling within FEF. They found that the D1R antagonist enhanced visual responses in V4, reproducing several of the signatures of covert attention (Fig. 3C). Blocking D1Rs in FEF increased the magnitude and stimulus selectivity of the corresponding V4 site, while simultaneously reducing the trial to trial variability of neuronal responses; each of these effects has previously been reported as a correlate of covert attention in neurophysiological studies (McAdams and Maunsell, 2000; Mitchell et al., 2007; Moran and Desimone, 1985; Reynolds et al., 2000). However only the D1R antagonist, and not the D2R agonist, produced the V4 response enhancements resembling the effects of attention (Noudoost and Moore, 2011a). The authors suggest that this dissociation reflects differential expression of the two receptor classes within PFC: D1Rs are expressed in both the infragranular and supragranular layers of PFC, while D2R expression is primarily infragranular (Lidow et al., 1991; Goldman-Rakic et al., 1992). The infragranular layers project to the superior colliculus and other oculomotor structures, while the supragranular layers project to visual areas including V4—thus the differential expression of D1Rs and D2Rs in these layers could account for the differing effects of signaling through these receptors on saccade targeting vs. visual modulation (Noudoost and Moore, 2011b) (Fig. 3D).

The basis for the differential effects of D1R and D2R manipulation was more rigorously examined using a biologically plausible cortical network model (Soltani et al., 2013), which reproduced the target selection bias and other behavioral effects of D1R and D2R manipulation, specifically differential effects of D1R and D2Rs on the impact of reward history on choice. The D1R and D2R manipulations differently impacted the influence of choice history, and hence reward history, on subsequent target choices: the D1R antagonist decreased the likelihood of the monkey choosing the same target as on the previous trial, whereas the D2R

agonist increased the probability of the monkey repeating his previous choice. The results of the network simulation suggest that D1Rs bias target selection primarily via their effects on FEF's sensitivity to excitatory input and its own recurrent connectivity, while D2Rs modulate the excitability of FEF output neurons, presumably those projecting to oculomotor areas. The modeling results support multiple, dissociable dopaminergic mechanisms involved in visual target selection and suggest how reward modulates adaptive choice behavior via prefrontal dopamine signals.

In order to understand which FEF neurons are involved in the top-down control of attention, a study by Gregoriou et al. (2012) examined the relationship between FEF neurons' response properties, characterized during a memory-guided saccade task, and simultaneously recorded FEF and V4 responses during a covert attention task. First, they found that FEF neurons without any visual activity (movement neurons) were not modulated by attention, ruling them out as a source of attention-dependent changes elsewhere; this finding replicates previous findings that visual and not motor FEF neurons show attentional modulation (Thompson et al., 2005). Additionally, only the purely visual FEF neurons (not neurons with a mix of visual and movement activity) showed enhanced gamma-band synchronization with V4 during attention, suggesting a unique interaction between PFC visual neurons and visual cortex. While these results are highly suggestive, several considerations indicate the need for further study before the question of which FEF neurons modulate responses in visual cortex can be considered closed. First, it is not known whether such changes in gamma-band synchronization are necessary for an increased FEF firing rate to influence V4 visual responses. Second, describing neurons as purely visual based on the memory guided saccade task may be misleading: many neurons which demonstrate only visual responses during a memory-guided saccade task nevertheless show increased activity around the time of visually guided saccades toward their RF (Bruce and Goldberg, 1985), thus providing a means for 'visual' neurons to pass movement-related modulations to other areas; any attempt to categorize FEF responses into specific classes based on a single task may be too simplistic. Lastly, although FEF neurons have traditionally been categorized along the visuo-motor spectrum, this characterization omits one important property of FEF responses: sustained activity during a memory task. This delay activity varies from neuron to neuron irrespective of their visuo-motor categorization (Lawrence et al., 2005; Sommer and Wurtz, 2000), but is strongly related to attentional modulation during stimulus presentation (Armstrong et al., 2009), and is therefore a strong candidate for a response property which correlates with which FEF neurons mediate attentional changes in visual areas. The proven ability of D1Rs to selectively modulate the strength of sustained activity within PFC (Williams and Goldman-Rakic, 1995), perhaps via D1R modulation of recurrent activity (Gao et al., 2001; Soltani et al., 2013; Compte et al., 2000; Clark and Noudoost, 2014), combined with their effect on V4 representations, also suggest that sustained activity may be a key characteristic of prefrontal neurons responsible for modulating visual activity during covert attention.

5. Conclusion

Given that numerous signatures of covert attention have now been reported throughout the brain, it becomes increasingly important to understand which of these signatures are actually related to the behavioral benefits of attention, and which are merely epiphenomenal. One method for assessing this relationship between an attentional signature and behavior is to look for correlations between them (Table 1, Section 1.3). A critical component in identifying the areas and signaling changes which drive attention is to distinguish attention from visual or motor deficits; measuring cue benefits is therefore critical to determining the sources of attention, and forms a significant gap in the existing literature (Table 2, Sections 2.2 and 2.3). A complete picture of the mechanisms behind attention will require causally linking source areas (Section 3) to both the neuronal signatures of attention and its behavioral effects (Section 4). Although no single study has linked a source area to both behavioral and neural measures of attention, the combined results of multiple studies suggest a model in which FEF modulates the activity within posterior visual cortices and that some of these changes are causally related to the behavioral benefits of attention. Many questions remain to be answered before we completely understand the mechanisms behind this prefrontal control of visual attention; the most critical experiments will require causal manipulations of frontal areas while simultaneously measuring the behavioral and neuronal correlates of attention. The scarcity of these studies (Fig. 3) is due to their evident technical difficulty. The literature reviewed here suggests critical characteristics of future work, especially (1) the importance of having behavioral measures of attention that can be studied in conjunction with its neuronal signatures (Table 1); (2) the importance of unambiguously identifying attentional benefits or deficits via cue-dependency (Table 2), and (3) recommends using a combination of activation and inactivation studies to evaluate the causal role of areas in driving attentional modulation. The identification of a handful of candidate source areas and neural signatures, combined with ever more sophisticated behavioral methods, high throughput recording systems and improved methods of causality testing make it very likely that in the near future neurobiologists will be able to explain the chain of neuronal events giving rise to attention and its behavioral benefits.

Conflict of interest

RFS is an employee of Lumos Labs (Lumosity.com) at the time this manuscript was submitted and has stock options with the company.

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References

Akert, K., 1964. Comparative anatomy of frontal cortex and thalamo-cortical connections. In: Warren, J., Akerts, K. (Eds.), The Frontal Granular Cortex and Behavior. McGraw-Hill, New York, pp. 372–396.

Albano, J.E., Mishkin, M., Westbrook, L.E., Wurtz, R.H., 1982. Visuomotor deficits following ablation of monkey superior colliculus. J. Neurophys. 48 (2), 338–351.

Albrecht, D.G., 1995. Visual cortex neurons in monkey and cat: effect of contrast on the spatial and temporal phase transfer functions. Vis. Neurosci. 12 (6), 1191–1210.

Anderson, J.C., Kennedy, H., Martin, K.A.C., 2011. Pathways of attention: synaptic relationships of frontal eye field to V4, lateral intraparietal cortex, and area 46 in macaque monkey. J. Neurosci. 31 (30), 10872–10881.

Anderson, E.B., Mitchell, J.F., Reynolds, J.H., 2013. Attention-dependent reductions in burstiness and action-potential height in macaque area V4. Nat. Neurosci. 16 (8), 1125–1131.

Anton-Erxleben, K., Carrasco, M., 2013. Attentional enhancement of spatial resolution: linking behavioural and neurophysiological evidence. Nat. Rev. Neurosci. 14 (3), 188–200.

Anton-Erxleben, K., Stephan, V.M., Treue, S., 2009. Attention reshapes center-surround receptive field structure in macaque cortical area MT. Cereb. Cortex 19 (10), 2466–2478.

Armstrong, K.M., Moore, T., 2007. Rapid enhancement of visual cortical response discriminability by microstimulation of the frontal eye field. Proc. Natl. Acad. Sci. U. S. A. 104 (22), 9499–9504.

Armstrong, K.M., Fitzgerald, J.K., Moore, T., 2006. Changes in visual receptive fields with microstimulation of frontal cortex. Neuron 50 (5), 791–798.

Armstrong, K.M., Chang, M.H., Moore, T., 2009. Selection and maintenance of spatial information by frontal eye field neurons. J. Neurosci. 29 (50), 15621–15629.

Arnsten, A.F., Dudley, A., 2005. Methylphenidate improves prefrontal cortical cognitive function through alpha2 adrenoceptor and dopamine D1 receptor actions: relevance to therapeutic effects in attention deficit hyperactivity disorder. Behav. Brain Funct. 1 (1), 2.

Awh, E., Jonides, J., Reuter-Lorenz, P.A., 1998. Rehearsal in spatial working memory. J. Exp. Psychol. Hum. Percept. Perform. 24 (3), 780–790.

Awh, E., Anllo-Vento, L., Hillyard, S.A., 2000. The role of spatial selective attention in working memory for locations: evidence from event-related potentials. J. Cogn. Neurosci. 12 (5), 840–847.

Awh, E., Armstrong, K.M., Moore, T., 2006. Visual and oculomotor selection: links, causes and implications for spatial attention. Trends Cogn. Sci. 10 (3), 124–130.

Balan, P.F., Gottlieb, J., 2009. Functional significance of nonspatial information in monkey lateral intraparietal area. J. Neurosci. 29 (25), 8166–8176.

Barceló, F., Suwazono, S., Knight, R.T., 2000. Prefrontal modulation of visual processing in humans. Nat. Neurosci. 3 (4), 399–403.

Bastos, A.M., Vezoli, J., Bosman, C.A., Schoffelen, J.-M., Oostenveld, R., Dowdall, J.R., De Weerd, P., Kennedy, H., Fries, P., 2015. Visual areas exert feedforward and feedback influences through distinct frequency channels. Neuron 85 (2), 390–401.

Beckstead, R.M., 1979. Convergent prefrontal and nigral projections to the striatum of the rat. Neurosci. Lett. 12 (1), 59–64.

Bell, A.H., Meredith, M.A., Van Opstal, A.J., Munoz, D.P., 2006. Stimulus intensity modifies saccadic reaction time and visual response latency in the superior colliculus. Exp. Brain Res. 174 (1), 53–59.

Bender, D.B., Youakim, M., 2001. Effect of attentive fixation in macaque thalamus and cortex. J. Neurophys. 85 (1), 219–234.

Berman, R.A., Joiner, W.M., Cavanaugh, J., Wurtz, R.H., 2009. Modulation of presaccadic activity in the frontal eye field by the superior colliculus. J. Neurophys. 101 (6), 2934–2942.

Bisley, J.W., Goldberg, M.E., 2003. Neuronal activity in the lateral intraparietal area and spatial attention. Science 299 (5603), 81–86.

Bisley, J.W., Goldberg, M.E., 2006. Neural correlates of attention and distractibility in the lateral intraparietal area. J. Neurophys. 95 (3), 1696–1717.

Bisley, J.W., Goldberg, M.E., 2010. Attention, intention, and priority in the parietal lobe. Annu. Rev. Neurosci. 33, 1–21.

Blanke, O., Spinelli, L., Thut, G., Michel, C.M., Perrig, S., Landis, T., Seeck, M., 2000. Location of the human frontal eye field as defined by electrical cortical stimulation: anatomical, functional and electrophysiological characteristics. Neuroreport 11 (9), 1907–1913.

Boehler, C.N., Tsotsos, J.K., Schoenfeld, M.A., Heinze, H.-J., Hopf, J.-M., 2009. The center-surround profile of the focus of attention arises from recurrent processing in visual cortex. Cereb. Cortex 19 (4), 982–991.

Bon, L., Lucchetti, C., 1997. Attention-related neurons in the supplementary eye field of the macaque monkey. Exp. Brain Res. 113 (1), 180–185.

Bosman, C.A., Schoffelen, J.-M., Brunet, N., Oostenveld, R., Bastos, A.M., Womelsdorf, T., Rubehn, B., Stieglitz, T., De Weerd, P., Fries, P., 2012. Attentional stimulus selection through selective synchronization between monkey visual areas. Neuron 75 (5), 875–888.

Boudreau, C.E., Williford, T.H., Maunsell, J.H.R., 2006. Effects of task difficulty and target likelihood in area V4 of macaque monkeys. J. Neurophys. 96 (5), 2377–2387.

Bressler, S.L., Richter, C.G., 2014. Interareal oscillatory synchronization in top-down neocortical processing. Curr. Opin. Neurobiol. 31C, 62–66.

Bressler, S.L., Tang, W., Sylvester, C.M., Shulman, G.L., Corbetta, M., 2008. Top-down control of human visual cortex by frontal and parietal cortex in anticipatory visual spatial attention. J. Neurosci. 28 (40), 10056–10061.

Briggs, F., Mangun, G.R., Usrey, W.M., 2013. Attention enhances synaptic efficacy and the signal-to-noise ratio in neural circuits. Nature 499 (7459), 476–480.

Brodmann, 1909. Vergleichende Lokalisationslehre der Grosshirnrinde. In: Prinzipien dargestellt auf Grund des ZellenbauesBarth, Leipzig.

Brozzoli, C., Demattè, M.L., Pavani, F., Frassinetti, F., Farnè, A., 2006. Neglect and extinction: within and between sensory modalities. Restor. Neurol. Neurosci. 24 (4–6), 217–232.

Bruce, C.J., Goldberg, M.E., 1985. Primate frontal eye fields. I. Single neurons discharging before saccades. J. Neurophys. 53 (3), 603–635.

Bruce, C.J., Goldberg, M.E., Bushnell, M.C., Stanton, G.B., 1985. Primate frontal eye fields. II. Physiological and anatomical correlates of electrically evoked eye movements. J. Neurophys. 54 (3), 714–734.

Buffalo, E.A., Bertini, G., Ungerleider, L.G., Desimone, R., 2005. Impaired filtering of distracter stimuli by TE neurons following V4 and TEO lesions in macaques. Cereb. Cortex 15 (2), 141–151.

Buffalo, E.A., Fries, P., Landman, R., Liang, H., Desimone, R., 2010. A backward progression of attentional effects in the ventral stream. Proc. Natl. Acad. Sci. U. S. A. 107 (1), 361–365.

Buffalo, E.A., Fries, P., Landman, R., Buschman, T.J., Desimone, R., 2011. Laminar differences in gamma and alpha coherence in the ventral stream. Proc. Natl. Acad. Sci. U. S. A. 108 (27), 11262–11267.

Buschman, T.J., Miller, E.K., 2007. Top-down versus bottom-up control of attention in the prefrontal and posterior parietal cortices. Science 315 (5820), 1860–1862.

Buschman, T.J., Miller, E.K., 2009. Serial, covert shifts of attention during visual search are reflected by the frontal eye fields and correlated with population oscillations. Neuron 63 (3), 386–396.

Cardin, J.A., Carlén, M., Meletis, K., Knoblich, U., Zhang, F., Deisseroth, K., Tsai, L.-H., Moore, C.I., 2009. Driving fast-spiking cells induces gamma rhythm and controls sensory responses. Nature 459 (7247), 663–667.

Carrasco, M., 2011. Visual attention: the past 25 years. Vis. Res. 51 (13), 1484–1525.

Carrasco, M., Ling, S., Read, S., 2004. Attention alters appearance. Nat. Neurosci. 7 (3), 308–313.

Cattaneo, A., Maffei, L., Morrone, C., 1981. Patterns in the discharge of simple and complex visual cortical cells. Proc. R. Soc. B: Biol. Sci. 212 (1188), 279–297.

Cavanaugh, J., Alvarez, B.D., Wurtz, R.H., 2006. Enhanced performance with brain stimulation: attentional shift or visual cue? J. Neurosci. 26 (44), 11347–11358.

Chalk, M., Herrero, J.L., Gieselmann, M.A., Delicato, L.S., Gotthardt, S., Thiele, A., 2010. Attention reduces stimulus-driven gamma frequency oscillations and spike field coherence in V1. Neuron 66 (1), 114–125.

Churchland, M.M., Cunningham, J.P., Kaufman, M.T., Foster, J.D., Nuyujukian, P., Ryu, S.I., Shenoy, K.V., 2012. Neural population dynamics during reaching. Nature 487 (7405), 51–56.

Clark, K.L., Noudoost, B., 2014. The role of prefrontal catecholamines in attention and working memory. Front. Neurosci. 8, 33.

Cohen, M.R., Kohn, A., 2011. Measuring and interpreting neuronal correlations. Nat. Neurosci. 14 (7), 811–819.

Cohen, M.R., Maunsell, J.H.R., 2009. Attention improves performance primarily by reducing inter-neuronal correlations. Nat. Neurosci. 12 (12), 1594–1600.

Cohen, M.R., Newsome, W.T., 2008. Context-dependent changes in functional circuitry in visual area MT. Neuron 60 (1), 162–173.

Colby, C.L., Duhamel, J.-R., Goldberg, M.E., 1996. Visual, presaccadic, and cognitive activation of single neurons in monkey lateral intraparietal area. J. Neurophys. 76 (5), 2841–2852.

Collin, N.G., Cowey, A., Latto, R., Marzi, C., 1982. The role of frontal eye-fields and superior colliculi in visual search and non-visual search in rhesus monkeys. Behav. Brain Res. 4 (2), 177–193.

Compte, A., Brunet, N., Goldman-Rakic, P.S., Wang, X.J., 2000. Synaptic mechanisms and network dynamics underlying spatial working memory in a cortical network model. Cereb. Cortex 10 (9), 910–923.

Condé, F., Audinat, E., Maire-Lepoivre, E., Crépel, F., 1990. Afferent connections of the medial frontal cortex of the rat. A study using retrograde transport of fluorescent dyes. I. Thalamic afferents. Brain Res. Bull. 24 (3), 341–354.

Connor, C.E., Gallant, J.L., Preddie, D.C., Van Essen, D.C., 1996. Responses in area V4 depend on the spatial relationship between stimulus and attention. J. Neurophys. 75 (3), 1306–1308.

Connor, C.E., Preddie, D.C., Gallant, J.L., Van Essen, D.C., 1997. Spatial attention effects in macaque area V4. J. Neurosci. 17 (9), 3201–3214.

Corbetta, M., Shulman, G.L., 2011. Spatial neglect and attention networks. Annu. Rev. Neurosci. 34, 569–599.

Cowey, A., Stoerig, P., 1995. Blindsight in monkeys. Nature 373 (6511), 247–249.

Cowey, A., Stoerig, P., 1997. Visual detection in monkeys with blindsight. Neuropsychologia 35 (7), 929–939.

Cowey, A., Stoerig, P., 2004. Stimulus cueing in blindsight. Prog. Brain Res. 144, 261–277.

Craighero, L., Nascimben, M., Fadiga, L., 2004. Eye position affects orienting of visuospatial attention. Curr. Biol. 14 (4), 331–333.

Crowne, D., Mah, L., 1998. A comparison of hemispatial neglect from posterior parietal and periarcuate lesions in the monkey. Psychobiology 26 (2), 103–108.

Crowne, D.P., Yeo, C.H., Russell, I.S., 1981. The effects of unilateral frontal eye field lesions in the monkey: visual-motor guidance and avoidance behaviour. Behav. Brain Res. 2 (2), 165–187.

Cutrell, E.B., Marrocco, R.T., 2002. Electrical microstimulation of primate posterior parietal cortex initiates orienting and alerting components of covert attention. Exp. Brain Res. 144 (1), 103–113.

De Haan, B., Karnath, H.-O., Driver, J., 2012. Mechanisms and anatomy of unilateral extinction after brain injury. Neuropsychologia 50 (6), 1045–1053.

De Weerd, P., Desimone, R., Ungerleider, L.G., 1996. Cue-dependent deficits in grating orientation discrimination after V4 lesions in macaques. Vis. Neurosci. 13 (03), 529.

De Weerd, P., Peralta, M.R., Desimone, R., Ungerleider, L.G., 1999. Loss of attentional stimulus selection after extrastriate cortical lesions in macaques. Nat. Neurosci. 2 (8), 753–758.

De Weerd, P., Desimone, R., Ungerleider, L.G., 2003. Generalized deficits in visual selective attention after V4 and TEO lesions in macaques. Eur. J. Neurosci. 18 (6), 1671–1691.

Desimone, R., Duncan, J., 1995. Neural mechanisms of selective visual attention. Annu. Rev. Neurosci. 18, 193–222.

Desimone, R., Wessinger, M., Thomas, L., Schneider, W., 1990. Attentional control of visual perception: cortical and subcortical mechanisms. Cold Spring Harb. Symp. Quant. Biol. 55, 963–971.

Deuel, R.K., Regan, D.J., 1985. Parietal hemineglect and motor deficits in the monkey. Neuropsychologia 23 (3), 305–314.

Dias, E.C., Segraves, M.A., 1999. Muscimol-induced inactivation of monkey frontal eye field: effects on visually and memory-guided saccades. J. Neurophys. 81 (5), 2191–2214.

Dias, E.C., Kiesau, M., Segraves, M.A., 1995. Acute activation and inactivation of macaque frontal eye field with GABA-related drugs. J. Neurophys. 74 (6), 2744–2748.

Dorris, M.C., Munoz, D.P., 1998. Saccadic probability influences motor preparation signals and time to saccadic initiation. J. Neurosci. 18 (17), 7015–7026.

Ekstrom, L.B., Roelfsema, P.R., Arsenault, J.T., Bonmassar, G., Vanduffel, W., 2008. Bottom-up dependent gating of frontal signals in early visual cortex. Science 321 (5887), 414–417.

Ekstrom, L.B., Roelfsema, P.R., Arsenault, J.T., Kolster, H., Vanduffel, W., 2009. Modulation of the contrast response function by electrical microstimulation of the macaque frontal eye field. J. Neurosci. 29 (34), 10683–10694.

Erlich, J.C., Bialek, M., Brody, C.D., 2011. A cortical substrate for memory-guided orienting in the rat. Neuron 72 (2), 330–343.

Everling, S., Tinsley, C.J., Gaffan, D., Duncan, J., 2002. Filtering of neural signals by focused attention in the monkey prefrontal cortex. Nat. Neurosci. 5 (7), 671–676.

Felleman, D.J., Van Essen, D.C., 1991. Distributed hierarchical processing in the primate cerebral cortex. Cereb. Cortex 1 (1), 1–47.

Ferrier, D., 1890. Cereb Localisation. Smith, Elder and Co., London.

Fries, W., 1984. Cortical projections to the superior colliculus in the macaque monkey: a retrograde study using horseradish peroxidase. J. Comp. Neurol. 230 (1), 55–76.

Fries, P., 2005. A mechanism for cognitive dynamics: neuronal communication through neuronal coherence. Trends Cogn. Sci. 9 (10), 474–480.

Fries, P., Reynolds, J.H., Rorie, A.E., Desimone, R., 2001. Modulation of oscillatory neuronal synchronization by selective visual attention. Science 291 (5508), 1560–1563.

Fries, P., Womelsdorf, T., Oostenveld, R., Desimone, R., 2008. The effects of visual stimulation and selective visual attention on rhythmic neuronal synchronization in macaque area V4. J. Neurosci. 28 (18), 4823–4835.

Funahashi, S., Bruce, C.J., Goldman-Rakic, P.S., 1989. Mnemonic coding of visual space in the monkey's dorsolateral prefrontal cortex. J. Neurophys. 61 (2), 331–349.

Funahashi, S., Bruce, C.J., Goldman-Rakic, P.S., 1993. Dorsolateral prefrontal lesions and oculomotor delayed-response performance: evidence for mnemonic scotomas. J. Neurosci. 13 (4), 1479–1497.

Fuster, J.M., Bauer, R.H., 1974. Visual short-term memory deficit from hypothermia of frontal cortex. Brain Res. 81 (3), 393–400.

Fuster, J.M., Bauer, R.H., Jervey, J.P., 1985. Functional interactions between inferotemporal and prefrontal cortex in a cognitive task. Brain Res. 330 (2), 299–307.

Galashan, F.O., Saßen, H.C., Kreiter, A.K., Wegener, D., 2013. Monkey area MT latencies to speed changes depend on attention and correlate with behavioral reaction times. Neuron 78 (4), 740–750.

Gao, W.-J., Krimer, L.S., Goldman-Rakic, P.S., 2001. Presynaptic regulation of recurrent excitation by D1 receptors in prefrontal circuits. Proc. Natl. Acad. Sci. U. S. A. 98 (1), 295–300.

Glowinski, J., Tassin, J., Thierry, A., 1984. The mesocortico-prefrontal dopaminergic neurons. Trends Neurosci. 7, 415–418.

Gnadt, J.W., Andersen, R.A., 1988. Memory related motor planning activity in posterior parietal cortex of macaque. Exp. Brain Res. 70 (1), 216–220.

Goldberg, M.E., Wurtz, R.H., 1972. Activity of superior colliculus in behaving monkey. II. Effect of attention on neuronal responses. J. Neurophys. 35 (4), 560–574.

Goldman-Rakic, P.S., 1995. Cellular basis of working memory. Neuron 14 (3), 477–485.

Goldman-Rakic, P.S., Lidow, M.S., Smiley, J.F., Williams, M.S., 1992. The anatomy of dopamine in monkey and human prefrontal cortex. J. Neural Trans. Suppl. 36, 163–177.

Gottlieb, J.P., Kusunoki, M., Goldberg, M.E., 1998. The representation of visual salience in monkey parietal cortex. Nature 391 (6666), 481–484.

Green, D., Swets, D., 1966. Signal Detection Theory and Psychophysics.

Gregoriou, G.G., Gotts, S.J., Zhou, H., Desimone, R., 2009a. High-frequency, long-range coupling between prefrontal and visual cortex during attention. Science 324 (5931), 1207–1210.

Gregoriou, G.G., Gotts, S.J., Zhou, H., Desimone, R., 2009b. Long-range neural coupling through synchronization with attention. Prog. Brain Res. 176, 35–45.

Gregoriou, G.G., Gotts, S.J., Desimone, R., 2012. Cell-type-specific synchronization of neural activity in FEF with V4 during attention. Neuron 73 (3), 581–594.

Gregoriou, G.G., Rossi, A.F., Ungerleider, L.G., Desimone, R., 2014. Lesions of prefrontal cortex reduce attentional modulation of neuronal responses and synchrony in V4. Nat. Neurosci. 17 (7), 1003–1011.

Grewe, J., Weckström, M., Egelhaaf, M., Warzecha, A.-K., 2007. Information and discriminability as measures of reliability of sensory coding. PLoS ONE 2 (12), e1328.

Guillery, R.W., Harting, J.K., 2003. Structure and connections of the thalamic reticular nucleus: advancing views over half a century. J. Comp. Neurol. 463 (4), 360–371.

Hanes, D.P., Wurtz, R.H., 2001. Interaction of the frontal eye field and superior colliculus for saccade generation. J. Neurophys. 85 (2), 804–815.

Heilman, K.M., Pandya, D.N., Geschwind, N., 1970. Trimodal inattention following parietal lobe ablations. Trans. Am. Neurol. Assoc. 95, 259–261.

Heilman, K.M., Valenstein, E., Watson, R.T., 2000. Neglect and related disorders. Semin. Neurol. 20 (4), 463–470.

Herrero, J.L., Roberts, M.J., Delicato, L.S., Gieselmann, M.A., Dayan, P., Thiele, A., 2008. Acetylcholine contributes through muscarinic receptors to attentional modulation in V1. Nature 454 (7208), 1110–1114.

Hikosaka, O., Wurtz, R.H., 1985. Modification of saccadic eye movements by GABA-related substances. I. Effect of muscimol and bicuculline in monkey superior colliculus. J. Neurophys. 53 (1), 266–291.

Hikosaka, O., Wurtz, R.H., 1986. Saccadic eye movements following injection of lidocaine into the superior colliculus. Exp. Brain Res. 61 (3), 531–539.

Hoffman, J.E., Subramaniam, B., 1995. The role of visual attention in saccadic eye movements. Percept. Psychophys. 57 (6), 787–795.

Hopf, J.-M., Boehler, C.N., Luck, S.J., Tsotsos, J.K., Heinze, H.-J., Schoenfeld, M.A., 2006. Direct neurophysiological evidence for spatial suppression surrounding the focus of attention in vision. Proc. Natl. Acad. Sci. U. S. A. 103 (4), 1053–1058.

Iba, M., Sawaguchi, T., 2003. Involvement of the dorsolateral prefrontal cortex of monkeys in visuospatial target selection. J. Neurophys. 89 (1), 587–599.

Ignashchenkova, A., Dicke, P.W., Haarmeier, T., Thier, P., 2004. Neuron-specific contribution of the superior colliculus to overt and covert shifts of attention. Nat. Neurosci. 7 (1), 56–64.

Ikkai, A., Curtis, C.E., 2011. Common neural mechanisms supporting spatial working memory, attention and motor intention. Neuropsychologia 49 (6), 1428–1434.

Jha, A.P., 2002. Tracking the time-course of attentional involvement in spatial working memory: an event-related potential investigation. Brain Res. Cogn. Brain Res. 15 (1), 61–69.

Kaping, D., Vinck, M., Hutchison, R.M., Everling, S., Womelsdorf, T., 2011. Specific contributions of ventromedial, anterior cingulate, and lateral prefrontal cortex for attentional selection and stimulus valuation. PLoS Biol. 9 (12), e1001224.

Katsuki, F., Constantinidis, C., 2012. Early involvement of prefrontal cortex in visual bottom-up attention. Nat. Neurosci. 15 (8), 1160–1166.

Keating, E.G., Gooley, S.G., 1988. Saccadic disorders caused by cooling the superior colliculus or the frontal eye field, or from combined lesions of both structures. Brain Res. 438 (1–2), 247–255.

Kennard, M., 1939. Alterations in response to visual stimuli following lesions of frontal lobe in monkeys. Arch. Neurol. Psychiatry 1153–1165.

Kermadi, I., Boussaoud, D., 1995. Role of the primate striatum in attention and sensorimotor processes: comparison with premotor cortex. Neuroreport 6 (8), 1177–1181.

Knight, R.T., Grabowecky, M.F., Scabini, D., 1995. Role of human prefrontal cortex in attention control. Adv. Neurol. 66, 21–34, discussion 34–36.

Knoblich, U., Siegle, J.H., Pritchett, D.L., Moore, C.I., 2010. What do we gain from gamma? Local dynamic gain modulation drives enhanced efficacy and efficiency of signal transmission. Front. Hum. Neurosci. 4, 185.

Knudsen, E.I., 2007. Fundamental components of attention. Annu. Rev. Neurosci. 30, 57–78.

Kodaka, Y., Mikami, A., Kubota, K., 1997. Neuronal activity in the frontal eye field of the monkey is modulated while attention is focused on to a stimulus in the peripheral visual field, irrespective of eye movement. Neurosci. Res. 28 (4), 291–298.

Krahe, R., Gabbiani, F., 2004. Burst firing in sensory systems. Nat. Rev. Neurosci. 5 (1), 13–23.

Kustov, A.A., Robinson, D., 1996. Shared neural control of attentional shifts and eye movements. Nature 384 (6604), 74–77.

Kusunoki, M., Goldberg, M.E., 2003. The time course of perisaccadic receptive field shifts in the lateral intraparietal area of the monkey. J. Neurophys. 89 (3), 1519–1527.

Latto, R., Cowey, A., 1971. Visual field defects after frontal eye-field lesions in monkeys. Brain Res. 30 (1), 1–24.

Lawrence, B.M., White, R.L., Snyder, L.H., 2005. Delay-period activity in visual, visuomovement, and movement neurons in the frontal eye field. J. Neurophys. 94 (2), 1498–1508.

Lebedev, M.A., Messinger, A., Kralik, J.D., Wise, S.P., 2004. Representation of attended versus remembered locations in prefrontal cortex. PLoS Biol. 2 (11), e365.

Lee, C., Rohrer, W.H., Sparks, D.L., 1988. Population coding of saccadic eye movements by neurons in the superior colliculus. Nature 332 (6162), 357–360.

Lee, H., Simpson, G.V., Logothetis, N.K., Rainer, G., 2005. Phase locking of single neuron activity to theta oscillations during working memory in monkey extrastriate visual cortex. Neuron 45 (1), 147–156.

Lee, J., Kim, H.R., Lee, C., 2010. Trial-to-trial variability of spike response of V1 and saccadic response time. J. Neurophys. 104 (5), 2556–2572.

Lee, J.H., Whittington, M.A., Kopell, N.J., 2013. Top-down beta rhythms support selective attention via interlaminar interaction: a model. PLoS Comput. Biol. 9 (8), e1003164.

Leonard, C.M., 1969. The prefrontal cortex of the rat. I. Cortical projection of the mediodorsal nucleus. II. Efferent connections. Brain Res. 12 (2), 321–343.

Li, C.S., Mazzoni, P., Andersen, R.A., 1999. Effect of reversible inactivation of macaque lateral intraparietal area on visual and memory saccades. J. Neurophys. 81 (4), 1827–1838.

Lidow, M.S., Goldman-Rakic, P.S., Gallager, D.W., Rakic, P., 1991. Distribution of dopaminergic receptors in the primate cereb cortex: quantitative autoradiographic analysis using [3H]raclopride, [3H]spiperone and [3H]SCH23390. Neuroscience 40 (3), 657–671.

Ling, S., Liu, T., Carrasco, M., 2009. How spatial and feature-based attention affect the gain and tuning of population responses. Vis. Res. 49 (10), 1194–1204.

Liu, Y., Yttri, E.A., Snyder, L.H., 2010. Intention and attention: different functional roles for LIPd and LIPv. Nat. Neurosci. 13 (4), 495–500.

Lovejoy, L.P., Krauzlis, R.J., 2010. Inactivation of primate superior colliculus impairs covert selection of signals for perceptual judgments. Nat. Neurosci. 13 (2), 261–266.

Lynch, J.C., McLaren, J.W., 1989. Deficits of visual attention and saccadic eye movements after lesions of parietooccipital cortex in monkeys. J. Neurophys. 61 (1), 74–90.
Mante, V., Sussillo, D., Shenoy, K.V., Newsome, W.T., 2013. Context-dependent computation by recurrent dynamics in prefrontal cortex. Nature 503 (7474), 78–84.
Markov, N.T., Ercsey-Ravasz, M.M., Ribeiro Gomes, A.R., Lamy, C., Magrou, L., Vezoli, J., Misery, P., Falchier, A., Quilodran, R., Gariel, M.A., Sallet, J., Gamanut, R., Huissoud, C., Clavagnier, S., Giroud, P., Sappey-Marinier, D., Barone, P., Dehay, C., Toroczkai, Z., Knoblauch, K., Van Essen, D.C., Kennedy, H., 2014. A weighted and directed interareal connectivity matrix for macaque cereb cortex. Cereb. Cortex 24 (1), 17–36.
Mazzoni, P., Bracewell, R.M., Barash, S., Andersen, R.A., 1996. Motor intention activity in the macaque's lateral intraparietal area. I. Dissociation of motor plan from sensory memory. J. Neurophys. 76 (3), 1439–1456.
McAdams, C.J., Maunsell, J.H., 1999. Effects of attention on orientation-tuning functions of single neurons in macaque cortical area V4. J. Neurosci. 19 (1), 431–441.
McAdams, C.J., Maunsell, J.H., 2000. Attention to both space and feature modulates neuronal responses in macaque area V4. J. Neurophys. 83 (3), 1751–1755.
McAdams, C.J., Reid, R.C., 2005. Attention modulates the responses of simple cells in monkey primary visual cortex. J. Neurosci. 25 (47), 11023–11033.
McAlonan, K., Cavanaugh, J., Wurtz, R.H., 2008. Guarding the gateway to cortex with attention in visual thalamus. Nature 456 (7220), 391–394.
McPeek, R.M., Keller, E.L., 2004. Deficits in saccade target selection after inactivation of superior colliculus. Nat. Neurosci. 7 (7), 757–763.
Messinger, A., Lebedev, M.A., Kralik, J.D., Wise, S.P., 2009. Multitasking of attention and memory functions in the primate prefrontal cortex. J. Neurosci. 29 (17), 5640–5653.
Mesulam, M.M., 1981. A cortical network for directed attention and unilateral neglect. Ann. Neurol. 10 (4), 309–325.
Miller, E.K., Cohen, J.D., 2001. An integrative theory of prefrontal cortex function. Annu. Rev. Neurosci. 24, 167–202.
Miller, B.T., D'Esposito, M., 2005. Searching for the top in top-down control. Neuron 48 (4), 535–538.
Milner, A.D., McIntosh, R.D., 2005. The neurological basis of visual neglect. Curr. Opin. Neurol. 18 (6), 748–753.
Mitchell, J.F., Sundberg, K.A., Reynolds, J.H., 2007. Differential attention-dependent response modulation across cell classes in macaque visual area V4. Neuron 55 (1), 131–141.
Mitchell, J.F., Sundberg, K.A., Reynolds, J.H., 2009. Spatial attention decorrelates intrinsic activity fluctuations in macaque area V4. Neuron 63 (6), 879–888.
Mohler, C.W., Wurtz, R.H., 1977. Role of striate cortex and superior colliculus in visual guidance of saccadic eye movements in monkeys. J. Neurophys. 40 (1), 74–94.
Monosov, I.E., Thompson, K.G., 2009. Frontal eye field activity enhances object identification during covert visual search. J. Neurophys. 102 (6), 3656–3672.
Monosov, I.E., Trageser, J.C., Thompson, K.G., 2008. Measurements of simultaneously recorded spiking activity and local field potentials suggest that spatial selection emerges in the frontal eye field. Neuron 57 (4), 614–625.
Monosov, I.E., Sheinberg, D.L., Thompson, K.G., 2011. The effects of prefrontal cortex inactivation on object responses of single neurons in the inferotemporal cortex during visual search. J. Neurosci. 31 (44), 15956–15961.
Monteon, J.A., Constantin, A.G., Wang, H., Martinez-Trujillo, J., Crawford, J.D., 2010. Electrical stimulation of the frontal eye fields in the head-free macaque evokes kinematically normal 3D gaze shifts. J. Neurophys. 104 (6), 3462–3475.
Moore, T., 1999. Shape representations and visual guidance of saccadic eye movements. Science 285 (5435), 1914–1917.
Moore, T., Armstrong, K.M., 2003. Selective gating of visual signals by microstimulation of frontal cortex. Nature 421 (January), 370L 373.
Moore, T., Chang, M.H., 2009. Presaccadic discrimination of receptive field stimuli by area V4 neurons. Vis. Res. 49 (10), 1227–1232.
Moore, T., Fallah, M., 2001. Control of eye movements and spatial attention. Proc. Natl. Acad. Sci. U. S. A. 98 (3), 1273–1276.
Moore, T., Fallah, M., 2004. Microstimulation of the frontal eye field and its effects on covert spatial attention. J. Neurophys. 91 (1), 152–162.
Moore, T., Rodman, H.R., Repp, A.B., Gross, C.G., 1995. Localization of visual stimuli after striate cortex damage in monkeys: parallels with human blindsight. Proc. Natl. Acad. Sci. U. S. A. 92 (18), 8215–8218.
Moore, T., Tolias, A.S., Schiller, P.H., 1998. Visual representations during saccadic eye movements. Proc. Natl. Acad. Sci. U. S. A. 95 (15), 8981–8984.
Moore, T., Armstrong, K.M., Fallah, M., 2003. Visuomotor origins of covert spatial attention. Neuron 40 (4), 671–683.
Moran, J., Desimone, R., 1985. Selective attention gates visual processing in the extrastriate cortex. Science 229 (4715), 782–784.
Motter, B.C., 1993. Focal attention produces spatially selective processing in visual cortical areas V1, V2, and V4 in the presence of competing stimuli. J. Neurophys. 70 (3), 909–919.
Müller, J.R., Philiastides, M.G., Newsome, W.T., 2005. Microstimulation of the superior colliculus focuses attention without moving the eyes. Proc. Natl. Acad. Sci. U. S. A. 102 (3), 524–529.
Newsome, W.T., Wurtz, R.H., Dürsteler, M.R., Mikami, A., 1985. Punctate chemical lesions of striate cortex in the macaque monkey: effect on visually guided saccades. Exp. Brain Res. 58 (2), 392–399.
Noudoost, B., Moore, T., 2011a. Control of visual cortical signals by prefrontal dopamine. Nature 474 (7351), 372–375.
Noudoost, B., Moore, T., 2011b. The role of neuromodulators in selective attention. Trends Cogn. Sci. 15 (12), 585–591.
Noudoost, B., Chang, M.H., Steinmetz, N.A., Moore, T., 2010. Top-down control of visual attention. Curr. Opin. Neurobiol. 20 (2), 183–190.
Noudoost, B., Clark, K.L., Moore, T., 2014. A distinct contribution of the frontal eye field to the visual representation of saccadic targets. J. Neurosci. 34 (10), 3687–3698.
Nummela, S.U., Krauzlis, R.J., 2010. Inactivation of primate superior colliculus biases target choice for smooth pursuit, saccades, and button press responses. J. Neurophys. 104 (3), 1538–1548.
Nummela, S.U., Krauzlis, R.J., 2011. Superior colliculus inactivation alters the weighted integration of visual stimuli. J. Neurosci. 31 (22), 8059–8066.
Ongür, D., Price, J.L., 2000. The organization of networks within the orbital and medial prefrontal cortex of rats, monkeys and humans. Cereb. Cortex 10 (3), 206–219.
Oram, M.W., Xiao, D., Dritschel, B., Payne, K.R., 2002. The temporal resolution of neural codes: does response latency have a unique role? Philos. Trans. R. Soc. Lond. B: Biol. Sci. 357 (1424), 987–1001.
Pandya, D.N., Yeterian, E.H., 1996. Comparison of prefrontal architecture and connections. Philos. Trans. R. Soc. Lond. B: Biol. Sci. 351 (1346), 1423–1432.
Pellegrino, G., di Wise, S.P., 1993. Effects of attention on visuomotor activity in the premotor and prefrontal cortex of a primate. Somatosens. Motor Res. 10, 245–262.
Petersen, S.E., Robinson, D., 1986. Damage to parietal cortex produces a similar deficit in man and monkey. Investig. Ophthalmol. Vis. Sci. 27 (3), 18.
Petersen, S.E., Robinson, D., Keys, W., 1985. Pulvinar nuclei of the behaving rhesus monkey: visual responses and their modulation. J. Neurophys. 54 (4), 867–886.
Petersen, S.E., Robinson, D., Morris, J.D., 1987. Contributions of the pulvinar to visual spatial attention. Neuropsychologia 25 (1A), 97L 105.
Peterson, M.S., Kramer, A.F., Irwin, D.E., 2004. Covert shifts of attention precede involuntary eye movements. Percept. Psychophys. 66 (3), 398–405.
Petrides, M., Pandya, D.N., 1999. Dorsolateral prefrontal cortex: comparative cytoarchitectonic analysis in the human and the macaque brain and corticocortical connection patterns. Eur. J. Neurosci. 11 (3), 1011–1036.
Petrides, M., Tomaiuolo, F., Yeterian, E.H., Pandya, D.N., 2012. The prefrontal cortex: comparative architectonic organization in the human and the macaque monkey brains. Cortex 48 (1), 46–57.
Pinsk, M.A., Doniger, G.M., Kastner, S., 2004. Push–pull mechanism of selective attention in human extrastriate cortex. J. Neurophys. 92 (1), 622–629.
Posner, M.I., 1980. Orienting of attention. Q. J. Exp. Psychol. 32 (1), 3–25.
Postle, B.R., Awh, E., Jonides, J., Smith, E.E., D'Esposito, M., 2004. The where and how of attention-based rehearsal in spatial working memory. Brain Res. Cogn. Brain Res. 20 (2), 194–205.
Preuss, T.M., 1995. Do rats have prefrontal cortex? The Rose–Woolsey–Akert program reconsidered. J. Cogn. Neurosci. 7 (1), 1–24.
Purushothaman, G., Marion, R., Li, K., Casagrande, V.A., 2012. Gating and control of primary visual cortex by pulvinar. Nat. Neurosci. 15 (6), 905–912.
Qi, X.-L., Katsuki, F., Meyer, T., Rawley, J.B., Zhou, X., Douglas, K.L., Constantinidis, C., 2010. Comparison of neural activity related to working memory in primate dorsolateral prefrontal and posterior parietal cortex. Front. Syst. Neurosci. 4, 12.
Raiguel, S.E., Xiao, D.K., Marcar, V.L., Orban, G.A., 1999. Response latency of macaque area MT/V5 neurons and its relationship to stimulus parameters. J. Neurophys. 82 (4), 1944–1956.
Raposo, D., Kaufman, M.T., Churchland, A.K., 2014. A category-free neural population supports evolving demands during decision-making. Nat. Neurosci. 17 (12), 1784–1792.
Reich, D.S., Mechler, F., Purpura, K.P., Victor, J.D., 2000. Interspike intervals, receptive fields, and information encoding in primary visual cortex. J. Neurosci. 20 (5), 1964–1974.
Reynolds, J.H., Heeger, D.J., 2009. The normalization model of attention. Neuron 61 (2), 168–185.
Reynolds, J.H., Chelazzi, L., Desimone, R., 1999. Competitive mechanisms subserve attention in macaque areas V2 and V4. J. Neurosci. 19 (5), 1736–1753.
Reynolds, J.H., Pasternak, T., Desimone, R., 2000. Attention increases sensitivity of V4 neurons. Neuron 26 (3), 703–714.
Ribot, T., 1890. The Psychology of Attention. Open Court Publishing, Chicago.
Riddoch, M.J., Rappaport, S.J., Humphreys, G.W., 2009. Extinction: a window into attentional competition. Prog. Brain Res. 176, 149–159.
Rigotti, M., Barak, O., Warden, M.R., Wang, X.-J., Daw, N.D., Miller, E.K., Fusi, S., 2013. The importance of mixed selectivity in complex cognitive tasks. Nature 497 (7451), 585–590.
Rizzolatti, G., Matelli, M., Pavesi, G., 1983. Deficits in attention and movement following the removal of postarcuate (area 6) and prearcuate (area 8) cortex in macaque monkeys. Brain 106 (Pt 3), 655–673.
Rizzolatti, G., Riggio, L., Dascola, I., Umiltá, C., 1987. Reorienting attention across the horizontal and vertical meridians: evidence in favor of a premotor theory of attention. Neuropsychologia 25 (1A), 31L 40.
Robinson, D.A., 1972. Eye movements evoked by collicular stimulation in the alert monkey. Vis. Res. 12 (11), 1795–1808.
Robinson, D.A., Fuchs, A.F., 1969. Eye movements evoked by stimulation of frontal eye fields. J. Neurophys. 32 (5), 637–648.
Robinson, D., Kertzman, C., 1995. Covert orienting of attention in macaques. III. Contributions of the superior colliculus. J. Neurophys. 74 (2), 713–721.
Rose, J.E., Woolsey, C.N., 1948. The orbitofrontal cortex and its connections with the mediodorsal nucleus in rabbit, sheep and cat. Res. Publ. Assoc. Res. Nerv. Ment. Dis. 27 (1), 210–232.

Rossi, A.F., Bichot, N.P., Desimone, R., Ungerleider, L.G., 2007. Top down attentional deficits in macaques with lesions of lateral prefrontal cortex. J. Neurosci. 27 (42), 11306–11314.
Ruff, D.A., Cohen, M.R., 2014. Attention can either increase or decrease spike count correlations in visual cortex. Nat. Neurosci. 17 (11), 1591–1597.
Ruff, C.C., Blankenburg, F., Bjoertomt, O., Bestmann, S., Freeman, E., Haynes, J.-D., Rees, G., Josephs, O., Deichmann, R., Driver, J., 2006. Concurrent TMS-fMRI and psychophysics reveal frontal influences on human retinotopic visual cortex. Curr. Biol. 16 (15), 1479–1488.
Saalmann, Y.B., Pigarev, I.N., Vidyasagar, T.R., 2007. Neural mechanisms of visual attention: how top-down feedback highlights relevant locations. Science 316 (5831), 1612–1615.
Saalmann, Y.B., Pinsk, M.A., Wang, L., Li, X., Kastner, S., 2012. The pulvinar regulates information transmission between cortical areas based on attention demands. Science 337 (6095), 753–756.
Sagvolden, T., Russell, V.A., Aase, H., Johansen, E.B., Farshbaf, M., 2005. Rodent models of attention-deficit/hyperactivity disorder. Biol. Psychiatry 57 (11), 1239–1247.
Salinas, E., Sejnowski, T.J., 2000. Impact of correlated synaptic input on output firing rate and variability in simple neuronal models. J. Neurosci. 20 (16), 6193–6209.
Salinas, E., Sejnowski, T.J., 2001. Correlated neuronal activity and the flow of neural information. Nat. Rev. Neurosci. 2 (8), 539–550.
Samonds, J.M., Bonds, A.B., 2004. From another angle: differences in cortical coding between fine and coarse discrimination of orientation. J. Neurophys. 91 (3), 1193–1202.
Sato, T., Murthy, A., Thompson, K.G., Schall, J.D., Hall, W., 2001. Search efficiency but not response interference affects visual selection in frontal eye field. Neuron 30, 583–591.
Schafer, R.J., Moore, T., 2007. Attention governs action in the primate frontal eye field. Neuron 56 (3), 541–551.
Schafer, R.J., Moore, T., 2011. Selective attention from voluntary control of neurons in prefrontal cortex. Science 332 (6037), 1568–1571.
Schall, J.D., Hanes, D.P., 1993. Neural basis of saccade target selection in frontal eye field during visual search. Nature 366 (6454), 467–469.
Schall, J.D., Morel, A., King, D.J., Bullier, J., 1995. Topography of visual cortex connections with frontal eye field in macaque: convergence and segregation of processing streams. J. Neurosci. 15 (6), 4464–4487.
Schiller, P.H., 1993. The effects of V4 and middle temporal (MT) area lesions on visual performance in the rhesus monkey. Vis. Neurosci. 10 (04), 717.
Schiller, P.H., Chou, I.H., 1998. The effects of frontal eye field and dorsomedial frontal cortex lesions on visually guided eye movements. Nat. Neurosci. 1 (3), 248–253.
Schiller, P.H., Chou, I., 2000. The effects of anterior arcuate and dorsomedial frontal cortex lesions on visually guided eye movements. 2. Paired and multiple targets. Vis. Res. 40 (10–12), 1627–1638.
Schiller, P.H., Lee, K., 1991. The role of the primate extrastriate area V4 in vision. Science 251 (4998), 1251–1253.
Schiller, P.H., Tehovnik, E.J., 2003. Cortical inhibitory circuits in eye-movement generation. Eur. J. Neurosci. 18 (11), 3127–3133.
Schiller, P.H., True, S.D., Conway, J.L., 1980. Deficits in eye movements following frontal eye-field and superior colliculus ablations. J. Neurophys. 44 (6), 1175–1189.
Schiller, P.H., Logothetis, N.K., Charles, E.R., 1990. Functions of the colour-opponent and broad-band channels of the visual system. Nature 343 (6253), 68–70.
Schmid, M.C., Mrowka, S.W., Turchi, J., Saunders, R.C., Wilke, M., Peters, A.J., Ye, F.Q., Leopold, D.A., 2010. Blindsight depends on the lateral geniculate nucleus. Nature 466 (7304), 373–377.
Seriès, P., Latham, P.E., Pouget, A., 2004. Tuning curve sharpening for orientation selectivity: coding efficiency and the impact of correlations. Nat. Neurosci. 7 (10), 1129–1135.
Sharma, J., Sugihara, H., Katz, Y., Schummers, J., Tenenbaum, J., Sur, M., 2014. Spatial attention and temporal expectation under timed uncertainty predictably modulate neuronal responses in monkey V1. Cereb. Cortex, first published online May 16, 2014.
Sheinberg, D.L., Logothetis, N.K., 2001. Noticing familiar objects in real world scenes: the role of temporal cortical neurons in natural vision. J. Neurosci. 21 (4), 1340–1350.
Sheliga, B.M., Riggio, L., Rizzolatti, G., 1994. Orienting of attention and eye movements. Exp. Brain Res. 98 (3), 507–522.
Sheliga, B.M., Riggio, L., Craighero, L., Rizzolatti, G., 1995. Spatial attention-determined modifications in saccade trajectories. Neuroreport 6 (3), 585–588.
Sherman, S.M., 2001. Tonic and burst firing: dual modes of thalamocortical relay. Trends Neurosci. 24 (2), 122–126.
Shih, J.Y., Atencio, C.A., Schreiner, C.E., 2011. Improved stimulus representation by short interspike intervals in primary auditory cortex. J. Neurophys. 105 (4), 1908–1917.
Shipp, S., 2003. The functional logic of cortico-pulvinar connections. Philos. Trans. R. Soc. Lond. B: Biol. Sci. 358 (1438), 1605–1624.
Siegel, M., Warden, M.R., Miller, E.K., 2009. Phase-dependent neuronal coding of objects in short-term memory. Proc. Natl. Acad. Sci. U. S. A. 106 (50), 21341–21346.
Sinnamon, H.M., Galer, B.S., 1984. Head movements elicited by electrical stimulation of the anteromedial cortex of the rat. Physiol. Behav. 33 (2), 185–190.
Smolyanskaya, A., Born, R.T., 2012. Neuroscience: attention is more than meets the eye. Nature 489 (7416), 371–372.
Smyth, M.M., 1996. Interference with rehearsal in spatial working memory in the absence of eye movements. Q. J. Exp. Psychol. A: Hum. Exp. Psychol. 49 (4), 940–949.
Soltani, A., Noudoost, B., Moore, T., 2013. Dissociable dopaminergic control of saccadic target selection and its implications for reward modulation. Proc. Natl. Acad. Sci. U. S. A. 110 (9), 3579–3584.
Sommer, M.A., Tehovnik, E.J., 1997. Reversible inactivation of macaque frontal eye field. Exp. Brain Res. 116 (2), 229–249.
Sommer, M.A., Wurtz, R.H., 2000. Composition and topographic organization of signals sent from the frontal eye field to the superior colliculus. J. Neurophys. 83 (4), 1979–2001.
Song, J.-H., Rafal, R.D., McPeek, R.M., 2011. Deficits in reach target selection during inactivation of the midbrain superior colliculus. Proc. Natl. Acad. Sci. U. S. A. 108 (51), E1433–E1440.
Spitzer, H., Desimone, R., Moran, J., 1988. Increased attention enhances both behavioral and neuronal performance. Science 240 (4850), 338–340.
Squire, R.F., Noudoost, B., Schafer, R.J., Moore, T., 2013. Prefrontal contributions to visual selective attention. Annu. Rev. Neurosci. 36 (1), 451–466.
Sridharan, D., Steinmetz, N.A., Moore, T., Knudsen, E.I., 2014. Distinguishing bias from sensitivity effects in multialternative detection tasks. J. Vis. 14 (9) .
Stanton, G.B., Goldberg, M.E., Bruce, C.J., 1988. Frontal eye field efferents in the macaque monkey. II. Topography of terminal fields in midbrain and pons. J. Comp. Neurol. 271 (4), 493–506.
Stanton, G.B., Deng, S.Y., Goldberg, M.E., McMullen, N.T., 1989. Cytoarchitectural characteristic of the frontal eye fields in macaque monkeys. J. Comp. Neurol. 282 (3), 415–427.
Stanton, G.B., Bruce, C.J., Goldberg, M.E., 1993. Topography of projections to the frontal lobe from the macaque frontal eye fields. J. Comp. Neurol. 330 (2), 286–301.
Stanton, G.B., Bruce, C.J., Goldberg, M.E., 1995. Topography of projections to posterior cortical areas from the macaque frontal eye fields. J. Comp. Neurol. 353 (2), 291–305.
Steinmetz, M.A., Constantinidis, C., 1995. Neurophysiological evidence for a role of posterior parietal cortex in redirecting visual attention. Cereb. Cortex 5 (5), 448–456.
Steinmetz, N.A., Moore, T., 2010. Changes in the response rate and response variability of area V4 neurons during the preparation of saccadic eye movements. J. Neurophys. 103 (3), 1171–1178.
Steinmetz, N.A., Moore, T., 2014. Eye movement preparation modulates neuronal responses in area V4 when dissociated from attentional demands. Neuron 83 (2), 496–506.
Sundberg, K.A., Mitchell, J.F., Gawne, T.J., Reynolds, J.H., 2012. Attention influences single unit and local field potential response latencies in visual cortical area V4. J. Neurosci. 32 (45), 16040–16050.
Suzuki, S., Cavanagh, P., 1997. Focused attention distorts visual space: an attentional repulsion effect. J. Exp. Psychol. Hum. Percept. Perform. 23 (2), 443–463.
Suzuki, M., Gottlieb, J., 2013. Distinct neural mechanisms of distractor suppression in the frontal and parietal lobe. Nat. Neurosci. 16 (1), 98–104.
Taylor, K., Mandon, S., Freiwald, W.A., Kreiter, A.K., 2005. Coherent oscillatory activity in monkey area v4 predicts successful allocation of attention. Cereb. Cortex 15 (9), 1424–1437.
Thompson, K.G., Biscoe, K.L., Sato, T.R., 2005. Neuronal basis of covert spatial attention in the frontal eye field. J. Neurosci. 25 (41), 9479–9487.
Treue, S., Martínez Trujillo, J.C., 1999. Feature-based attention influences motion processing gain in macaque visual cortex. Nature 399 (6736), 575–579.
Treue, S., Maunsell, J.H.R., 1996. Attentional modulation of visual motion processing in cortical areas MT and MST. Nature 382 (6591), 539–541.
Uylings, H.B., van Eden, C.G., 1990. Qualitative and quantitative comparison of the prefrontal cortex in rat and in primates, including humans. Prog. Brain Res. 85, 31–62.
Uylings, H.B.M., Groenewegen, H.J., Kolb, B., 2003. Do rats have a prefrontal cortex? Behav. Brain Res. 146 (1–2), 3–17.
Van Eden, C.G., Lamme, V.A.F., Uylings, H.B.M., 1992. Heterotopic cortical afferents to the medial prefrontal cortex in the rat. A combined retrograde and anterograde tracer study. Eur. J. Neurosci. 4 (1), 77–97.
Van Kerkoerle, T., Self, M.W., Dagnino, B., Gariel-Mathis, M.-A., Poort, J., van der Togt, C., Roelfsema, P.R., 2014. Alpha and gamma oscillations characterize feedback and feedforward processing in monkey visual cortex. Proc. Natl. Acad. Sci. U. S. A. 111 (40), 14332–14341.
Wang, M., Vijayraghavan, S., Goldman-Rakic, P.S., 2004. Selective D2 receptor actions on the functional circuitry of working memory. Science 303 (5659), 853–856.
Wardak, C., Olivier, E., Duhamel, J.-R., 2002. Saccadic target selection deficits after lateral intraparietal area inactivation in monkeys. J. Neurosci. 22 (22), 9877–9884.
Wardak, C., Olivier, E., Duhamel, J.-R., 2004. A deficit in covert attention after parietal cortex inactivation in the monkey. Neuron 42 (3), 501–508.
Wardak, C., Ibos, G., Duhamel, J.-R., Olivier, E., 2006. Contribution of the monkey frontal eye field to covert visual attention. J. Neurosci. 26 (16), 4228–4235.
Welch, K., Stuteville, P., 1958. Experimental production of unilateral neglect in monkeys. Brain 81 (3), 341–347.
Wilke, M., Turchi, J., Smith, K., Mishkin, M., Leopold, D.A., 2010. Pulvinar inactivation disrupts selection of movement plans. J. Neurosci. 30 (25), 8650–8659.
Williams, G.V., Goldman-Rakic, P.S., 1995. Modulation of memory fields by dopamine D1 receptors in prefrontal cortex. Nature 376 (6541), 572–575.
Wise, S.P., 2008. Forward frontal fields: phylogeny and fundamental function. Trends Neurosci. 31 (12), 599–608.

Womelsdorf, T., Anton-Erxleben, K., Pieper, F., Treue, S., 2006a. Dynamic shifts of visual receptive fields in cortical area MT by spatial attention. Nat. Neurosci. 9 (9), 1156–1160.

Womelsdorf, T., Fries, P., Mitra, P.P., Desimone, R., 2006b. Gamma-band synchronization in visual cortex predicts speed of change detection. Nature 439 (7077), 733–736.

Womelsdorf, T., Anton-Erxleben, K., Treue, S., 2008. Receptive field shift and shrinkage in macaque middle temporal area through attentional gain modulation. J. Neurosci. 28 (36), 8934–8944.

Wurtz, R.H., Goldberg, M.E., 1972. Activity of superior colliculus in behaving monkey. IV. Effects of lesions on eye movements. J. Neurophys. 35 (4), 587–596.

Yeshurun, Y., Carrasco, M., 1998. Attention improves or impairs visual performance by enhancing spatial resolution. Nature 396 (6706), 72–75.

Yeterian, E.H., Pandya, D.N., Tomaiuolo, F., Petrides, M., 2012. The cortical connectivity of the prefrontal cortex in the monkey brain. Cortex 48 (1), 58–81.

Zénon, A., Krauzlis, R.J., 2012. Attention deficits without cortical neuronal deficits. Nature 489 (7416), 434–437.

Zhang, K., Sejnowski, T.J., 1999. Neuronal tuning: to sharpen or broaden? Neural Comput. 11 (1), 75–84.

Zhang, Y., Meyers, E.M., Bichot, N.P., Serre, T., Poggio, T.A., Desimone, R., 2011. Object decoding with attention in inferior temporal cortex. Proc. Natl. Acad. Sci. U. S. A. 108 (21), 8850–8855.

Zhou, H., Desimone, R., 2011. Feature-based attention in the frontal eye field and area V4 during visual search. Neuron 70 (6), 1205–1217.

Zikopoulos, B., Barbas, H., 2006. Prefrontal projections to the thalamic reticular nucleus form a unique circuit for attentional mechanisms. J. Neurosci. 26 (28), 7348–7361.