Spatial attention enhances the selective integration of activity from area MT

Abbreviated title: Spatial attention enhances selective integration

Nicolas Y. Masse1,2, Todd M. Herrington3 and Erik P. Cook1

1. Department of Physiology, McGill University, Montreal, Quebec, Canada
2. Department of Neurobiology, University of Chicago, USA
3. Department of Neurobiology, Harvard Medical School, Boston, USA

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Corresponding author:
Nicolas Y. Masse
Department of Neurobiology
University of Chicago
Chicago, Illinois 60637
Phone 773-834-5186
masse@uchicago.edu

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Abstract

Distinguishing which of the many proposed neural mechanisms of spatial attention actually underlies behavioral improvements in visually guided tasks has been difficult. One attractive hypothesis is that attention allows downstream neural circuits to selectively integrate responses from the most informative sensory neurons. This would allow behavioral performance to be based on the highest quality signals available in visual cortex. We examined this hypothesis by asking how spatial attention affects both the stimulus sensitivity of Middle Temporal (MT) neurons and their corresponding correlation with behavior. Analyzing a data set pooled from two experiments involving four monkeys, we found that spatial attention did not appreciably affect either the stimulus sensitivity of the neurons or the correlation between their activity and behavior. However, for those sessions in which there was a robust behavioral effect of attention, focusing attention inside the neuron’s receptive field significantly increased the correlation between these two metrics, an indication of selective integration. These results suggest that, similar to mechanisms proposed for the neural basis of perceptual learning, the behavioral benefits of focusing spatial attention are attributable to selective integration of neural activity from visual cortical areas by their downstream targets.
Introduction

Although much progress has been made, we do not fully understand how sensory activity is combined across a pool of neurons to form a perceptual decision. For example, small fluctuations in the activity of cortical neurons encoding a stimulus are often correlated with the ability of a subject to discriminate, or detect, the stimulus on a trial-by-trial basis. What underlying sensory processes do these neural-behavioral correlations, commonly referred to as choice probability (CP) or detect probability (DP), reflect? And how might they be related to attention?

It has been suggested that CP and DP represent a causal measure of a sensory neuron’s contribution to a perceptual decision (Britten et al. 1996; Gu et al. 2007; Gu et al. 2011; Jazayeri 2008; Smith et al. 2011; Uka and DeAngelis 2004). However, inter-neuronal correlations (Cohen and Newsome 2009a; Shadlen et al. 1996) or feedback from downstream brain areas, such as attention (Herrington and Assad 2009; Nienborg andCumming 2009), could also explain CP and DP. Regardless of how these metrics arise, many studies have shown that neurons with stronger CP/DP tend to be more sensitive to the underlying stimulus (Britten et al. 1996; Cook and Maunsell 2002b; Dodd et al. 2001; Gu et al. 2007; Law and Gold 2008; Purushothaman and Bradley 2005; Smith et al. 2011; Uka and DeAngelis 2004). This observation is consistent with an optimal decoding strategy in which perceptual decisions are based on the activity of neurons with the highest stimulus sensitivity (Jazayeri and Movshon 2007; 2006).
Interestingly, the correlation between CP/DP and neural sensitivity may develop during behavioral training (Gold et al. 2010; Law and Gold 2008), which agrees with studies that suggest perceptual learning occurs when downstream networks learn to selectively integrate the activity of the most sensitive neurons (Dosher and Lu 1998; Gold et al. 2010; Jacobs 2009; Law and Gold 2008). Similarly, it has also been proposed that spatial attention allows downstream areas to selectively integrate the activity of neurons whose receptive fields overlap with the attended target (Fries et al. 2008; Womelsdorf et al. 2006). This proposal has been supported by several studies that have shown that attention synchronizes activity between visual and downstream cortical areas (Buschman and Miller 2007; Gregoriou et al. 2009; Saalmann et al. 2007; Siegel et al. 2008).

Although these past results do not discount the idea that CP/DP may reflect inter-neuronal correlations or feedback from downstream brain areas, taken together, they suggest that the correlation between CP/DP and neural sensitivity provides a measure of how sensory activity is integrated by downstream networks. If this were the case, then the correlation between CP/DP and neural sensitivity would depend on a subject’s attentional state.

To examine this hypothesis we analyzed the effects of attention on neural sensitivity and DP in area MT using data pooled from four monkeys performing two different motion-detection tasks. Spatial attention had little effect on either DP or neuronal sensitivity. However, for those recording sessions with a large behavioral effect of attention, there was a significant increase in the correlation between these two metrics.
In addition to selectively integrating activity from neurons with receptive fields at the attended location, our results suggest that spatial attention also allows downstream areas to selectively integrate the activity from the most informative neurons within this group. This result provides a mechanism by which spatial attention improves visual performance, complimentary to other proposed mechanisms (Buschman and Miller 2007; Cohen and Maunsell 2009; Cook and Maunsell 2004; Gregoriou et al. 2009; Khayat et al. 2010; Lee and Maunsell 2010; Mitchell et al. 2009; Reynolds and Heeger 2009; Saalmann et al. 2007; Siegel et al. 2008; Womelsdorf and Fries 2007). It also suggests a mechanism by which attention is sometimes necessary for perceptual learning (Ahissar and Hochstein 1993; Crist et al. 2001; Polley et al. 2006). Finally, we present a simple model to demonstrate how attention could allow for selective integration by synchronizing the spiking of the most informative neurons to oscillations in the local field.
Methods

Behavioral task for the motion-step experiment

The motion-step data set analyzed for this study comes from several previous studies (Cook and Maunsell 2002a; 2004; 2002b; Herrington et al. 2009; Masse and Cook 2008). Two male monkeys (Macaca mulatta) were trained to perform a spatially cued motion detection task (Figure 1A). The monkey initiated the trial by depressing a lever and fixating upon a central point. The monkeys were trained to release a lever when coherent motion began in one of two random dot patches diametrically opposite of the fixation point. After the cue was presented, 0% coherent motion began in the two patches followed by coherent motion occurring in one of the two patches at a random time (flat hazard function) 500 to 8000 ms afterwards. The location of the coherent motion was cued to the monkey at the start of the trial with static dots and this cue was valid on 80% of the trials. For validly cued trials, the strength of coherent motion was varied between three levels (low, medium and high) with the monkey correctly detecting the coherent motion 50%, 90% and 99% of the time for the three levels. For invalidly cued trials, only medium-strength coherent motion was presented to the monkey.

For our analysis in Figures 4, 5 and 7, behavioral detection performance and neural sensitivity (except when calculating for the correlation in Figure 7) were calculated using trials with medium-strength coherent motion which was common for both valid and invalid trials. For our analysis in Figures 5, 6 and 7, there were too few validly cued failed trials with medium-strength coherent motion to accurately calculate the DP; thus, we used trials with low-strength
coherent motion. For the correlations calculated in Figure 6 and 7, to ensure that the neural
sensitivity was calculated in a similar manner as the DP for validly cued trials, we also used trials
with low-strength coherent motion; the neural sensitivity and the DP for invalidly cued trials were
calculated using medium-strength coherent motion.

In all experimental sessions, the direction and the speed of the coherent motion were
matched to the preferred direction and speed of the neuron under study. The coherent motion
lasted 750 ms and the monkey had to release the lever from 200 to 750 ms after the onset of
cohort motion to obtain a reward. Trials where the monkey failed to release the lever or
released the lever too late were deemed missed trials. Only correct and missed trials were
included in the analysis. Trials where the monkey released the lever too early or was unable to
maintain fixation were discarded from the analysis.

Visual stimulus for the motion-step experiment

The animals sat 62 cm from a computer monitor (+/-17° by +/-13° of visual angle; 1600 by
1200 pixels; 75 Hz refresh). The stimuli consisted of two patches of white dots (each dot 0.25°
diameter; 78 cd/m²) on a dark gray background (12 cd/m²) with a dot density of 2.1 dots/degree².
Details of the visual stimulus have been previously (Cook and Maunsell 2002b).

Data collection for the motion-step experiment
All procedures were done in compliance with standards of the Baylor College of Medicine Animal Research Committee. Recordings were made from well-isolated single neurons in areas MT and details have been previously published (Cook and Maunsell 2002b; Cook and Maunsell 2002c). The monkey’s performance varied with patch location, size, and motion speed, which were determined by the response properties of the neuron under study. Consequently, different neurons were tested with different coherence levels. The animal’s eye position was measured every 5 ms using a scleral search coil and the times of action potentials were recorded to the nearest millisecond. In total, 92 MT were recorded in two monkeys. Four MT neurons were excluded because they did not have at least one correct and one failed trial in each attentional condition.

**Behavioral task for the speed-pulse experiment**

Two male monkeys (*Macaca mulatta*) were trained to perform a spatially cued speed-pulse detection task (Figure 1B). At the beginning of a trial the stimulus consisted of a central fixation spot and two annuli, one red and one green, in opposite hemifields at equal eccentricity. The monkey had to maintain gaze within a fixation window throughout the trial (2˚ by 2˚ square, centered on a fixation spot). After the monkey fixated, there was a 500 ms delay before two fields of coherently moving random dots appeared within the annuli. The monkey’s task was to detect a transient increase in the speed (53 ms, 4 video frames) of either dot patch and respond by releasing a touch bar within a requisite time window (200 – 600 ms). The color of the fixation point (red or green) cued the monkey as to which patch (surrounded by red or green annulus) was
more likely to contain the motion pulse (85% valid cues, 15% invalid cues). On 60% of
trials there was no change in the fixation point color cue (left column). On 40% of trials
the fixation point color cue switched at an unpredictable time during the trial to indicate
that the likely motion pulse location had switched (right column). Trials in which the
color cue switched were discarded for this analysis, although including these trials did not
substantially alter our results (data not shown). After an initial fixed delay of 400 ms,
additional delays until motion pulses and cue switches, as well as between cue switches
and motion pulses, were selected randomly from an exponential distribution (mean = 1 s).
This main purpose of the study was to examine effects of switching spatial attention, but
those results are not relevant to the questions examined here.

Visual stimulus for speed-pulse experiment

Stimuli were presented on a computer monitor positioned 57 cm in front of the
animal (40° by 30°, 75 Hz refresh, 1152 by 870 pixels). Details of the visual stimulus
have been previously published (Herrington and Assad 2009). Where possible, dot
patches were placed in the center of the receptive field of the recorded neuron. The dot-
patch motion in the receptive field was set in the neuron’s preferred direction as
determined by a direction-mapping task that we ran before the main task for each neuron.
The other dot patch was always placed at the equivalent position reflected across the
fixation point and had the opposite direction of motion. The size of the dot patches was
scaled with eccentricity (ranging from 4.5 to 9.4 degrees in diameter). The magnitude of
the speed pulse was chosen to maintain valid correct performance in the target range (65 – 75% correct) and varied from session to session (range for monkey B: 1.6x to 2.5x, range for monkey M: 1.35x to 1.7x).

Data collection for the speed-pulse experiment

All animal procedures, including animal surgeries and postoperative care, conformed to guidelines of the National Institutes of Health and the Institutional Animal Care and Use Committee at Harvard Medical School. Recordings were made from well-isolated single neurons in areas MT in both animals as previously described (Herrington and Assad 2009; 2010). MT cells were identified by reference to sulcal anatomy and characteristic physiology. MT cells were characterized by highly direction-selective receptive fields with diameters approximately equal to eccentricity (Maunsell and Van Essen 1983). All such stably isolated units were recorded. In all, we recorded from 67 MT neurons. Two neurons were discarded because they did not have at least one correct and one failed trial for both valid and invalid conditions.

Microsaccade detection

Microsaccades, which are small fixational eye-movements, have been shown to increase the correlation between neural activity and behavior when they occur near stimulus onset (Herrington et al. 2009). Thus, we removed all trials if they a microsaccade occurred from 100 ms before to 200 ms after stimulus onset. The peak angular speed of a movement had to exceed 10
deg/sec for it to be considered a microsaccade. Full details describing how eye movements were
classified as microsaccades have previously been published (Herrington et al. 2009).

Comparing two correlation coefficients using a bootstrap analysis

To determine whether the difference between two correlation coefficients was significant,
we performed two different bootstrap analyses. The first analysis was based on resampling
neurons. Given $n$ neurons in our population, we sampled (with replacement) $n$ neurons, and
calculated the correlation coefficient between the neural sensitivity and the DP. If we were
comparing valid to invalid trials, we would use the same $n$ neurons; if we were comparing strong
and weak group, we would sample two different sets of neurons. We then determined which
correlation coefficient was larger. This was repeated 100,000 times, and the p-value is equal to
either the probability that the correlation coefficient for the invalid trials was larger than the
correlation coefficient for the valid trials, or the probability that the correlation coefficient for the
weak attention group was larger than the correlation coefficient for the strong attention group.

The second bootstrap analysis was based on resampling trials. To compare correlation
coefficients between weak and strong attention groups, if a neuron had $n$ valid correct and $m$ valid
failed trials, we sampled (with replacement) $n$ valid correct and $m$ valid failed trials, and then
calculated its neural sensitivity and DP. After calculating these values for all neurons, we then
calculated the correlation between the neural sensitivity and the DP for both the weak strong
attention groups, and determined which correlation coefficient was greater. We repeated this
procedure 10,000 times, where the p-value is equal to the probability that the correlation
The coefficient for the weak attention group was larger than the correlation coefficient for the strong attention group.

To compare correlation coefficients between valid and invalid trials, if a neuron had $n$ invalid correct and $m$ invalid failed trials, we sampled (with replacement) $n$ invalid correct and $m$ invalid failed trials, and then calculated the invalid neural sensitivity and DP. We then sampled (with replacement) $n$ valid correct and $m$ valid failed trials, and then calculated the valid neural sensitivity and DP. Thus, we used an equal number of valid and invalid trials to compute the two metrics. After calculating these values for all neurons, we then calculated the correlation between the neural sensitivity and the DP for both valid and invalid trials, and determined which correlation coefficient was greater. We repeated this procedure 10,000 times, where the p-value is equal to the probability that the correlation coefficient for invalid trials was larger than the correlation coefficient for valid trials.

Simulation model

To understand how attention could simultaneously increase visual performance and the correlation between the neural sensitivity and DP while leaving the distribution of both of these values unchanged, we simulated the response of a population of MT neurons and a downstream area to a motion stimulus under weakly and strongly focused attention (Figure 8). The stimulus was similar to the motion-step and motion-pulse experiments: a 0% coherent motion stimulus was followed by 100 ms of coherent motion. All neurons fired with Poisson statistics in time bins of 2 milliseconds. We simulated 500 trials under both attentional states.
The model consisted of 1000 MT neurons whose mean firing rate to 0% coherent motion was 20 Hz and whose mean rate to coherent motion was uniformly sampled between 20 and 30 Hz. Additionally, the MT neurons were modulated by a common oscillatory signal. The oscillatory signal was obtained by filtering a Gaussian signal between 17 and 23 Hz with 4th order Butterworth filter. This signal acted on the MT neurons in a multiplicative manner, so that the firing rate of neuron $i$ was $r_i(t) = b_i (1 + \sqrt{\alpha_i} n(t))$, where $r_i(t)$ was the firing rate, $b_i$ was the base firing rate to either 0% coherent or coherent motion, $n(t)$ was the common oscillatory signal, and $\alpha_i$ was the coupling constant. All 1000 MT neurons, along with constant inhibition, projected onto a single neuron in the downstream network. The response in this downstream neurons was determined by $f(t) = \max (0, \sum_{i=1}^{1000} r_i(t) - 100)$, where $r_i$ is the firing rate of the $i^{th}$ MT neuron.

In this model, only the coupling constants were changed under attentional state. When attention was strongly focused, the coupling constants were proportional to the neuron’s increase in firing rate to coherent motion: $\alpha_i = \frac{c_i - 20}{10}$, where $c_i$ is the mean response of the $i^{th}$ MT neuron to coherent motion. When attention was weakly focused, we randomly shuffled these coupling constants between neurons.
Results

We were interested in whether spatial attention improves the selective integration of sensory activity when forming a perceptual decision. To test this hypothesis, we measured the effect of spatial attention on the neuronal correlation with behavior (DP), the neural sensitivity, and the correlation between the two.

Behavioral paradigms

We analyzed neuronal activity from 153 MT neurons collected from four monkeys participating in two similarly designed motion-detection experiments conducted in two different laboratories. Data from these experiments have been previously published in several different studies (Cook and Maunsell 2004; 2002b; Herrington and Assad 2009; 2010; Herrington et al. 2009; Masse and Cook 2008; Maunsell and Cook 2002) (see Methods). Although there are a number of studies that have measured the effects of attention on MT responses, what makes these two data sets relatively unique and critical to the goals of this analysis is that they measured both the behavioral and neural effects of spatial attention simultaneously.

Both experiments required the detection of a motion signal in one of two, diametrically opposite, random dot patches (RDPs) simultaneously presented to the monkey (Figure 1). One dot patch overlapped the MT neuron’s receptive field. The subject had to report with a lever
release the occurrence of a relatively weak signal that occurred at a random time. In both experiments, the dot patch that would likely contain this motion signal was cued to the monkey.

In the motion-step experiment (Figure 1A) two monkeys had to detect the onset of a coherent motion step in one of two, initially 0% coherent, RDPs (see Methods). A static dot patch before the start of the trial indicated the likely location of the coherent motion and was valid on 80% of the trials. In the speed-pulse experiment (Figure 1B), two monkeys had to detect a transient (53 ms) increase in speed in one of two, 100% coherently moving random dot patches. The color of the fixation point indicated the likely location of the speed pulse and was valid 85% of the time.

Neural sensitivity and detect probability

We wanted to know if spatial attention affected neural sensitivity to the motion signals the animals were trying to detect or their correlation with detection performance (“detect probability” or DP). In both experiments, the location, motion direction and size of the RDP were matched to the tuning properties of the neuron under observation, while in the motion-step experiment, the speed was also matched to the preferred speed of the neuron (see Methods). This was an important aspect of these experiments because it increased the chance that the recorded neural activity contributed to the monkey’s detection of the motion signal.
Neural sensitivity (also referred to as a neurometric) was used to quantify how well a neuron encodes the motion stimulus the animal was trained to detect. In our analysis, it was a trial-by-trial comparison of the neural activity in a window just after the motion stimulus occurred compared to the activity in a window just before the stimulus. It was calculated using an ROC analysis that provides an unbiased measure, ranging from 0 to 1, of the neural sensitivity (Britten et al. 1996; Cook and Maunsell 2002b; Green and Swets 1966). A neural sensitivity near 0.5 indicates that the neural response was on average equal between the two windows and the neuron conveyed little reliable information about the stimulus. A neural sensitivity much greater (or less) than 0.5 suggests high sensitivity and that the neuron reliably conveyed information about the onset of the motion stimulus.

Although the latency and the duration of the average neural response slightly differed between the two experiments, for simplicity, we used the same 150 ms analysis windows for both experiments to calculate the neural sensitivity: neural activity from 100 to 250 ms after stimulus onset (Figure 2A, green bar) was compared to activity from 0 to 150 ms before onset (gray bar). We only used neural activity from 100 ms after stimulus onset to match the neural latency to the speed-pulse (Herrington and Assad 2009), which was longer than the response latency to the motion-step (Cook and Maunsell 2002b). It should be noted small changes in window positions had no appreciable effects on any of our results (data not shown).

An example calculation of the neural sensitivity is shown in Figure 2 for a neuron recorded during the motion-step experiment. In the spike raster (Figure 2A), trials are aligned to the onset of the motion pulse and the blue dots indicate spikes recorded during correct trials while red dots indicate spikes recorded during failed trials. For this example neuron, the spike rate
distribution for the time period after stimulus onset (Figure 2B, green curve) was shifted to the right relative to the spike rate distribution measured before stimulus onset (gray curve). Thus, this neuron was relatively sensitive and did a good job of signaling the occurrence of the coherent motion pulse (neural sensitivity = 0.89).

DP (detect probability) is another widely used ROC-based metric that expresses how neural responses vary between correct and failed trials on a trial-by-trial basis (Cook and Maunsell 2002b), and is similar to choice probability (Britten et al. 1996). To compute DP we applied the same time window as used in the neural sensitivity calculation (Figure 2A, green bar). Similarly to neural sensitivity, a DP value of 0.5 indicates that spike rate was on average equal between correct and failed trials and was not correlated with behavioral report. Values close to 0 or 1 indicate that neural responses were strongly correlated with behavioral report. For the example neuron in Figure 2, the spike rate distribution for correct trials (Figure 2C, blue curve) was shifted to the right compared to the spike rate distribution for failed trials (red curve), indicating that the spike rate was on average greater for correct trials and that this neuron was relatively well correlated with the behavioral report (DP = 0.76).

The correlation between neural sensitivity and detect probability

Past studies have shown that there exists a weak correlation between neural sensitivity and the neuronal correlation with behavior (DP), which suggests that the activity of the most sensitive neurons are weighted more heavily when forming the perceptual decision (Britten et al. 1996; Cook and Maunsell 2002b; Dodd et al. 2001; Gu et al. 2007; Law and Gold 2008;
Purushothaman and Bradley 2005; Smith et al. 2011; Uka and DeAngelis 2004). We wanted to confirm this observation in our two experiments. Since the mean neural sensitivity and the DP values can differ between monkeys, combining raw neural sensitivity and DP values from multiple monkeys could introduce spurious correlations. Thus, we first normalized the neural sensitivity and the DP values for each monkey by subtracting the mean and dividing by the standard deviation. Neural sensitivity and DP were calculated using validly cued trials only (see Methods).

Consistent with past results, the normalized neural sensitivity was correlated with the normalized DP for both experiments (Figure 3A, motion-step, $R = 0.24$, $p = 0.02$; Figure 3B, speed-pulse, $R = 0.60$, $p < 10^{-6}$; Figure 3C, both experiments combined, $R = 0.39$, $p < 10^{-6}$). Note that the correlation was significantly weaker for the motion-step experiment ($p = 0.003$, bootstrap). This is possibly because there were many more trials for the speed-pulse experiment allowing for more accurate estimates of the neural sensitivity and the DP, leading to stronger correlations between the two (average number of trials for the motion-step experiment = 43; average number of trials for the speed-pulse experiment = 230, t-stat = 14.25, $p < 10^{-29}$, two-sided t-test). Alternatively, the stronger correlation for the speed-pulse experiment is possibly because of the transient nature of the stimulus (53 ms for the speed-pulse experiment compared to 750 ms for the motion-step experiment). In the speed-pulse task the subjects likely formed decisions based on shorter periods of neural activity that were better matched to our analysis window. Regardless of windowing, these correlation values are consistent with past results that suggest the activity of the most informative neurons tend to be more strongly linked to the perceptual decision.
The effect of attention on behavioral detection performance

We were interested in the interplay between attentional improvements in visual performance and the affects of attention during the integration of sensory activity. We characterized the behavioral effect of attention as the difference in performance between valid trials, in which the monkey was cued to the patch that contained the motion or speed signal, and invalid trials, in which the monkey was cued to the patch which did not contain the signal. Our behavioral effect of attention was quantified using the ratiometric score

\[ \Delta \text{Perf} = \frac{\% \text{ correct valid} - \% \text{ correct invalid}}{\% \text{ correct valid} + \% \text{ correct invalid}} \]

As expected, the detection performance for both experiments was better for valid trials (Figure 4A, motion-step experiment: \( \Delta \text{Perf} = 0.34 \pm 0.02 \), t-stat = 14.19, \( p < 10^{-23} \); Figure 4B, speed-pulse experiment: \( \Delta \text{Perf} = 0.36 \pm 0.03 \), t-stat = 11.47, \( p < 10^{-16} \), two-sided t-test).

As demonstrated by Posner (1980) and many others, there is a direct link between how a subject allocates spatial attention and their ability to detect a weak visual stimulus presented in one of two possible locations. Thus, we assigned neurons to one of two attentional groups, which were of equal size for both experiments. If the difference in detection performance between the valid and invalid trials was small, then we assumed the subject’s attention was not focused on a single RDP, but either broadly spread across both RDPs or possible undefined (e.g., a lack of motivation). We refer to this as the “weak attention” group, which includes all neurons to the left of the vertical dashed-lines in Figure 4A&B. When the performance for valid trials was much greater than invalid trials, then we assumed the subject had reasonably focused their attention on the cued RDP at the expense of the non-cued RDP. This is referred to as the “strong attention” group, which includes all neurons to the right of the vertical dashed-lines in Figures 4A&B.
The importance of having a behavioral estimate of attentional allocation cannot be overstated when using metrics such as neural sensitivity and DP, which are both subject to noise when calculated over a finite number of trials. By focusing on the strong attention group, we minimized the potential noise associated with a weak, or undefined, attentional state. We hypothesized that the effects of attention on neural sensitivity and DP would be more distinct between valid and invalid trials for the strong attention group, given the larger difference in detection performance. However, comparing neural correlates between valid and invalid trials introduces a potential confound because there were far fewer invalid than valid trials. Alternatively, one can compare differences between the strong and weak attention groups using only valid trails, which were more numerous. However, this analysis introduces a second potential confound because we are comparing two different sets of neurons.

Given the distinct advantage and disadvantage of each comparison, we performed both:

1) We measured how neural sensitivity, DP and their correlation changed between valid and invalid trials for the strong attention group (schematically illustrated in Figure 4C), which provided a “within neuron” comparison using fewer number of trials. 2) We measured how these two metrics changed between the strong and weak attention groups using only valid trials (Figure 4D), which provided a “between neuron” comparison using the large number of validly cued trials.

The effect of attention on neural sensitivity and detect probability
The behavioral effects of spatial attention could be the result of a change in the sensitivity of the neurons (neural sensitivity) and/or a change in their correlation with behavior (DP). For example, focused attention could increase visual performance by increasing the reliability of neurons encoding the stimulus, which would be revealed by an increase in the neural sensitivity (McAdams and Maunsell 1999). Alternatively, the increase in visual performance could occur if activity from neurons whose RF overlapped the attended location became more tightly linked to the perceptual process, producing a corresponding increase in DP. We compared how neural sensitivity and DP varied between valid and invalid cued trials for those neurons in the strong attention group, and how the neural sensitivity and DP varied between the strong attention and weak attention groups using only valid trials (Figure 5).

For the motion-step experiment, combining neurons from both monkeys, there was no difference for either the neural sensitivity or the DP values when comparing valid to invalid trials (red versus blue) for the strong attention group (Figure 5A, \( \Delta \) Neural sensitivity = \(-0.01 \pm 0.01\), t-stat = \(-0.73\), \( p = 0.47 \); \( \Delta \) DP = \(0.01 \pm 0.03\), t-stat = \(0.27\), \( p = 0.79 \), two-sided, paired t-test, \( \Delta \) = difference in means) or when comparing the strong to weak attention groups (black versus gray) using only valid trials (Figure 5B, \( \Delta \) Neural sensitivity = \(-0.01 \pm 0.03\), t-stat = \(-0.24\), \( p = 0.81 \); \( \Delta \) DP = \(-0.01 \pm 0.03\), t-stat = \(-0.25\), \( p = 0.80 \), two-sided t-test). For the speed-pulse experiment, there was a small, but non-significant increase in the neural sensitivity and decrease in DP between valid and invalid trials for the strong attention group (Figure 5C, \( \Delta \) Neural sensitivity = \(0.02 \pm 0.02\), t-stat = \(1.20\), \( p = 0.24 \); \( \Delta \) DP = \(-0.03 \pm 0.03\), t-stat = \(-1.10\), \( p = 0.28 \), two-sided, paired t-test). This trend was also true when comparing the strong attention to weak attention group, although the differences were marginally significant (Figure 5D, \( \Delta \) Neural sensitivity = \(0.07 \pm 0.034\), t-stat = \(1.98\), \( p = 0.05 \); \( \Delta \) DP = \(-0.05 \pm 0.020\), t-stat = \(-2.34\), \( p = 0.02 \), two-sided t-test).
Overall, the effect of attention on both neural sensitivity and DP was small and not consistent across the two experiments. A lack of change in these metrics might be expected when comparing the strong and weak attention groups using only the validly cued trials (Figure 5B&D). This is because neural sensitivity and DP in these two groups would likely depend on the response properties of the neurons themselves and less on the behavioral performance of the animal.

The lack of an appreciable change in neural sensitivity for the valid versus invalid comparison (Figure 5A&C, left column), however, was somewhat surprising. For example, attention has been shown to increase a similar measure of neural sensitivity in area V4 by ~23% (McAdams and Maunsell 1999). One explanation could be that compared with the larger attentional modulation reported in the V4 study, the median attentional modulation in firing rate for the MT neurons analyzed here was relatively small (10 and 11% for the analysis windows located before and after the signal, respectively). Another reason could be due to the choice of our short 150 ms analysis window. However, a previous study that used a longer 300 ms window in the motion-step experiment showed only a 3.5% increase in neural sensitivity between validly and invalidly cued trials (Maunsell and Cook 2002). We were also puzzled that DP showed no systematic change between the valid and invalid conditions (Figure 5A&C, right column), as attention might be expected to alter the functional link between MT neurons and behavior. Using a computational model (below), we will illustrate how improvements in behavioral performance due to attention can arise with no change in either neural sensitivity or DP.
These results in Figure 5 raise the question that if attentional state had a no appreciable effect on either neural sensitivity or DP, then how can we explain the large changes of attention on behavioral performance? To answer this, we next examined the relationship between these two metrics.

The effect of attention on the correlation between neural sensitivity and detect probability

It has been repeatedly observed that there is a positive correlation between a cortical neuron’s ability to encode the stimulus (neural sensitivity) and its functional link to behavior (measured as DP in our study) (Britten et al. 1996; Cook and Maunsell 2002b; Dodd et al. 2001; Law and Gold 2008; Purushothaman and Bradley 2005; Uka and DeAngelis 2004). It has been suggested that this positive correlation implies a causal relationship where the more informative neurons contribute more to a visually guided behavioral task (Britten et al. 1996; Cook and Maunsell 2002b; Dodd et al. 2001; Law and Gold 2008; Purushothaman and Bradley 2005; Uka and DeAngelis 2004). This view, however, has recently been challenged by studies that suggest that a neuron’s correlation with behavior is not causal, but rather attributable to top-down modulation (Nienborg and Cumming 2009). Both interpretations, which are not mutually exclusive, have important implications for our understanding of how cortical neurons underlie perception and behavior.

Consistent with past studies and shown in Figure 3 above, the correlation between DP and neural sensitivity in our two data sets tended to be dependent on the noise levels in our estimates of the neural sensitivity and DP metrics. This presented a challenge when we grouped
our neurons based on attentional state, which reduced the number of trials available to compute each metric. Thus, to increase statistical power, we normalized neural sensitivity and DP values for each monkey and then combined neurons from both experiments. As emphasized above when comparing neural sensitivity and DP for only the validly cued trials (Figure 3), the normalization process is critical to prevent the introduction of spurious correlations due to differences in the mean values of each metric. We then compared the correlation coefficients using two bootstrap analyses; in one we resampled neurons with replacement and in the other we resampled trials with replacement (see Methods).

We first compared the correlation between neural sensitivity and DP for validly versus invalidly cued trials for the strong attention group (Figure 6A). Combining the data across animals and experiments we found that the correlation between neural sensitivity and DP for valid trials (blue) was significantly greater than the correlation for invalid trials (red) (Figure 6A, R-valid = 0.50, R-invalid = -0.01, p(resampling neurons) < 0.001, p(resampling trials) = 0.005, R is the Pearson correlation coefficient). We next compared the correlation between the strong and weak attention groups (Figure 6B). Using only the validly cued trials, the correlation between the neural sensitivity and the DP for the strong attention group was significantly greater than the correlation for the weak attention group (Figure 6B, R-strong = 0.60, R-weak = 0.18, p(resampling neurons) < 0.001, p(resampling trials) < 0.001).

These results were consistent across both experiments. For the motion-step experiment, the correlation between the neural sensitivity and the DP for the strong attention group was greater for valid than for invalid trials (R-valid = 0.51, R-invalid = 0.12, p(resampling neurons) = 0.02, p(resampling trials) = 0.04) and was greater for the strong attention group compared to the
weak attention group using only valid trials (R-strong = 0.51, R-weak = –0.03, p(resampling neurons) = 0.003, p(resampling trials) = 0.004). This was also true for the speed-pulse experiment R-valid = 0.49, R-invalid = –0.26, p(resampling neurons) < 0.001, p(resampling trials) = 0.02; R-strong = 0.75, R-weak = 0.45, p(resampling neurons) = 0.02, p(resampling trials) = 0.04).

In both our comparison groups, focusing spatial attention increased the correlation between DP and neural sensitivity. These results suggest that attention allows downstream areas to selectively integrate the activity of the most informative MT neurons. In the following sections, we examined if two potential confounds, different numbers of trials between conditions and common feedback, biased our results.

**Controlling for the number of trials**

The variance in the estimates of neural sensitivity and DP depends on the number of trials used to compute the metrics. Thus, the number of trials used to calculate neural sensitivity and DP can affect the correlation between the two. Fewer trials could lead to a noisier estimate of both the neural sensitivity and the DP, reducing the correlation between the two. For the “between neuron” comparison between the strong and weak attentional groups (Figure 6B), we found that the number of trials could not explain the difference in the correlation between the neural sensitivity and the DP: there was on average 131.3 trials for the weak attention group while there was on average 116.8 trials for the strong attention group.
Slightly more care had to be taken in the “within neuron” comparison of the neural sensitivity versus DP correlation between validly and invalidly cued trials for the strong attention group (Figure 6A). For the speed-pulse experiment, 85% of the trials were valid while 15% were invalid. Thus, for this experiment, we used all invalid trials to calculate the invalid neural sensitivity and DP. However, to calculate the valid neural sensitivity and DP we only used the same number of failed and correct as there was for the invalid case. If there were more valid failed (correct) trials than invalid failed (correct) trials, we eliminated valid failed (correct) trials to produce an equal number of valid and invalid trials. For the motion-step experiment, the task was designed so that an equal number of low-coherence valid trials and medium-coherence invalid trials were presented to the monkey. However, due to differing number of aborted trials, there was on average 38.1 valid trials compared to 32.6 invalid trials. To ensure that the greater number of valid trials was not responsible for the difference in the correlation between the neural sensitivity and the DP, we eliminated 11 out of 44 neurons with the largest difference between valid and invalid trials so that the mean number of valid and invalid trials was essentially matched in the remaining group (33.7 valid trials compared to 33.5 invalid trials) and recalculated the correlation between the neural sensitivity and the DP. Eliminating these 11 neurons increased the difference in the neural sensitivity versus DP correlation between valid and invalid trials (R-valid = 0.57 versus R-invalid = –0.007, p(resampling neurons) < 10^{-4}, p(resampling trials) = 0.02 ). Thus, we conclude that the differing number of trials between conditions cannot easily explain the difference in the neural sensitivity versus DP correlation.

Controlling for common feedback
Two recent studies have suggested that neuronal correlations with behavior (DP) arise, at least partly, through feedback mechanisms, possibly related to attention (Herrington and Assad 2009; Nienborg and Cumming 2009). For example, feedback could produce a positive neuronal correlation with behavior in the experiments examined here if the subjects rapidly focused more spatial attention on the dot patch immediately after the motion stimulus was detected. Support for this hypothesis was given in a previous analysis of the speed-pulse experiment (Herrington and Assad 2009), where the authors found that the correlation between neuronal sensitivity and DP (calculated differently) was greater when neuronal sensitivity was calculated using only correct trials compared to using only failed trials. Thus, we wondered if a lack of feedback during failed trials would eliminate the increase in DP versus neural sensitivity correlation for the strongly focused attention group.

We repeated our analysis from Figure 6 using only failed trials to calculate the neural sensitivity. As in the previous study, we also found that the correlation between neural sensitivity and DP was reduced when the neural sensitivity was calculated using only failed trials, but the significant differences between valid and invalid trials for the strong attention group, and the significant difference between the strong attention and weak attention groups remained intact (R-valid = 0.29 versus R-invalid = −0.28, p(resampling neurons) < 0.001, p(resampling trials) = 0.005; R-strong = 0.38 versus R-weak = −0.18, p(resampling neurons) < 0.001, p(resampling trials) = 0.003). Thus, these results suggest top-down feedback signals only partly explain DP and its correlation with the neural sensitivity.

Summary of experimental results
The effects of spatial attention on behavioral performance, spike rate, neural sensitivity, DP, and the correlation between neural sensitivity and DP are summarized for the valid versus invalid groups (Figure 7A) and strong versus weak attentional groups (Figure 7B). In this analysis we normalized the attentional effects to either the validly cued or the strong attention group (white bars) to illustrate the relative change of each metric when spatial attention was removed (or not focused) on the motion stimulus (gray bars). Note that we normalized the effects for each experiment individually, and thus Figure 7 shows the average correlation between neural sensitivity and DP while Figure 6 shows the change in correlation when the two experiments are combined.

For the valid versus invalid summary (Figure 7A), this “within neuron” analysis compared each metric for the invalidly cued trials (gray bar) normalized to the validly cued trials (white bars). On average, the invalid cue produced a large reduction in both behavioral performance ($p < 10^{-6}$, paired t-test) and the correlation between neural sensitivity and DP. Although there was a small reduction in mean spike rate (calculated using the same time window used to calculate the DP, $p = 0.0001$), withdrawing attention due to the invalid cue had no net effect on either neural sensitivity or DP ($p = 0.76$ and 0.59, respectively).

Our strong versus weak attentional analysis “between neurons” compared each metric using only validly cued trials (Figure 7B). To be comparable with the valid versus invalid effects in Figure 7A, however, the behavioral effects in Figure 7B show the average normalized $\Delta$ Perf, which is strength of the attentional modulation of behavior for each group (see Figure 4). The strongly focused attention group (white) had a greater modulation of behavior compared to the weakly focused attention group (gray), which was due to how we divided the neurons (see Figure...
In addition, there was a much higher correlation between neural sensitivity and DP for the strong versus weak attentional groups. However, there was no significant change in average firing rate, neural sensitivity and DP (p = 0.88, 0.57 and 0.53, respectively, t-test) between these two groups.

This summary highlights that the change in correlation between neural sensitivity and DP shows the greatest amount of modulation between the two attention conditions. Interpreting what these results mean in terms of a neural mechanism of attention, however, is not straightforward. This is because a change in the correlation between two metrics is not as intuitive a change in a single metric such as neural sensitivity or firing rate. Thus, we next used a computational model to explore how attentional changes in the correlation between neural sensitivity and DP, with no change in either metric, might come about.

Modeling the selective integration of the most informative sensory activity

The results of this study suggest that attention improves visual performance by selectively integrating the activity of the most informative sensory neurons, which strengthens the correlation between neural sensitivity and DP. To investigate potential mechanisms for this selective integration, we constructed a computer model of attention that would increase the correlation between DP and neural sensitivity, but not change their individual distributions.
Many models of perceptual decision-making integrate responses from pools of sensory neurons (Cohen and Newsome 2009b; Law and Gold 2009; Shadlen et al. 1996). We used a similar architecture with a downstream area integrating the inputs from many MT neurons (Figure 8; see Methods for details). We varied the sensitivity of different neurons in our model to produce a range of neural sensitivity values. The input to the model was a short 100 ms step to mimic the motion signal and the output was a decision neuron, labeled $D$, whose activity represented the downstream network’s estimate of the input signal. It was assumed that the value of the decision variable, $D$, was directly linked to behavioral performance.

We first tested an existing model in which motion-detection is based on a weighted sum of MT activity (Law and Gold 2009). Consistent with their results, we found that preferentially weighting the output of the most sensitive MT neurons during strongly focused attention could account for our results provided that inter-neuron correlations were greater for pairs of neurons with similar motion sensitivity (data not shown). Although the strength of the synaptic connections between area MT and downstream networks might underlie this preferential weighting, this would require a large number of synaptic changes (at least $M \times N$ given $M$ MT neurons and $N$ downstream neurons) in a very short time period. Given that spatial attention requires hundreds of milliseconds to be re-allocated (Herrington and Assad 2009), this re-weighting mechanism seemed unlikely.

Thus, we focused on a model inspired by studies suggesting that attention synchronizes spiking activity to oscillations in the local field potential (Buschman and Miller 2007; Fries et al. 2001; Gregoriou et al. 2009; Saalmann et al. 2007; Siegel et al. 2008; Womelsdorf et al. 2006) that would require only $M$ synaptic changes (as opposed to $M \times N$) under changes in attentional
We modeled a population of 1000 MT neurons that were equally weighted by one downstream “decision” neuron (D). The decision neuron also received a constant inhibitory input. The gain of all MT neurons was modulated by a common oscillatory signal, \( n(t) \), that acted in a multiplicative manner: \( 1 + \sqrt{\alpha} n(t) \), where \( \alpha \) was a scalar value giving the strength of the modulation. The oscillatory signal \( n(t) \) had a mean of zero, thus it had no net effect on the MT neurons neural sensitivity.

The intuition behind this model is that the downstream neuron requires strong excitation from MT in order to overcome the constant inhibition it receives. This strong excitation can only be provided if many MT neurons are active within a short time window. In other words, those MT neurons that are strongly modulated by the oscillatory signal will have the greatest of chance of firing together during the peak of the oscillatory signal. Thus, neurons that are strongly modulated by the oscillatory signal will contribute most to the response of the downstream.

In our model, when attention was strongly focused, the oscillatory signal modulated the gain of the MT neurons proportional to their sensitivity to coherent motion (Figure 8A, bottom panel). Thus, the more informative MT neurons (high neural sensitivity, filled circles) were more strongly modulated by the oscillatory signal (high \( \alpha \), thick lines). We make no attempt to explain the neural mechanism that could allow for this proportional modulation. When attention was
weakly focused, we randomly shuffled the strength of the modulation so that the oscillatory modulation and neuronal sensitivity was mismatched (Figure 8A, top panel).

We simulated 500 trials for both attentional cases. Strongly focusing attention increased the mean response of the downstream decision neuron, \( D \), to a simulated motion step by 18.7%, which allowed downstream neurons to better “detect” the motion signal (Figure 8B). Importantly, this mechanism of attention had no effect on the neural sensitivity or DP values of the 1000 MT neurons (Figure 8C). Note that we did not optimize the model to mimic the same neural sensitivity and DP distributions observed in the data. Focusing attention, however, strongly increased the correlation between neural sensitivity and DP (Figure 8D). While this model does not attempt to fully capture all aspects of attention or how downstream areas process MT activity, it suggests that a simple neural mechanism based on effectively synchronizing activity can mimic our experimental results.
Discussion

We investigated whether spatial attention affected the selective integration of sensory activity in area MT of visual cortex. We pooled data from two experiments that simultaneously measured both the behavioral and neural effects of attention in order to link attentional effects of task performance to neural sensitivity and the neuronal correlation with behavior (DP). As expected, spatial attention improved behavioral performance. We also found that the strength of attentional focus, as determined by our behavioral analysis, had no systematic effect on MT neuronal sensitivity or DP. However, we found that the correlation between the neural sensitivity and DP changed with the behavioral impact of attention. For those neurons associated with a large behavioral effect of attention, the correlation was greater when attention was cued inside the neuron’s RF. For those neurons for which there was a weak behavioral effect of attention, the correlation between neural sensitivity and DP was significantly reduced.

These results support the hypothesis that spatial attention increases visual performance by enhancing the brain’s ability to selectively integrate activity from the most informative neurons. These results could be replicated using a relatively simple model where the activity of MT neurons was modulated by an oscillatory signal (Fries et al. 2001; Gregoriou et al. 2009; Saalmann et al. 2007; Womelsdorf et al. 2006). If this signal selectively modulated the gain of the most informative neurons during focused attention, then downstream neurons responded more strongly to the stimulus and it reproduced the observed correlation between neural sensitivity and DP values. At the same time, however, the overall sensitivity and correlation with behavior of the population of model MT neurons did not change between attentional conditions.
Multiple mechanisms of attention

This study complements previous results that have shown that attention increases the coherence between visual neurons with receptive fields at the attended location and downstream areas (Buschman and Miller 2007; Gregoriou et al. 2009; Saalmann et al. 2007; Siegel et al. 2008). However, attention has been proposed to increase visual performance by other mechanisms. One longstanding idea is that attention modulates firing rates via a normalizing mechanism (Khayat et al. 2010; Lee and Maunsell 2010; Reynolds and Heeger 2009) that enhances the neural sensitivity of neurons in visual cortex (McAdams and Maunsell, 2002).

Although the valid versus invalid cued conditions produced a small modulation in firing rate for our two data sets (Figure 7A), it was not large enough to produce a reliable increase in the average ROC-measured neural sensitivity. This does not mean that increased neural sensitivity would not be useful. For example, if an increase in neural sensitivity occurred with attention, it would enhance the benefits of selectively integrating sensory information. In this model, attention would allow downstream areas to selectively integrate the activity from the most informative neurons whose sensitivity is further maximized by attention.

Other studies have recently shown that attention increases the signal-to-noise ratio of the population response by reducing noise correlations (Cohen and Maunsell 2009; Mitchell et al. 2009). These studies also found that improvements in performance with attention are not primarily due to changes in neural sensitivity, consistent with our results. In general, reducing correlations between neurons, increasing neural sensitivity and selective integration of the most informative neural signals are all mutually compatible mechanisms, and the brain may employ many methods to enhance visual performance at the attended location.
Attention and perceptual learning

Our results linking attention with selective integration also complement recent findings in perceptual learning. Several studies have suggested that perceptual learning occurs when downstream areas learn to selectively integrate neural activity from the most informative neurons (Dosher and Lu 1998; Gold et al. 2010; Jacobs 2009; Law and Gold 2008). Some of the most direct evidence for this hypothesis comes from a recent perceptual learning experiment by Law and Gold (2008). The authors measured behavioral improvements in a motion discrimination task during training, while simultaneously recording MT activity that encoded the motion stimulus. Over the course of training, the sensitivity of the MT neurons remained constant, while the neuronal correlation with behavior increased slightly. Notably, the correlation between these two metrics increased steadily over the training period, suggesting that the readout of area MT became more selective as the animals improved their behavioral performance.

If training increases performance in perceptual tasks because downstream brain areas learn to selectively integrate the most informative sensory neural activity, then we propose that spatial attention aids perceptual learning by enhancing this selective integration. This model would also explain related findings that task-specific attention is usually required for perceptual learning (Ahissar and Hochstein 1993; Crist et al. 2001; Polley et al. 2006).

Interpretation of detect probability
Because we were unable to measure directly how downstream neural circuits integrate neural activity from MT, we used the correlation between MT neural activity and behavior (DP) as a surrogate. However, the mechanisms responsible for neuronal correlations with behavior are currently under debate. It was originally proposed that neuronal correlations with behavior represent feed-forward (i.e., causal) contributions of sensory neuron activity to the perceptual decision (Britten et al. 1996). Conversely, it has been shown that neuronal correlations with behavior can arise from intrinsic correlations between neurons in the population (Shadlen et al. 1996; Zohary et al. 1994) or other common inputs such as microsaccades (Herrington et al. 2009).

Thus, it is possible for a neuron to contribute little towards a perceptual decision and yet to be correlated with it by virtue of its correlation with other neurons. However, this does not necessarily mean that neuronal correlations with behavior do not reflect how downstream networks integrate neural activity. The model we presented in Figure 8 is one example of how a neuron’s detect probability reflects how its activity is integrated downstream.

Two studies have suggested that feedback signals, possibly in the form of attention, can partly explain the neural correlation with behavior (Herrington and Assad 2009; Nienborg and Cumming 2009). Given that attention affects task behavioral performance, and also modulates the gain of MT neural activity, it is entirely possible that attention can induce a correlation between the two. However, if the subjects were switching attention near stimulus onset, then attentional modulation could equally affect both the neural sensitivity and the DP, inducing a correlation between the two and biasing our results. To counter this possibility, we recalculated the neural sensitivity using only failed trials, and then measured correlation between the neural sensitivity
and the DP. If the subject failed to detect the stimulus, then there is no reason to suspect that the subject switched his attention towards a stimulus it did not observe.

Another possibility is that the subjects switched their attention away from the patch containing the stimulus, possibly explaining the failure to detect it. This would induce a positive correlation between neural activity and behavior (DP) and also decrease the neural sensitivity, which would lead to a negative correlation between the neural sensitivity and the DP. Although the correlation between the neural sensitivity and DP was reduced when only using failed trials to calculate the neural sensitivity (to be expected since using fewer trials to calculate the neural sensitivity would increase the variability in our estimate), it could not explain our results. Thus, our results are consistent with a feedback mechanism partly explaining DP, but we do not believe it can fully explain the correlation we observed between neural sensitivity and DP. This correlation is instead consistent with a large body of evidence that suggests DP versus neural sensitivity correlations provide an estimate of how downstream networks integrate sensory neural activity (reviewed by Jazayeri (2008)).

Implications of model

What might be the neural mechanism of spatial attention that allows downstream networks to properly integrate neural activity? In Figure 8, we presented a possible model where the responses of MT neurons were modulated by oscillations in the local field potential, and the strength of this modulation depended on the motion sensitivity of the neuron. Studies have shown that attention increases synchrony between oscillations in the local field potential and spiking...
activity from V4 (Fries et al. 2001; Gregoriou et al. 2009; Womelsdorf et al. 2006) and MT (Saalmann et al. 2007); our model predicts that the degree of synchrony should depend on the neuron’s stimulus sensitivity and its DP. Thus, the spiking of the most sensitive neurons or those with the largest DP values should be more phase locked to oscillations of the LFP within a certain frequency band than less sensitive neurons. Although we don’t predict which frequency band, several studies have suggested a role for oscillations within the gamma band for coordinating activity during attention (Fries et al. 2001; Fries et al. 2008; Womelsdorf et al. 2006).

Unfortunately, our model leaves several issues unresolved. First, past studies have shown that attention reduces noise correlations between neurons in area V4 (Cohen and Maunsell 2009; Mitchell et al. 2009); we cannot confirm this was also true for area MT since only a single neuron was recorded at a time during these experiments. Thus, we did not attempt to incorporate this property into our model. Second, our model also does not address the increase in coherence between LFP oscillations in visual and downstream areas with attention (Buschman and Miller 2007; Fries et al. 2008; Saalmann et al. 2007; Siegel et al. 2008). Finally, how the brain learns the identity of those neurons most sensitive to the motion stimulus, and how they potentially selectively modulate the activity of these neurons, is unknown. A combination of further experimental and theoretical studies can hopefully address these outstanding issues.
Figure Legends

Figure 1 Experimental design. (A) Motion-step experiment from Cook and Maunsell (2002a). The goal of this task was to detect a coherent motion step in one of two diametrically opposed random dot patches (RDPs). The monkey was first presented with a single static dot patch which indicated the likely location of the coherent motion signal, followed by 0% coherent motion (500 to 8,000 ms), followed by a 750 ms coherent motion step in one of the two dot patches. The monkey had to release a lever from 200 to 750 ms after the coherent motion onset to receive a reward. For 80% of trials, the coherent motion step occurred in the RDP indicated by the static cue at the start of the trial (valid trials, left column). For 20% of trials, the coherent motion step occurred in the uncued dot patch (invalid trials, right column). (B) Speed-pulse experiment from Herrington and Assad (2009). The goal of this task was to detect a brief speed change in one of two diametrically opposed RDPs. Two colored annuli and a colored fixation point appeared for 500 ms to cue the animal which RDP would contain the motion signal. This was followed by 100% coherent motion inside the annuli and the monkey’s task was to release a lever in response to a transient (53 ms) increase in speed at either of the RDPs. The color cue was valid for 85% of trials (valid trials, left column). In 15% of trials, the motion pulse occurred in the uncued patch (invalid trials, right column). The monkey had to release a lever within 200 to 600 ms of the speed pulse to obtain a reward. On 60% of trials, fixation point color cue would remain constant. Trials in which the color cue changed during a trial were discarded from this analysis.

Figure 2. Example neural sensitivity and neural correlation with behavior (detect probability or DP). (A) Spike raster for an example neuron recorded during the motion-step experiment. Spike times relative to coherent motion onset from correct trials are represented by blue dots while
spike times from failed trials are represented by red dots. Dots lying along the same horizontal line represent spike times from a single trial. The DP compared neural activity between correct and failed trials from 100 to 250 ms after stimulus onset (green shaded area), while the neural sensitivity compared neural activity from 100 to 250 ms after stimulus onset to 0 to 150 ms prior to stimulus onset (gray shaded area). (B) The distribution of spike counts occurring immediately before (shown in black) and after (shown in green) the coherent motion signal was used to compute the neural sensitivity. The time windows used to compute counts are those shown in (A). An area under the ROC curve (AROC) was computed from the two distributions of spike counts to produce a measure of neural sensitivity. (C) The distribution of spike counts for correct (blue) and failed (red) trials after the coherent motion signal onset. The distribution of spike counts for correct and failed trials were used to calculate the DP using a similar AROC calculation. This example neuron had a relatively strong neural sensitivity and correlation with behavior (DP).

**Figure 3** The correlation between the neural sensitivity and the detect probability (DP). (A) Scatter plot of the normalized neural sensitivity and the normalized DP for 88 MT neurons from the two monkeys that performed the motion-step task. Values were normalized for each monkey by mean-subtracting and dividing by the standard deviation. (B) Same as (A) with 65 MT neurons from the two monkeys that performed the speed-pulse task. (C) Same as (A) with all 153 MT neurons combined from all four monkeys from both experiments. \( R \) is the Pearson’s correlation coefficient.

**Figure 4** The behavioral effect of attention. (A) The attentional modulation of performance between trials with valid and invalid cues for the motion-step experiment. Attentional modulation of behavior was quantified by \( \Delta \text{Perf} = \frac{\% \text{correct valid} - \% \text{correct invalid}}{\% \text{correct valid} + \% \text{correct invalid}} \). The vertical dashed grey
line evenly divides neurons for which there was a correspondingly small amount of attentional
modulation of behavior (the weak attention group) from those neurons for which there was a
large modulation in performance (the strong attention group). (B) Same as (A) for the speed-
pulse experiment. (C) We made two comparisons to assess the effects of attention on neural
sensitivity, DP, and their correlation. The first comparison was between valid and invalid cueing
for the neurons in the strong attention group only. The allocation of attention (red highlight), RF
(dashed circle) and location of the motion signal (arrow) are illustrated for each group. For the
valid cue, both cue and motion signal occurred in the neuron’s RF. For the invalid cue, the cue
was outside the neuron’s RF while the motion signal occurred inside the RF. (D) The second
comparison was between the strong attention and the weak attention groups, using only validly
cued trials. The valid versus invalid comparison provided a “within” neuron comparison while
the strong versus weak provided a “between” neuron comparison.

Figure 5 Spatial attention had no systematic effect on either neural sensitivity or the neuronal
correlations with behavior (DP). (A) Histogram of the neural sensitivity (left panel) and DP
(right panel) from the motion-step experiment for neurons from the strong attention group
calculated using valid trials (blue line) and invalid trials (red line). The difference in the mean
values (Δ) as well as the p-value of the t-test comparing the two distributions is shown in the top-left
of the panel. (B) Histogram of the neural sensitivity (left panel) and DP (right panel) for
neurons from the motion-step experiment for the strong attention group (black line) and the weak
attention group (grey line) calculated using valid trials. (C) Same as (A), except for the speed-
pulse experiment. (D) Same as (B), except for the speed-pulse experiment.
Figure 6 The correlation between the neural sensitivity and DP for MT neurons combined from both experiments. (A) Scatter plot of the normalized neural sensitivity and the normalized DP for validly cued (left panel, blue) and invalidly cued (right panel, red) trials in the strong attention group. For each data point from the speed-pulse experiment, the number of valid trials used to compute neural sensitivity and DP was reduced to match the corresponding number of invalidly cued trials (see text). Data from the same neurons were used to compare the valid and invalid cueing groups. (B) Scatter plot of the normalized neural sensitivity and the normalized DP for the strong attention (left panel, black) and weak attention (right panel, grey) groups calculated using all validly cued trials. Different neurons were used to compare the strong and weak attentional groups. R is the Pearson’s correlation coefficient.

Figure 7 Summary of the experimental results. (A) The relative difference of five metrics (behavioral performance, mean spike rate, neural sensitivity, DP and the correlation between neural sensitivity and DP) between valid and invalid trials for the strong attention group using data combined from both experiments. For each metric of each individual experiment, the values for valid and invalid trials were normalized by the same amount so that the value for valid trials was equal to one. (B) Same as (A), except comparing the difference between the strong and weak attention groups using only valid trials. Note that the behavioral performance in this panel refers to the ratiometric score measuring the difference in performance between valid and invalid trials as calculated in Figure 4. For each metric of each individual experiment, the values for the strong and the weak attention groups were normalized by the same amount so that the value for the strong attention group was equal to one. Error bars are SEM except for the correlation between neural sensitivity and DP, which shows the average of the two normalized correlations for the motion-step and speed-pulse experiments.
**Figure 8** Modeling the effects of attention on MT and downstream networks. (A) Schematic of our model. Both highly sensitive (filled circles) and less sensitive (open circles) MT neurons project onto downstream neurons with equal weight. A common oscillatory signal modulates firing of MT neurons. When attention is weakly focused, the oscillatory signal modulates MT firing randomly, independent of the neuron’s motion sensitivity. When attention is strongly focused, the more sensitive MT neurons are modulated strongly (thick lines), while less sensitive MT neurons are modulated weakly (thin dashed lines). (B) Neural response of downstream neuron “D” to a step stimulus. Response to the step is greater under strongly focused attention (dashed black line) compared to the response under weakly focused attention (solid grey line). (C) Distribution of neurometric and detect probability values for strongly focused attention (dashed black) or weakly focused attention (solid grey). Attention does not affect either distribution. (D) Scatter plot of neurometric versus detect probability values for all MT neurons. The two metrics become more correlated when attention is strongly focused (black, right) compared to weak attention (grey, left). See Methods for additional details of the model.
References


Figure 1
Neural sensitivity
Detect probability

Time relative to stimulus onset (ms)

Spike count

Corrected Failed Frequency

Example neuron (motion-step)

A

B Neural sensitivity

C Detect probability

Figure 2
Figure 3

A. Motion-step experiment

B. Speed-pulse experiment

C. Combined neural sensitivity and normalized DP

Figure 3
Attentional modulation of behavior

**Motion-step experiment**
- Weak vs Strong
- Valid vs Invalid

**Speed-pulse experiment**
- Weak vs Strong
- Valid vs Invalid

**C.** Within neuron comparison:
- Valid versus invalid cued trials
- Valid cue versus Invalid cue

**D.** Between neuron comparison:
- Strong versus weak attention using valid cued trials only
- Strong attention versus Weak attention

Figure 4
Figure 5
Motion-step and speed-pulse combined

A

Valid cue vs Invalid cue

Normalized DP

Normalized neural sensitivity

R = 0.50
P < 10^-5

R = -0.01
P = 0.90

B

Strong attention vs Weak attention

Normalized DP

Normalized neural sensitivity

R = 0.60
P < 10^-8

R = 0.18
P = 0.12

Figure 6
Figure 7

Motion-step and speed-pulse combined

A

Valid cue vs Invalid cue

B

Strong attention vs Weak attention

Normalized change

Behavioral performance, Mean spike rate, Neural sensitivity, DP, DP / neural sensitivity correlation
Common modulation and neuronal sensitivity are **mismatched**

Common modulation and neuronal sensitivity are **matched**

Figure 8