# The detection of visual signals by macaque frontal eye field during masking

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**The neural link between a sensory signal and its behavioral report was investigated in macaques trained to locate an intermittently detectable visual target. Neurons in the frontal eye field, an area involved in converting the outcome of visual processing into motor commands, responded at short latencies to the target stimulus whether or not the monkey reported its presence. Neural activity immediately preceding the visual response to the mask was significantly greater on hits than on misses, and was significantly greater on false alarms than on correct rejections. The results show that visual signals masked by light are not filtered out at early stages of visual processing; furthermore, the magnitude of early visual responses in prefrontal cortex predicts the behavioral report.**



Neural correlates of perceptual decisions have been identified in the visual cortex of monkeys observing weak or ambiguous stimuli[1–4]. Implicit in such studies is a behavioral report about a sensory event that allows an inference about the subject's perceptual state. To examine the link between sensation and action, we have used a signal-detection protocol to investigate visual signals in an area of the brain known to be involved in generating behavioral reports. Backward masking by light was used to create a condition in which the same physical stimulus might or might not be detected and localized. Monkeys decided whether and where to shift gaze based on variable neural responses to a visual stimulus with fixed physical properties. We recorded the visual responses of neurons located in the frontal eye field (FEF). FEF is an area located in the prefrontal cortex that is reciprocally connected with many extrastriate visual areas[5] and is central to the generation of voluntary eye movements[6]. We compared neuronal responses during correct and error trials and when the target stimulus was either present or not.

This experiment had three purposes. The first was to evaluate the hypothesis that neurons in prefrontal cortex respond only to stimuli that guide action[7,8]. Visual responses in frontal cortex are dictated more by meaning or by value than by visual features of a stimulus[6], but there has been no systematic study of frontal cortex using intermittently visible stimuli that support two alternative reports. The second purpose was to expand on earlier studies of visual cortex using ambiguous stimuli by monitoring sensory-related neural activity in an area that is involved in producing the behavioral report. Specifically, we tested the hypothesis that the representation of the visual stimulus by FEF neurons in a backward masking task is sufficient to explain the behavioral response. The third purpose was to better understand the phenomenon of backward masking by testing the proposal that responses to stimuli that are effectively masked by light do not leave the retina[9].

## Results

We analyzed responses from sixty neurons from the FEF of two *Macaca mulatta*. The results were virtually identical for the two monkeys, so they are considered together. The monkeys' task was to report the presence or absence of a dim target preceding a bright masking stimulus (**Fig. 1a**). Monkeys indicated 'yes' (target present) by shifting gaze to the target location or 'no' (target not present) by maintaining fixation on the central spot. Following the conventions of signal-detection theory[10], trials were divided into four groups based upon trial type (target present or target absent) and behavioral report (yes or no). Correctly responding yes to the target was scored a hit; incorrectly responding no was a miss. Responding yes on a target-absent trial was a false alarm; and correctly responding no, a correct rejection. Target-present trials made up 68.4% of the trials. If the monkey broke fixation from the central spot before the mask stimulus appeared, the trial was aborted and the data discarded. Of the target-present trials, 54.0% resulted in hits, 38.6% in misses, and the remaining 7.4% in target mislocalization. Of the target-absent trials, 29.8% resulted in false alarms and 70.2% in correct rejections. On any given trial, the monkey had either five or nine possible choices: to make a saccade to one of four or eight possible target locations or to maintain fixation on the central spot. Overall, the monkeys correctly reported the presence or absence of the target on 59.3% of the trials, which was above chance (20.0% for 4 targets, 11.1% for 8 targets).

The time between the appearance of the target and the appearance of the mask (the stimulus-onset asynchrony or SOA) was adjusted in a staircase before each trial according to the monkeys' performance on the previous target-present trials. Performance on no-target trials did not influence SOA adjustment. The SOA step was 16.7 ms (one video refresh). The SOA was decreased by one step after three correct trials in a row, or increased by one step after three incorrect trials in a row. The maximum SOA allowed was 66.7 ms (four video refreshes) to discourage monkeys from maintaining fixation on the central spot until the SOA reached intervals for which the target was easily detected. This approach insured that the detectability of the target was maintained near psychophysical threshold, so that on

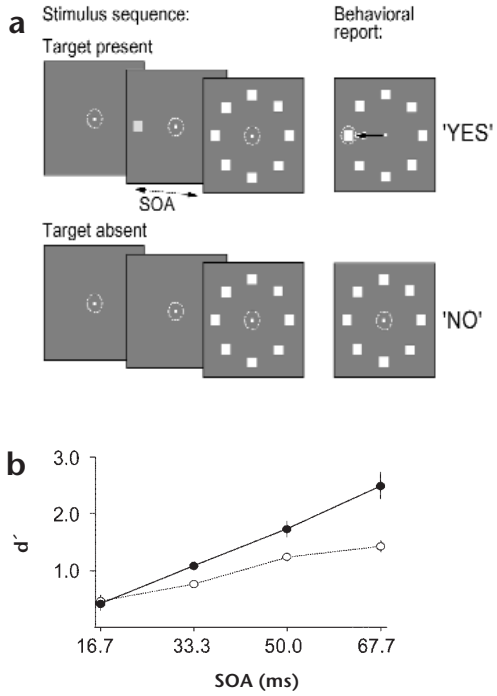


**Fig. 1.** Backward masking task and behavioral performance. **(a)** The trial began when monkeys fixated a central spot. Two possible stimulus sequences are shown. On target-present trials, a dim square target appeared at one of four or eight positions. This was followed by a bright masking stimulus consisting of white squares equal in size to the target at all of the possible target locations. On target-absent trials, only the mask stimulus was presented. The two possible behavioral reports are shown on the right. A report of yes was a saccade to the location where the target appeared. A report of no was maintenance of fixation. Dotted circle represents the current focus of gaze; arrow represents the saccade. **(b)** Performance as a function of target–mask stimulus-onset asynchrony (SOA). The measure of psychophysical sensitivity, d′, is shown for monkey A (open symbols) and monkey C (closed symbols). Error bars indicate the standard error of the mean; error bars smaller than the symbols are not plotted.

approximately half of the target-present trials, the monkeys reported the detection of the target. After the SOA converged on a level that resulted in 50% performance on target-present trials, significant changes in SOA occurred only when the monkey's behavioral response criterion changed.

The SOA staircase usually stabilized at 33.3 or 50.0 ms, but enough trials with 16.7 and 67.7 ms SOAs were obtained to calculate psychophysical d′ for each SOA. A monotonically increasing d′ as a function of SOA was observed for both monkeys (**Fig. 1b**). Guessing would result in no change in d′ with increasing SOA. Therefore, we conclude that the monkeys were using the target to guide their gaze behavior. Achievement of d′ values greater than or equal to 1.0 signifies reliable perceptual sensitivity[10].

Here the term detection refers to the monkey's behavioral report. Thus, by definition, a detected target was one that monkeys localized with a gaze shift, and an undetected target was one that monkeys failed to localize behaviorally. Of course the monkeys' willingness to report the presence of a target is an important factor. In psychophysics, this willingness to respond to the target has traditionally been referred to as the subjects' response criterion level. We discouraged guessing, that is, raised the monkeys' criterion level for detection, by including a substantial number of target-absent trials. This resulted in a high proportion of misses even though the target was present. A sufficient number of these trials was necessary to evaluate the hypothesis that neurons in prefrontal cortex respond only to stimuli that guide action.

Trials included in the analysis met the following conditions. For hits and misses, the target appeared in the neurons' receptive field. For false alarms, the errant saccade was made into the neurons' receptive field. All correct rejections were included in the analysis. There were not enough trials in which the monkeys mislocalized the target to be included in this study.

A typical visually responsive FEF neuron (**Fig. 2**) illustrates two salient observations. First, this FEF neuron responded to the undetected target on miss trials. Second, the initial visual responses were greater on trials that ended with a saccade into the neuron's receptive field, even though the physical stimulus was identical. For target-present trials, the initial visual response was greater on hits than on misses. For target-absent trials, the initial visual response was greater on false alarms than on correct rejections.

The magnitude of the initial visual responses was compared across the different trial types. First, the visual response latency to the mask stimulus was determined for the correct-rejection trials. The visual response latency to the mask stimulus was on average 58.8 ms (s.d. = 11.4 ms; range, 35–95 ms) for the neurons included in this study. For each trial, activity occurring in an interval equal in duration to the SOA and immediately preceding the mask response was measured. The premask response was measured for both target-present and target-absent trials. Because there was no SOA on target-absent trials, the premask response was measured over the same interval of time as that of the subsequent target-present trial. Thus, premask activity was measured in the same way in both target-present and target-absent trials. Activity was measured first as an average rate across all trials regardless of SOA and then separately for trials with different SOAs. The same pattern of results was obtained for individual or for combined SOAs (compare **Fig. 2** with **Figs. 3** and **4**). For simplicity, we report results of the analyses when all trials, regardless of SOA, are combined.

To test whether undetected targets evoke a response in prefrontal cortex, we compared the premask activity on misses to the premask activity on correct rejections. In both cases, the monkeys reported that the target was not present. The comparison between the groups of trials was made by calculating a modulation index, which was the difference of the average activity in the two groups of trials divided by their sum; this value can range from –1.0 to 1.0, with a value of 0 resulting when the activity in the two conditions is identical. The premask response was greater on misses than on correct rejections for 97% (58 of 60) of the neurons (**Fig. 3a**). The mean modulation index was 0.36, and the distribution was significantly greater than 0.0 ($t_{59} = 11.7$, $p < 0.001$). This means that a significant visual response in prefrontal cortex to a target does not necessarily lead to the detection of that target.

To determine if the visual responses in FEF predicted the behavioral report, we compared the premask activity on trials with identical visual stimulation but with opposite reports by the monkey, that is, hits and misses. We found that a detected target evoked a greater premask response than did an undetected target in 95% (57 of 60) of the neurons (**Fig. 3b**). The mean modulation index was 0.14, and the distribution was significantly greater than 0.0 ($t_{59} = 8.9$, $p < 0.001$).

We also compared the activity on trials with no target that resulted in opposite behavioral reports, that is, false alarms and correct rejections. Only 45 of the neurons were recorded during enough false-alarm trials to make this comparison. The premask response was greater on false-alarm trials than on correct-rejection trials for 89% (40 of 45) of these neurons (**Fig. 3c**). The mean modulation index was 0.17, and the distri-

bution was significantly greater than 0.0 ($t_{44} = 4.6$, $p < 0.001$).

For some neurons, we noticed that the level of activity before the target appeared was slightly higher when the monkey reported the target to be present, regardless of whether it actually appeared (**Fig. 2**). For each neuron, the pretarget activity on hits and false alarms was combined to obtain the average pretarget activity on 'yes' trials. Likewise, the pretarget activity on misses and correct rejections was combined to obtain the average pretarget activity on 'no' trials. The distribution of pretarget modulation indices comparing the 'yes' and 'no' trials in which the target or the endpoint of the errant saccade was inside the neurons' receptive field was significantly greater than 0.0 (mean, 0.10; $t_{59} = 4.2$, $p < 0.001$; **Fig. 3d**). However, the distribution of pretarget modulation indices comparing 'yes' and 'no' trials on which the target or the errant saccade was outside the neurons' receptive was not different from 0.0 (mean, –0.009; $t_{59} = -0.6$; **Fig. 3e**). Because there was no target or errant saccade on correct rejections, they were included with the 'no' trials for comparisons of inside the receptive field trials and outside the receptive field trials.

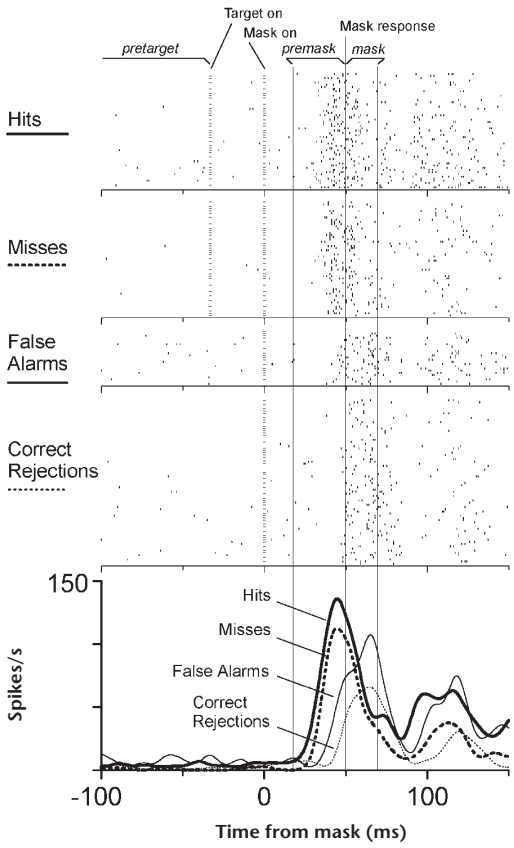


**Fig. 2.** Responses of a visual neuron in the frontal eye field during the backward-masking task. Raster plots of neural activity are shown for hits, misses, false alarms and correct rejections. Each small tickmark indicates the time of an action potential; each row represents activity from a different trial. The activity is aligned on the time of mask presentation, which is indicated by a horizontal tickmark and labeled 'Mask on' at the top of the plot. For hits and misses, the time of target presentation is indicated by the horizontal tickmark labeled 'Target on'. Only trials with an SOA of 33.3 ms are shown. For hits, misses and correct rejections. All false-alarm trials are shown. Intervals in which pretarget, premask and mask activity were measured are indicated across the top. The vertical line at 49 ms is the average visual response latency to the mask stimulus, which separates the premask interval from the mask interval. At the bottom are the average spike-density functions obtained from each of the trial groups. Solid lines, activity during trials with yes responses. Dotted lines, activity during trials with no responses. Thick lines, activity during target-present trials. Thin lines, activity during target-absent trials. The average spike density was obtained by convolving each spike train with a gaussian filter (s.d. = 5 ms).

For the trials in which the target or the errant saccade was in the receptive field, we determined whether the premask response was correlated with the pretarget activity for each neuron. The pretarget and premask modulation indices were not correlated for hits versus misses ($r = 0.03$), but they were for false alarms versus correct rejections ($r = 0.54$, $p < 0.001$). A logistic regression determined the relative contributions of pretarget and premask activity to the monkeys' decision. Significant regressions including both the pretarget and premask activity were obtained from target-present trials ($p = 0.036$) and target-absent trials ($p = 0.0006$). However, if premask activity is included in the regression, then pretarget activity only contributed significantly in distinguishing false alarms from correct rejections (pretarget term, $p = 0.0065$; premask term, $p = 0.0018$) but not in distinguishing hits from misses (pretarget term, $p = 0.36$; premask term $p = 0.02$).

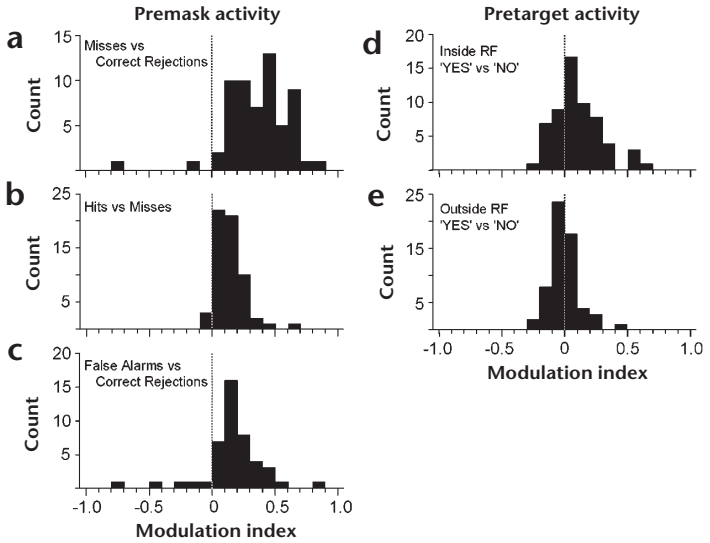


**Fig. 3.** Contrast of responses for different conditions. Distributions of modulation indices comparing **(a)** premask activity on misses and correct rejections, **(b)** premask activity on hits and misses, **(c)** premask activity on false alarms and correct rejections, **(d)** pretarget activity on trials resulting in a 'yes' report and those resulting in a 'no' report for which the target or errant saccade was inside the neurons' receptive field, and **(e)** pretarget activity on trials resulting in a 'yes' report and those resulting in a 'no' report for which the target or errant saccade was outside the neurons' receptive field. Each neuron contributed one data point to each distribution. The modulation index was calculated by subtracting the average activation rate obtained from the second trial type from that obtained from the first and dividing by their sum. The modulation indices range from –1 to 1. Positive values indicate that the activity of the first type of trials listed was greater; negative values indicate that the activity of the second group was greater.

The analyses to this point have compared the activity of single cells in one group of trials with that in another group of trials. To examine the signals at the population level, we compared activity from all neurons recorded in all conditions. It was necessary first to normalize activity so comparisons could be made across neurons with different firing rates. For 45 of the 60 neurons, enough data was obtained from all four types of trials to be included in this analysis. To normalize firing rates across neurons, the mask response was first averaged across hits, misses, false alarms and correct rejections, each providing one value. The mask response was the average firing rate in the 20 ms following the visual response latency to the mask stimulus. Next, the average mask response of each neuron was scaled to a value of 100, and all measures of activity for that neuron were adjusted according to the scaling factor (**Fig. 4**).

The Friedman test was used to identify response groups (hits, misses, false alarms and correct rejections) that differed significantly in normalized activity distribution within each time interval (**Table 1**). During the pretarget interval, only the miss and false-alarm distributions differed significantly. During the premask interval, each group differed significantly from all the other groups. During the mask interval, activity was significantly higher on hits than on misses or on correct rejections. The higher mask activity on hits may be a carryover from the higher premask activity or may be due to a saccade target-selection response that is evident after about 100 ms following stimulus presentation for many visually responsive FEF neurons[11]. Neither mask activity on hit and false-alarm trials nor activity on miss and correct-rejection trials differed significantly. Of the three intervals, activity during the premask interval most reliably discriminated among the four trial types.

**Table 1. Means of the normalized activity distributions shown in Fig. 4.**

| Trial interval | Correct rejections | False alarms | Misses | Hits |
|---|---|---|---|---|
| Pretarget | 22.5 | 26.8* | 20.7* | 23.8 |
| Premask | 30.0* | 44.9* | 67.6* | 87.2* |
| Mask | 93.9+ | 97.3 | 99.9* | 109.0*+ |

An asterisk (*) or cross (+) denotes significant difference ($p < 0.05$) from the other trial types marked with the same symbol within the same trial interval (Friedman test).

## Discussion

### Relation to previous studies of masking

Previous studies have reported neural correlates of backward masking in the dorsal lateral geniculate nucleus and visual cortex by showing that stimuli presented shortly before the mask elicit weaker sensory responses as compared to stimuli presented earlier before mask presentation or to stimuli that are not masked[12–18]. These studies led to the view that the critical neural events for visual masking by light occur in the retina[9,13,17]. Our finding of significant visual responses in prefrontal cortex to undetected masked stimuli challenges this view.

The present study differs from earlier studies of masking in two important ways. First, whereas all previous studies only inferred the relation between a perceptual judgement and the visual responses to masked stimuli, we directly correlated the responses of neurons with whether or not the target survived the mask as indexed by a behavioral report. Second, whereas all previous studies recorded neural responses in subcortical or cortical visual structures, we recorded neural activity in an area of prefrontal cortex that responds to visual stimuli and also is necessary for the production of purposive eye movements[6].

### Nature of the visual responses in FEF

Previous neurophysiological studies using bistable[1–3] or masked stimuli[12–18] or attention protocols[19,20] have indicated that visual signals become more strongly correlated with inferred perceptual state higher in the visual-area hierarchy. Other studies have shown that visual responses in frontal cortex are related more to the instructional meaning or reward value than to the visual properties of the stimulus[21–23]. As a result, it has been proposed that prefrontal cortex only registers sensory activity that reaches awareness to guide voluntary behavior[7,8]. We now show that responses of neurons in prefrontal cortex to a visual stimulus *per se* did not necessarily lead to a report of the detection of that stimulus. Instead, detection of the target was reported only on those trials in which the initial visual activity was slightly stronger than otherwise.

We do not think that the slightly greater visual response leading to detection of a target can be explained by motor preparation because the response to the masked target occurred at too short a latency. The latencies of visual responses in FEF coincide with the latencies of visual responses in dorsal stream visual areas such as MT and MST and follow only shortly after the earliest visual responses in V1 (refs. 24, 25) Such short-latency visual responses in FEF are most likely carried by direct afferents from areas MT, MST and posterior parietal cortex[5]. The differential premask responses that we observed occurred well before the onset of activity in FEF associated with movement preparation[26]. The premask response also occurs before the onset of the target-selection process that we have reported before[11]; the differential activ-

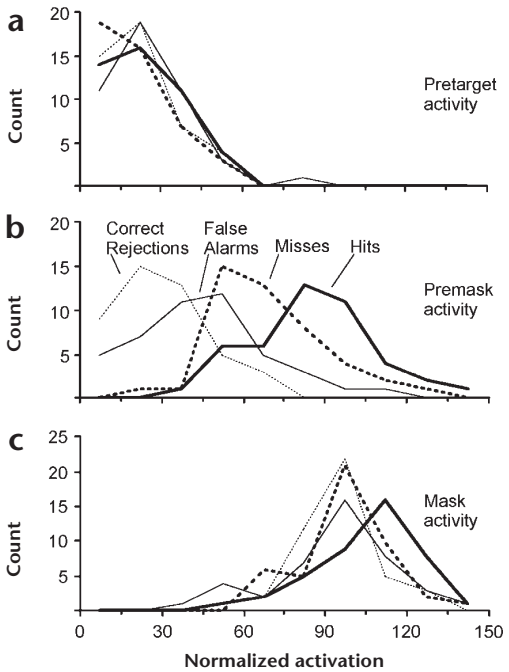


**Fig. 4.** Frequency of normalized firing rates for 45 FEF neurons. **(a)** Pretarget activity. **(b)** Premask activity. **(c)** Mask activity. Line types follow the same conventions as spike density plots in Fig. 2. Each neuron contributes one data point to each distribution. Binwidth, 15.

ity after 100 ms in Fig. 2 is probably related to target selection.

We also do not think that the slightly greater visual response when the monkeys detected the target can be explained entirely by the saccade-related enhancement phenomenon[27,28]. Visual enhancement occurs when monkeys predict the location of the target for a saccade. However, the higher activation that we observed on hit and false-alarm trials occurred without the monkey's knowledge of where or even whether the target would appear on any given trial. One may still argue that the monkeys were preselecting a location for a saccade before a trial, but the behavioral evidence argues against this because errant saccades were rare—only 7% of all trials resulted in mislocalized targets, and 30% of trials in false alarms. Furthermore, the increase in d′ with increasing SOA indicates that the monkeys were using the target stimulus to guide their behavior. In addition, the lack of a pretarget bias in neural activity on trials in which the target or errant saccade was outside the neurons' receptive field indicates that there was not a nonspecific enhancement related to the monkeys' intent to make a saccade on one trial versus another.

We suspect it is unlikely that the differential activity in the premask period arises *de novo* in FEF. Most likely, the differences observed across the different trial groups in the premask activity reflect variations in visual activation in earlier stages of the visual pathway. Some of this variation is probably random fluctuation, but there may be some degree of systematic adjustment related to the monkeys' performance. The slightly greater pretarget activity we observed in some neurons before hits and false alarms may reflect recent choice and reward history. The existence of an occasional pretarget bias is consistent with an earlier study reporting slow variations in neural responsiveness that predict the magnitude of an evoked response in visual cortex[29]. However, when the target was presented, the premask activity was not correlated with the pretarget activity. Moreover, the premask activity was a much better predictor of the monkeys' choice than was the pretarget activity, especially on trials when the target appeared.

Regardless of the origin of the pretarget and premask activation differences, this study shows that opposite reports on physically identical trials by monkeys performing at psychophysical threshold arise from small differences in neural activation. According to signal-detection theory, subjects in this type of experiment make a decision based on an internal representation of some attribute of the stimulus[10]. The analysis shown in Fig. 4 indicates that during backward masking, the most information for the decision occurs in the premask response. Because this interval is so brief, it constrains theories of what the neural decision variable can be. Further studies are needed to determine whether a simple criterion threshold is sufficient or whether a more complex coding mechanism is required to explain the relationship of these brief visual responses to the behavioral report.

## Methods

Data were collected from two *Macaca mulatta* (8 and 10 kg). Described[11,30] methods conformed to NIH guidelines.

**Task.** Using operant conditioning with a juice reward, monkeys were trained to shift gaze to a dim target presented alone or followed by a bright mask. The target presented without the mask stimulus was used to classify neurons and to map receptive fields. The mapping task and the visual masking task were run in separate blocks of trials. The sequence of the masking trials is illustrated in Fig. 1a. Each trial began with the appearance of a fixation spot at the screen center. After the monkey fixated the spot for 500–700 ms, a dim target usually appeared at one of four or eight possible locations around the fixation spot. On average, no target appeared in 32.6% of trials (the percentage of target-absent trials ranged from 16% to 48% across sessions). At a short interval following the appearance of the target, a bright mask stimulus equal in size to the target appeared at every possible target location. The interval between the appearance of the target and the appearance of the mask was called the stimulus-onset asynchrony (SOA). Monkeys were rewarded for correctly reporting whether or not the target was present; absence of a reward provided error feedback. Monkeys indicated yes (target present) by shifting gaze to the target location within 500 ms of target presentation, or no (target not present) by maintaining fixation on the central spot for at least 750 ms following target presentation. Single trials were scored according to the conventions of signal-detection theory as either hits (correct yes responses), misses (incorrect no responses), correct rejections (correct no responses) or false alarms (incorrect yes responses)[10].

The measure of perceptual sensitivity d′ (**Fig. 1b**) was calculated for each SOA. Target-absent trials were assigned the SOA of the following target-present trial. Any tendency to make inadvertent saccades to mask stimuli without detection of a target must be accounted for in both the hit rate and the false-alarm rate. Therefore, all saccades were scored as a yes. Although mislocalized target trials were rare (7%), these were scored as a hit for the calculation of d′.

Stimuli were presented on a video monitor (Mitsubishi XC-3315C, 60 Hz, noninterlaced) under computer control (PDP 11/83). The fixation spot subtended 0.3° of visual angle. The stimuli were presented on a dark gray background (0.1 cd/m$^2$). The target stimulus was a dim blue square (CIE x = 161, y = 73; Y = 0.3 cd/m$^2$). The mask stimuli were bright white squares (CIE x = 276, y = 284; Y = 83.0 cd/m$^2$). To provide approximately equal visibility across the eccentricities tested, the sizes of stimuli were scaled linearly from 0.5° wide at 7° eccentricity to 1.5° at 15° eccentricity, in proportion to cortical magnification.

**Neuronal activity analysis.** The analysis of each neuron involved several steps. Initially, the visual response latency to the mask stimulus on correct target-absent trials was determined using a Poisson spike train analysis[11,31]. Then spikes were counted during the three time intervals indicated in Fig. 2. The first, termed the pretarget interval, was the 100 ms before target presentation on target-present trials or the corresponding interval on target-absent trials. The second, termed the premask interval, equal in duration to the SOA, began at the visual latency following target presentation and ended at the time of the visual response latency to the mask. For target-absent trials, the premask interval was identical to the target-present trial that immediately followed. The third, termed the mask interval, was the first 20 ms of the response to the mask stimulus following the visual response latency to the mask stimulus.

Neuronal activity was measured as the sum of the spikes counted across all trials divided by the total amount of time summed across all trials in which spikes were counted. For each neuron, the average firing rate was calculated in this manner separately for each of the three intervals (pretarget, premask and mask) and for each of the trial types (hits, misses, false alarms and correct rejections). Average activation was calculated for all trials combined and for trials separated according to SOA. Any group with fewer than three trials was not included in the analysis.

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