Effects of microsaccades on contrast detection and V1 responses in macaques

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Microsaccades can elevate contrast detection thresholds of human observers and modulate the activity of neurons in monkey visual cortex. Whether microsaccades elevate contrast detection thresholds in monkey observers is not known and bears on the interpretation of neurophysiological experiments. To answer this question, we trained two monkeys to perform a 2AFC contrast detection task. Performance was worse on trials in which a microsaccade occurred during the stimulus presentation. The magnitude of the effect was modest (threshold changes of <0.2 log unit) and color specific: achromatic sensitivity was impaired, but red–green sensitivity was not. To explore the neural basis of this effect, we recorded the responses of individual V1 neurons to a white noise stimulus. Microsaccades produced a suppression of spiking activity followed by an excitatory rebound that was similar for L – M cone-opponent and L + M nonopponent V1 neurons. We conclude that microsaccades in the monkey increase luminance contrast detection thresholds and modulate the spiking activity of V1 neurons, but the luminance specificity of the behavioral suppression is likely implemented downstream of V1.

Keywords: color vision, visual cortex, eye movements, contrast sensitivity, electrophysiology

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Introduction

During free viewing, and even nominal fixation, humans and monkeys make several saccades per second. Each saccade shifts the pattern of light falling on the retina, but the visual world remains perceptually stable. This stability is thought to be due in part to saccadic suppression, which is a reduction in visual sensitivity around the time of saccades (for a review, see Matin, 1974).

Saccadic suppression is profound during large amplitude saccades but is subtle or absent during microsaccades, which are small, involuntary saccades that occur naturally during fixation. Depending on the stimulus conditions and behavioral task, microsaccades can decrease visual sensitivity (Beeler, 1967; Ditchburn, 1955; Riggs, Ratliff, Cornsweet, & Cornsweet, 1953; Zuber & Stark, 1966), increase visual sensitivity (Deubel & Elsner, 1986; Kelly, 1990; Martinez-Conde, Macknik, Troncoso, & Dyar, 2006; Rucci & Desbordes, 2003), or exert no effect (Krauskopf, Graf, & Gaarder, 1966) in human observers. These disparate observations can be understood as combinations of top-down suppressive signals (related to corollary discharge), bottom-up suppressive signals (e.g., blur and visual masking), and bottom-up facilitatory signals (e.g., relief from image fading).

We know less about the effects of microsaccades on the vision of macaque monkeys, a popular animal model in studies of visual neurophysiology. Understanding how microsaccades affect the vision of this animal is important for interpreting the results of neurophysiological experiments (e.g., Chen, Geisler, & Seidemann, 2006, 2008; Geisler & Albrecht, 1997; Harwerth, Smith, & DeSantis, 1993; Palmer, Cheng, & Seidemann, 2007). For example, microsaccades made by monkeys performing visual motion detection tasks exert two (possibly related) effects: they increase thresholds for changes in visual motion and suppress the responses of visual motion-sensitive neurons (Herrington et al., 2009). Together, these effects contribute to a correlation between neural activity and behavioral responses. We sought to determine whether microsaccades also influence contrast detection.

Large amplitude saccades (~20°) increase detection thresholds in humans for luminance but not chromatic contrast (Burr & Morrone, 1996; Burr, Morrone, & Ross, 1994; Diamond, Ross, & Morrone, 2000; Uchikawa & Sato, 1995). This specificity has motivated the hypothesis that the magnocellular visual pathway is suppressed selectively during saccades. Electrophysiological tests of this hypothesis have yielded mixed results (Ramcharan, Gnadt, & Sherman, 2001; Reppas, Usrey, & Reid, 2002; Royal, Sary, Schall, & Casagrande, 2006). An alternative possibility is that mechanisms responsible for the lumi-

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nance specificity of saccadic suppression act on cone-nonopponent neurons at a higher level of the visual system. To address this possibility, we asked whether cone-opponent and nonopponent neurons in area V1, the next stage of visual processing, are differentially modulated by microsaccades.

Methods

Two monkeys (*M. mulatta*) participated in the experiments. All procedures conformed to the guidelines provided by the NIH and the University of Washington Animal Care and Use Committee. Each monkey was implanted with a titanium head post to eliminate head movements and a monocular scleral search coil to measure eye position (Judge, Richmond, & Chu, 1980). During experimental sessions, the monkey sat in a primate chair 100 cm from a cathode ray tube (CRT) monitor in an otherwise dark room. Eye position signals were low-pass filtered with an 8-pole Bessel filter (180-Hz high-frequency cutoff), digitized at 1 kHz, and stored to disk at 500 Hz. Event timing and eye position monitoring were mediated by a PC running the REX software package (NIH). Data were acquired for offline analysis with a Plexon MAP system (Plexon). Single unit recordings were made with extracellular glass-insulated transdural tungsten electrodes with impedances ranging from 1 to 2 MΩ (Frederick Haer). Neural signals were amplified and filtered with standard techniques, and spikes were isolated offline on the basis of waveform timing, shape, and amplitude criteria.

Monkeys were trained to perform the two-alternative forced choice (2AFC) contrast detection task shown in Figure 1A. Each trial began when the monkey acquired a central fixation point. Three hundred milliseconds later, a pair of black 3.6 × 3.6° square frames appeared, one 5.0° to the right and 3.5° above the fixation point and the other 5.0° to the left and 3.5° below the fixation point. Two hundred milliseconds later, a horizontally oriented Gabor stimulus appeared inside one square for 667 ms. The monkeys' task was to identify the square in which the Gabor stimulus appeared.

Monkeys indicated the location of the stimulus by making a saccadic eye movement. At a random time 100–600 ms after the stimulus presentation, the fixation point disappeared and a pair of saccade targets appeared simultaneously. A saccade to the target in the direction of the Gabor stimulus was a correct response and was reinforced with a juice reward. Feedback was not provided following incorrect responses. In most experiments, saccade targets appeared 3° from the fixation point, but in a few experiments, they were positioned asymmetrically to mitigate choice biases. Trials were aborted whenever the monkeys' eye position prematurely left a 1 × 1° electronically defined window around the fixation point.

Microsaccade detection

Microsaccades were detected using an algorithm used previously by Horwitz and Albright (2003). Eye velocity was computed by filtering horizontal and vertical eye position records with a smoothed differencing operator (a differencing operator convolved with a Gaussian of 4 ms standard deviation). A microsaccade was identified as a deflection of the eye during which eye speed (the vector norm of the horizontal and vertical velocities) exceeded 10°/s for at least 8 consecutive ms. Pairs of microsaccades occurring within 40 ms of each other were considered a single movement. Visual inspection of eye position records confirmed that the movements identified by the algorithm appeared to be microsaccades.

Stimuli: Psychophysics

Stimuli were presented on a CRT monitor (Sony Trinitron) whose phosphor emission spectra and voltage–intensity relationships had been characterized with a spectroradiometer (PR650, PhotoResearch). A digital video signal processor (Bits++, Cambridge Research) increased the depth of each color channel from 8 to 14 bits. This increase was brought at the expense of spatial resolution; each pixel in the display was twice as wide as it was tall. Stimuli were generated using the Psychophysics Toolbox (Brainard, 1997) for Matlab on a Mac Pro computer. The background of the screen was uniform gray (CIE coordinates: $x = 0.33$, $y = 0.33$, $Y = 90$ cd/m^2).

The Gabor stimulus had a standard deviation of 0.8° and was truncated beyond 2 standard deviations. The sinusoidal component drifted upward at 3 Hz and had a spatial frequency of 0.25, 0.89, or 3.22 cycles/degree. These spatial frequencies were selected to span the range over which saccadic suppression occurs (Burr et al., 1994) without extending to yet lower spatial frequencies that are unobtainable given the Gaussian envelope we used. To avoid detectable visual transients, the contrast of the Gabor was modulated according to a trapezoidal temporal envelope; the contrast ramped up linearly over the first half-cycle of the stimulus (167 ms), remained constant over the next cycle (333 ms), and ramped down linearly over the last half-cycle (167 ms).

The stimulus modulated in two color directions based on the Stockman, MacLeod, and Johnson 2° cone fundamentals (Stockman, MacLeod, & Johnson, 1993). The achromatic stimulus modulated each of the three cone types with identical cone contrast. The red–green stimulus modulated the L- and M-cones with identical contrast but in opposite phase. Stimulus contrast was determined on each trial by the QUEST adaptive staircase procedure (Watson & Pelli, 1983). The QUEST procedure decrements the contrast following correct trials and increments the contrast following incorrect trials, thereby ensuring that the stimulus remains near detection threshold

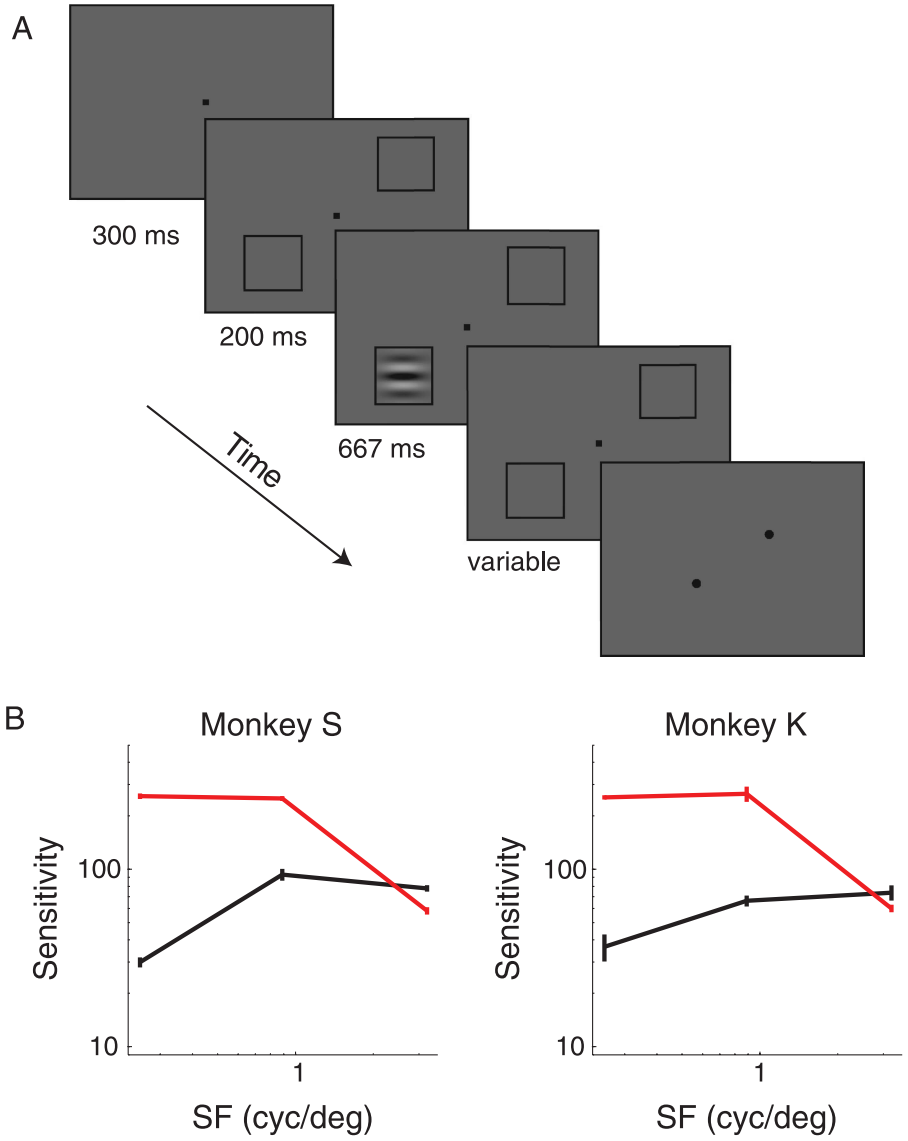


Figure 1. (A) Ordered screenshots from the 2AFC contrast detection task. See text for details of the experimental methods. (B) Contrast sensitivity of two monkeys as a function of color direction and spatial frequency. Stimuli varied in red–green contrast (red curve) and achromatic (luminance) contrast (black curve). Sensitivity is quantified as the reciprocal of the detection threshold, as estimated by the QUEST adaptive procedure (Watson & Pelli, 1983). Throughout this report, contrast is represented in units of cone-contrast vector lengths $\sqrt{\left(\frac{\Delta L}{L}\right)^2 + \left(\frac{\Delta M}{M}\right)^2 + \left(\frac{\Delta S}{S}\right)^2}$. A sensitivity of "1" thus represents a contrast of 0.58 to each cone type in the achromatic condition and a contrast of 0.71 to the L- and M-cones in the red–green condition. Error bars are ±1 *SEM*.

throughout the experiment. The first 10 trials of each staircase were omitted from the analysis. In each experimental session, at least six independent staircases were randomly interleaved: one for each combination of color and spatial frequency (except for 5 sessions with Monkey K in which the 0.89-cpd stimulus did not appear).

Stimuli: Neurophysiology

We stimulated most of the V1 neurons we studied with a dynamic white noise stimulus. The stimulus was a 10 × 10 square grid of 0.1 × 0.1° elements whose colors changed synchronously and independently at 75 Hz. The color of each stimulus element was determined by independent Gaussian draws from red, green, and blue monitor phosphor intensity distributions. This stimulus modulates in chromaticity and luminance, and it modulates the spiking activity of cone-opponent and non-opponent neurons. The cone weights of V1 neurons stimulated in this way can be estimated through an analysis of the spike-triggered average stimulus (STA; Horwitz, Chichilnisky, & Albright, 2007). We studied the responses of a smaller set of neurons to the Gabor stimulus during performance of the contrast detection task. In these experiments, the orientation and spatial

frequency of the Gabor stimulus was tailored to the preferences of the neuron under study.

Statistics

Contrast detection thresholds in Figure 1B were obtained from the modal values of the QUEST function at the end of each session (Watson & Pelli, 1983). This approach cannot be used to measure detection thresholds separately for trials on which a microsaccade occurred or did not occur during the stimulus presentation; QUEST does not maintain a separate staircase for these two types of trials. Instead, we quantified changes in detection threshold due to microsaccades by fitting the data from each stimulus condition with a cumulative Weibull function:

$$y = 1 - 0.5e^{-\left(\frac{x}{\beta_0 + \beta_1 I_{sac}}\right)^{\beta_2}}, \quad (1)$$

where y is a binary variable that equals 1 for correct trials and 0 for incorrect trials, x is the cone contrast of the stimulus (quantified as the length of the vector of cone contrasts), and I_{sac} is a binary variable that equals 1 for *saccade* trials and 0 for *no saccade* trials. The β_i are parameters fitted by maximum likelihood assuming binomially distributed errors. β_0 is the threshold on *no saccade* trials, $\beta_0 + \beta_1$ is the threshold on *saccade* trials, and β_2 is a slope parameter. The change in threshold due to microsaccades in log units is

$$\log_{10}\left(\frac{\beta_0 + \beta_1}{\beta_0}\right). \quad (2)$$

Statistical significance of threshold changes was assessed by comparing the fit of the full model (Equation 1) with the fit of the nested model in which β_1 was constrained to be 0 (analysis of deviance, McCullagh & Nelder, 1989).

We used a nonparametric bootstrap procedure to test whether microsaccade vectors predicted task performance. Microsaccade vectors (direction and amplitude) preceding correct and incorrect responses were sorted into separate groups and the Euclidean distance between the centroids (mean vectors) of each group was recorded. Vectors were then resampled with replacement from the empirical distribution (the union of vectors from correct and incorrect trials binned at $0.1 \times 0.1°$), randomly reassigned to "correct" and "incorrect" groups, and the distance between centroids was recalculated. This procedure was repeated 10,000 times to estimate the distribution of intercentroid distances under the null hypothesis. The fraction of intercentroid distances from the resampled data sets that exceeded the intercentroid distance from the original data set is the p-value reported. The same procedure was used to test the hypothesis that microsaccade vectors depended on the target choice, but in this analysis vectors were grouped by which target the monkey chose at the end of the trial.

We calculated microsaccade-triggered spike-density functions for 21 V1 neurons during the detection of low contrast, red–green Gabor stimuli. These functions show changes in firing rate that are time-locked to microsaccades. To measure changes in firing rate that were not time-locked to microsaccades, we also performed a control version of this analysis in which we randomly permuted the association between the times of microsaccades and spikes across trials. These permutations preserve the temporal statistics of microsaccades and spikes but break any relationship between them. In this control analysis, we constructed 200 randomly permuted data sets for each neuron, calculated a spike-density function from each data set, normalized each function by dividing it by its maximum, and averaged the 200 normalized spike-density functions together.

Results

Monkey K performed 14,577 trials of the 2AFC detection task and Monkey S performed 22,452 trials. Monkey K made 9,397 microsaccades during task performance and Monkey S made 35,996 microsaccades. Below, we summarize the metrics and timing of microsaccades and show that they are associated with increases in achromatic, but not red–green, detection thresholds. We then show that the responses of cone-opponent and nonopponent neurons in V1 are modulated similarly by microsaccades, suggesting that the color specificity of microsaccadic suppression originates downstream of V1.

Detection performance

Figure 1B shows contrast sensitivity for the 2 monkeys as a function of color direction and spatial frequency. The monkeys, like human observers, exhibited low-pass sensitivity for chromatic stimuli and attenuated sensitivity for low-frequency achromatic stimuli (Mullen, 1985). The monkeys were slightly more sensitive than a human observer viewing the identical display (Hass & Horwitz, 2010). These observations demonstrate that the monkeys' behavior was under stimulus control.

Microsaccades

Monkeys made frequent microsaccades while performing the 2AFC detection task. The movements we analyzed were necessarily small (mean amplitudes for Monkeys K

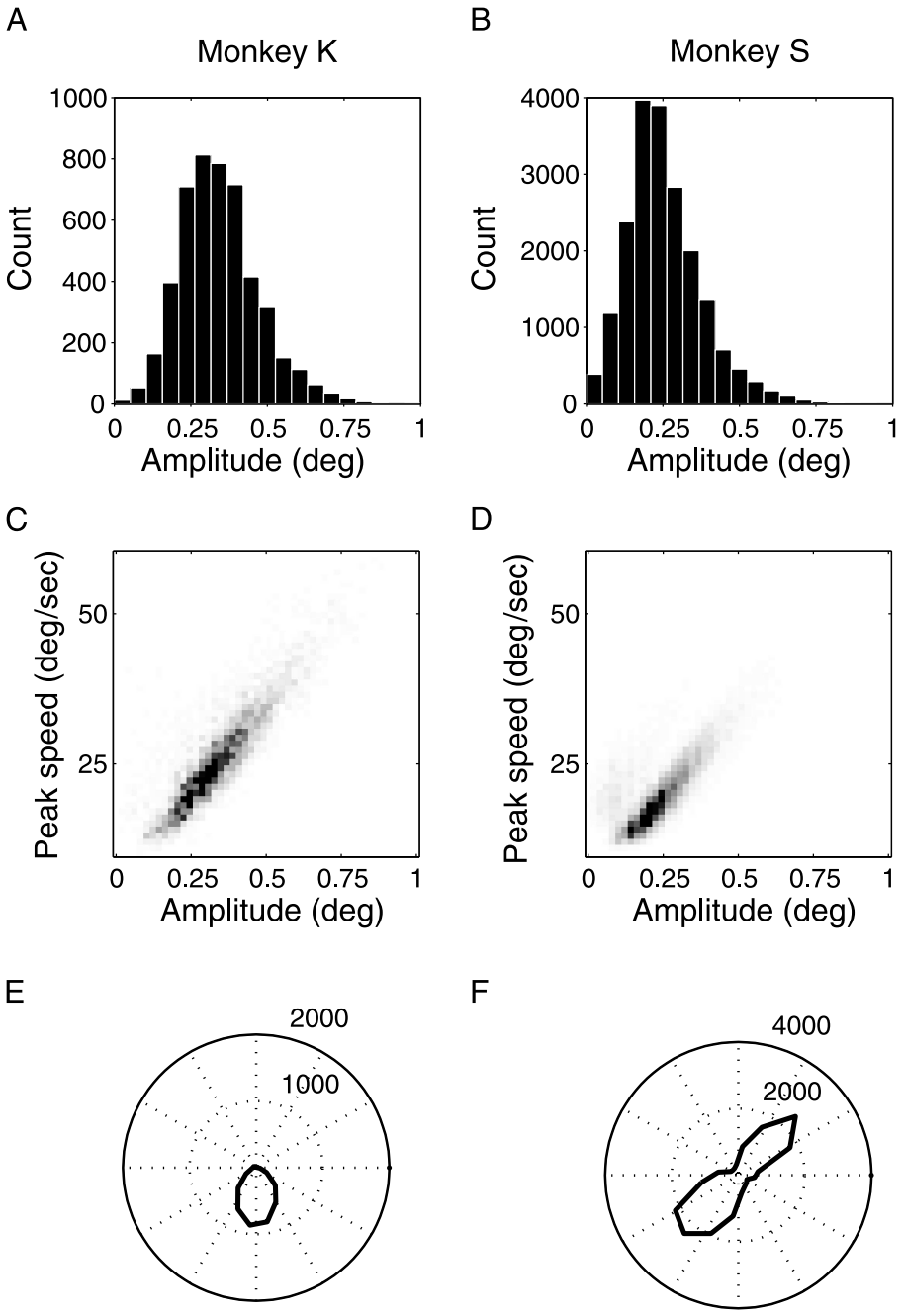


Figure 2. Metrics of microsaccades made during performance of the contrast detection task. (A, B) Histogram of microsaccade amplitudes. (C, D) Microsaccade peak speed vs. amplitude (saccadic main sequence). (E, F) Polar histograms of saccade directions.

and S were 0.34° and 0.25°, respectively; see Figures 2A and 2B) because movements that caused the eye to leave the fixation window aborted trials. The relationship between amplitude and speed shown in Figures 2C and 2D follows the "main sequence" and is consistent with the classification of these movements as saccades (Zuber & Stark, 1965). Monkey K tended to make saccades with a downward component (Figure 2E), whereas Monkey S tended to make saccades along the axis of the stimulus locations (Figure 2F).

Figures 3A and 3B show the temporal distribution of microsaccades across the trial. For both monkeys, microsaccade rates peaked around the time of the onset of the Gabor stimulus. This peak is not due to any detectable visual event occurring at this time; the stimulus contrast ramped up gradually over the course of 167 ms and remained close to detection threshold. Omitting the square frames from the display eliminated these peaks (see dashed curves in Figures 3A and 3B) suggesting that they might represent a brief release from microsaccadic

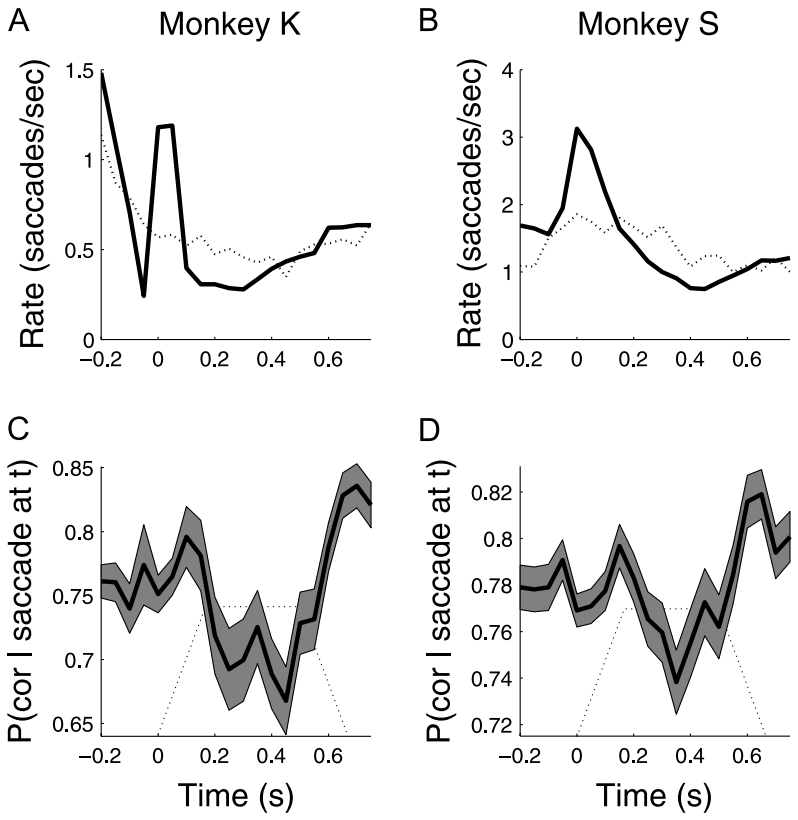


Figure 3. Time course of microsaccade occurrence and relationship to task performance. (A, B) Peri-stimulus time histogram of microsaccade initiations. Data are aligned on stimulus onset ($t = 0$). Dotted curves show microsaccade initiations in a set of control trials in which the frames did not appear. (C, D) Proportion of correct choices as a function of when a microsaccade was made. Shaded area represents ±1 standard error of binomial proportion. Dotted line indicates time course of stimulus presentation. *Y*-axis position of dotted line in (C) and (D) is arbitrary. Note the different *y*-axis scales in (A) and (B) and in (C) and (D).

inhibition associated with the change in the visual display (Engbert & Kliegl, 2003; Rolfs, Kliegl, & Engbert, 2008). Microsaccade rates during the stimulus presentation were low for both monkeys (Monkey K: 0.5 saccade/s, Monkey S: 1.2 saccades/s), suggesting that microsaccades are actively suppressed during this time.

Relationship between microsaccades and task performance

Suppressing microsaccades would be adaptive if they impair contrast detection. To explore this possibility, we compared performance on trials that had at least one microsaccade during the plateau phase of the stimulus (*saccade* trials) to those lacking microsaccades during this interval (*no saccade* trials). *No saccade* trials outnumbered *saccade* trials; the percentage of *no saccade* trials was 88% for Monkey K and 68% for Monkey S. Consistent with the idea that microsaccades impair contrast detection, the percentage of correct *saccade* trials was lower than the percentage of correct *no saccade* trials (Monkey K: 70 vs. 76%, $p < 0.01$; Monkey S: 76 vs. 78%, $p < 0.05$, z-tests of differences in binomial proportions). This result could be trivial if the monkeys made more microsaccades on low contrast trials than on high contrast trials, but this was not the case; mean stimulus contrast was actually higher on *saccade* trials than on *no saccade* trials in 4 out of 6 conditions for Monkey K and in 6 out of 6 conditions for Monkey S.

The preceding analysis leaves open the possibility that microsaccades during trial epochs other than the stimulus plateau were also related to task performance. To measure the time course of the relationship between microsaccades and percent correct, we divided each trial into non-overlapping 100-ms bins. Within each bin, the proportion of correct trials was calculated across the subset of trials containing a microsaccade in that bin. As shown in Figures 3C and 3D, a dip in performance is apparent around $t = 0.3$ for both monkeys. Thus, poor performance is associated specifically with microsaccades during the stimulus plateau and not other times during the trial. The improvement in performance associated with microsaccades made late in the trial ($t > 0.6$ in Figures 3C and 3D) will be discussed in the Relationship between microsaccade metrics and choices section.

Influence of color and spatial frequency

Large amplitude saccades preferentially impair the detection of low spatial frequency, achromatic patterns (Burr et al., 1994). We asked whether microsaccadic suppression in our experiment had similar stimulus specificity. For each of the 6 conditions (2 colors × 3 spatial frequencies), we compared percent correct between *no saccade* and *saccade* trials. Both monkeys had a significantly higher percent correct on *no saccade* trials than on *saccade* trials when the stimulus was achromatic and had a spatial frequency ≥0.89 cpd (z-tests of differences in binomial proportions, $p < 0.05$; Figures 4A and 4B, black bars). For Monkey K, but not Monkey S, this detrimental effect of microsaccades extended to lower spatial frequency achromatic stimuli (0.25 cpd). When the stimulus was red–green, performance on *saccade* and *no saccade* trials did not differ significantly for either monkey at any spatial frequency ($p > 0.05$; Figures 4A and 4B, red bars). Microsaccades are thus associated with poor task performance when the stimulus is achromatic but not when it is red–green.

Detection thresholds, unlike percent correct, are unbounded and provide an intuitive measure of the magnitude of the change in visual sensitivity. Threshold changes, shown in Figures 4C and 4D, were significant in all achromatic conditions ($p < 0.05$) except for the 0.25-cpd condition for Monkey S ($p = 0.34$). The magnitudes of these effects were modest: across spatial frequencies, the median change in threshold was 0.17 log unit for Monkey K and 0.13 log unit for Monkey S. When the stimulus was red–green, threshold changes were not significant at any spatial frequency. Together, these analyses support the idea that microsaccades impair the detection of achromatic stimuli more than red–green stimuli across a range of spatial frequencies.

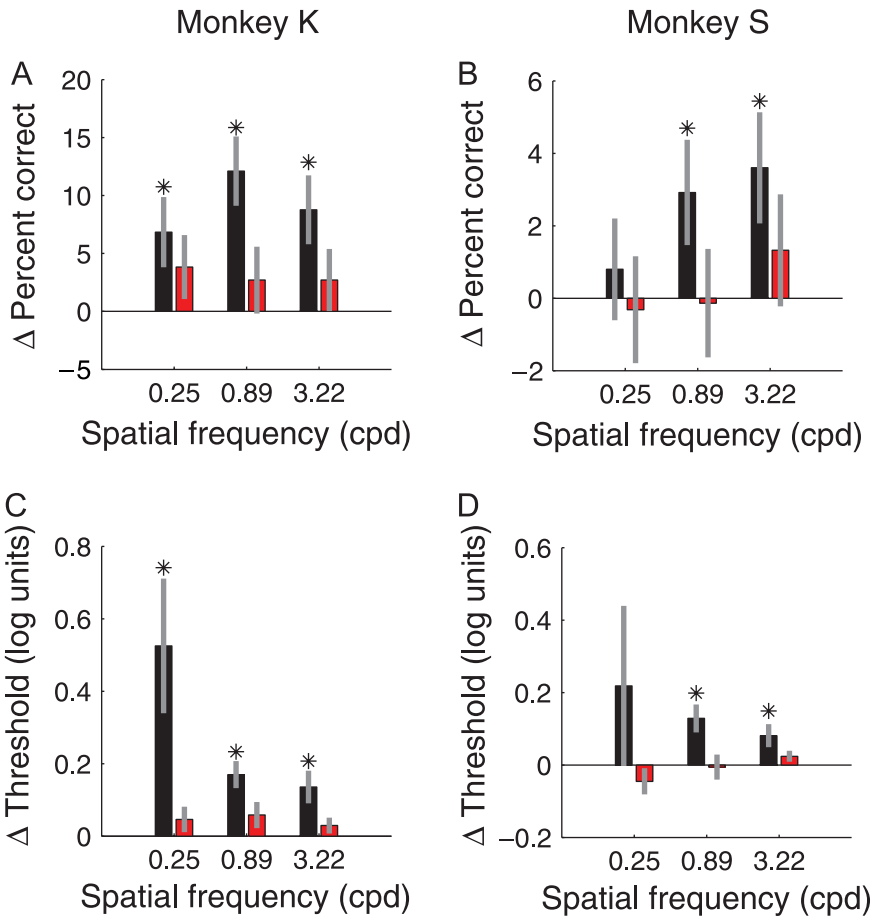


Figure 4. Summary of microsaccadic suppression. (A, B) Effects of microsaccades during the stimulus plateau on percent correct. ΔPercent correct is defined as the percentage of *no saccade* trials ending in a correct choice minus the percentage of *saccade* trials ending in a correct choice. Asterisks indicate significant differences in percentage of correct trials (z-tests for binomial proportions, $p < 0.05$). Error bars are ±1 standard error of the difference in binomial proportion. (C, D) Effects of microsaccades during the stimulus plateau on detection thresholds assessed from psychometric function fits. Asterisks indicate significant differences in thresholds between *saccade* and *no saccade* trials (analysis of deviance, $p < 0.05$; McCullagh & Nelder, 1989). Error bars are ±1 standard error obtained by bootstrap resampling. Black bars show data from achromatic trials, and red bars show data from red–green trials.

Relationship between microsaccade metrics and choices

Microsaccades vary in direction and amplitude, and the relationship between detection performance and microsaccade occurrence might depend on these metrics. For example, downward microsaccades increase the speed of the (upward moving) stimulus on the retina, which in turn could make the stimulus easier or more difficult to detect. In this case, the vertical component of microsaccades would predict whether the monkey will make the correct choice at the end of the trial. Alternatively, microsaccades might invoke suppressive mechanisms when their amplitude exceeds a threshold, in which case microsaccade amplitude would be predictive of percent correct.

We addressed these possibilities with two analyses. In the first analysis, we binned microsaccades made during the stimulus plateau according to their amplitude and then computed percent correct across trials that contained a microsaccade in each bin. Bins containing fewer than 10 microsaccades were omitted from this analysis. As shown in Figures 5A and 5B, percent correct decreased with microsaccade amplitude for both monkeys (Monkey K: weighted least-squares regression slope = −0.24, $p < 0.01$; Monkey S: slope = −0.11, $p < 0.001$). On trials containing a large microsaccade (≥0.5°) during the stimulus plateau, percent correct was lower than on trials containing a microsaccade of lower amplitude (Monkey K: 65 vs. 71%, $p = 0.05$; Monkey S: 72 vs. 78%, $p < 0.05$, z-tests for equality of binomial proportions). Consistent with this relationship, microsaccade amplitudes during the stimulus presentation were slightly larger on error trials than on correct trials (Monkey K: 0.35 vs. 0.34°, $p = 0.08$, two-sample t-test; Monkey S: 0.27 vs. 0.26°, $p < 0.05$). Figures 5C and 5D show the time course of microsaccadic suppression separated by saccade amplitude.

To probe the relationship between percent correct and microsaccade vector (amplitude and direction considered jointly), we plotted distributions of microsaccade vectors at different times during the trial and asked whether

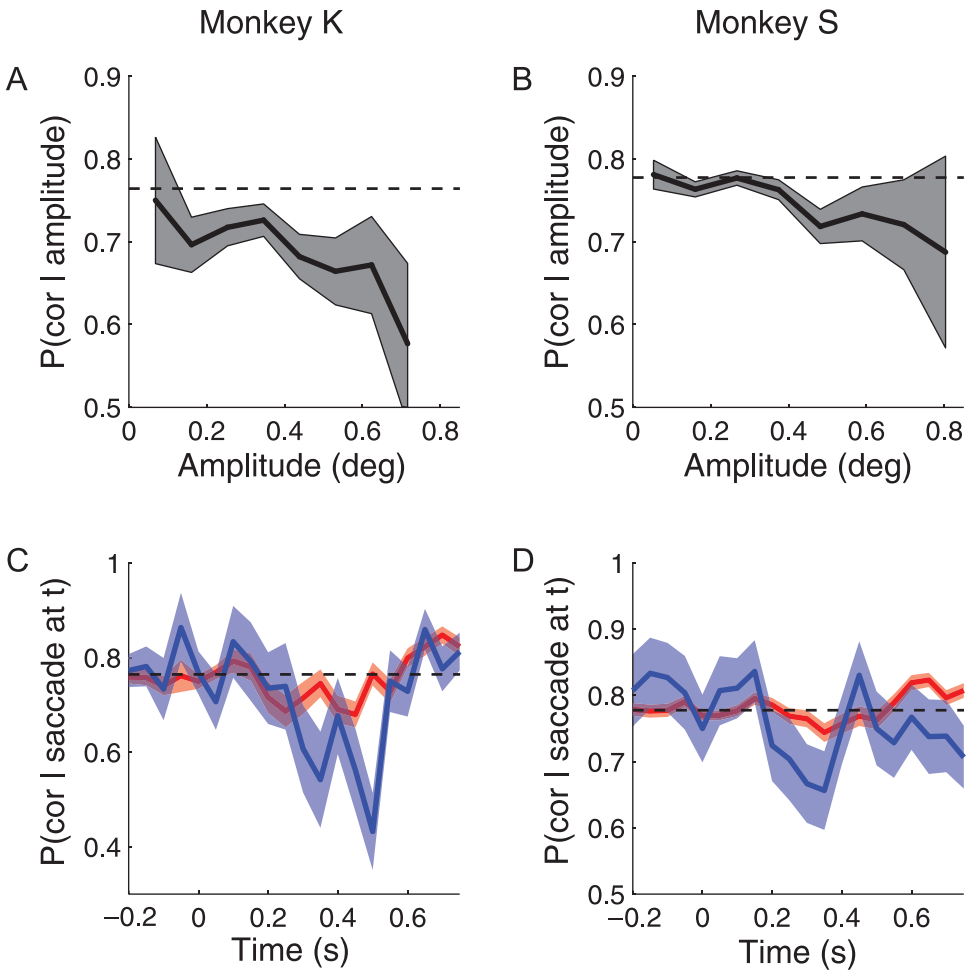


Figure 5. Effect of microsaccade amplitude on suppression. (A, B) Proportion of correct choices as a function of microsaccade amplitude. (C, D) Proportion of correct choices as a function of when a microsaccade occurred. Blue curve shows data from large (≥0.5°) amplitude microsaccades and red curve shows data from small (<0.5°) amplitude microsaccades. In all panels, dashed lines show the proportion of correct choices on *no saccade* trials, and shaded areas represent ±1 standard error of a binomial proportion.

probability correct depended on microsaccade vector. In the top rows of the panels in Figure 6 (labeled "*p* (correct)"), color represents the probability that the monkey makes a correct response at the end of the trial. Warm and cool colors represent high and low probabilities, respectively. Colors were assigned by binning microsaccade vectors, coding them according to trial outcome (correct or incorrect), and computing the proportion of correct trials in each bin. For visual clarity, bins with fewer than 10 vectors were omitted from the analysis.

These plots, which appear in the upper row of the panels of Figure 6 for each monkey, lack clear spatial structure suggesting that microsaccade vectors are not strongly related to percent correct. Nevertheless, a bootstrap test on the centroids of microsaccade vectors preceding correct and incorrect choices uncovered a weak relationship that evolves late in the trial. Microsaccades with a large downward component tend to precede incorrect choices. In Figure 6, centroids of microsaccade vectors preceding correct and incorrect choices are represented by white and black dots, respectively, in panels for which the difference between them was significant. The distance between the white and black dots was increased by a factor of 10 to better illustrate their relative positions. The intercentroid distance was significant for Monkey K from $t \geq 0.55$ and for Monkey S from $t \geq 0.4$ (asterisks in Figure 6, $p < 0.05$) and the white

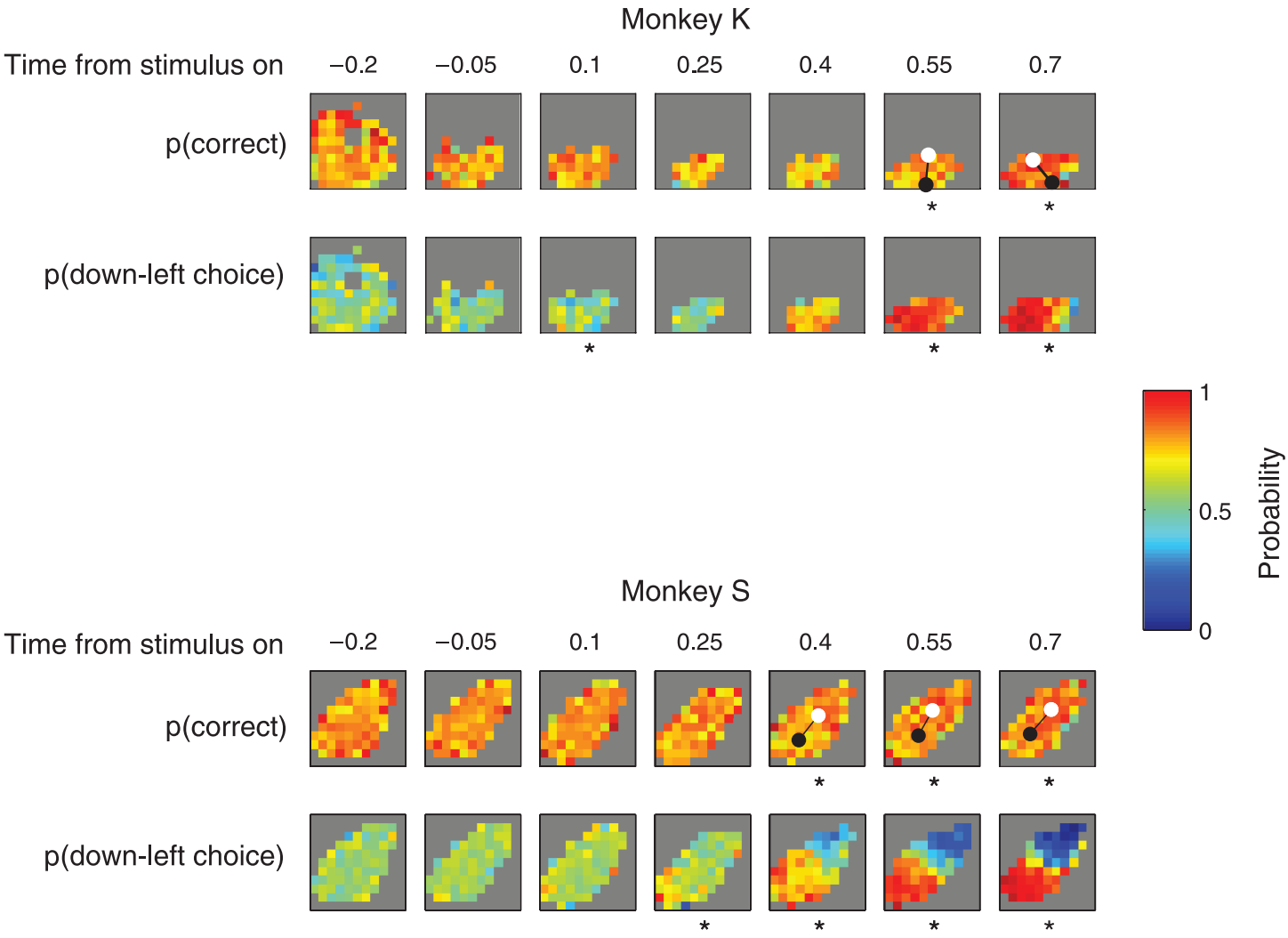


Figure 6. Probability of correct choices and down-leftward choices as a function of microsaccade vector and time. The center of each time bin (in seconds) appears above the upper panels (stimulus onset at time 0). Upper panels for each monkey: Color represents probability correct. Lower panels for each monkey: Color represents the probability of a choice to the lower left-hand target. Bins containing fewer than 10 trials were excluded from the analysis. Asterisks indicate epochs during which the mean microsaccade vector differed significantly ($p < 0.05$) between conditions (correct vs. incorrect in the upper panels or target 1 vs. target 2 choices in the lower panels) by a nonparametric bootstrap test. White and black dots in the upper panels represent the mean vector of microsaccades preceding correct and incorrect choices, respectively. The distance between the white and black dots has been increased 10-fold to prevent overlap between the symbols.

dot lies consistently above the black one. At first glance, this result is consistent with the idea that downward microsaccades induce stronger suppression than upward microsaccades, but the emergence of this effect late in the trial suggests a different interpretation.

The relationship between microsaccade direction and percent correct can be explained by a choice bias. Both monkeys made more choices (and more errors) to the lower left target than the upper right target (data not shown). Moreover, before making a choice to the lower left target, the monkeys tended to make more downward microsaccades than upward microsaccades (shown below). Together, these two effects account for the fact that downward microsaccades tended to precede incorrect choices (to the lower left target).

An analysis of microsaccade vectors preceding upward and downward choices supports this interpretation. This analysis was identical to that shown in the upper panels of Figure 6 except that vectors were colored to reflect the probability that the monkey chose the lower left target. Results from this analysis appear in the lower row of the panels in Figure 6 (labeled "*p*(down-left choice)"). Using a bootstrap test analogous to the one described above, we found that microsaccade vectors predicted target choices ~550 ms after the onset of the visual stimulus for Monkey K and ~250 ms after the onset of the stimulus for Monkey S. Monkey K tended to make downward microsaccades preceding downward choices but rarely made upward microsaccades preceding upward choices. Monkey S tended to make microsaccades in the direction of the chosen target. These effects became stronger as the trial progressed; for Monkey S, an upward microsaccade following stimulus disappearance preceded an upward choice ~10 times more often than a downward choice.

The relationship between microsaccade vectors and percent correct can thus be explained as a consequence of the relationship between microsaccade vectors and target choice. Can we account for the relationship between microsaccade *occurrence* and percent correct (Figures 3–5) this way also? We cannot; performance on achromatic *saccade* trials was worse than on achromatic *no saccade* trials even when the analysis was restricted to choices made to single targets (Monkey K: downward target, 66 vs. 74% correct, $p < 0.01$; upward target, 70 vs. 80% correct, $p < 0.01$; Monkey S: downward target, 74 vs. 77% correct, $p < 0.01$; upward target, 82 vs. 82% correct, $p = 0.81$). We conclude that microsaccades during the presentation of an achromatic stimulus are associated with elevated detection thresholds and that this effect depends little on the target the monkey chooses or microsaccade vector.

Influence of microsaccades on V1 neuron responses

Microsaccades affect the firing rates of neurons at many stages of the visual system (Bair & O'Keefe, 1998; Hafed & Krauzlis, 2010; Kagan, Gur, & Snodderly, 2008; Leopold & Logothetis, 1998; Martinez-Conde, Macknik, & Hubel, 2000; Snodderly, Kagan, & Gur, 2001). At least some of these modulations likely contribute to saccadic suppression. The fact that microsaccades affect chromatic and achromatic detection thresholds differently implies a differential modulation of achromatic and chromatic signals somewhere in the brain. Whether magnocellular neurons in the LGN are suppressed preferentially during saccades is controversial (Ramcharan et al., 2001; Reppas et al., 2002; Royal et al., 2006). We tested the idea that microsaccades preferentially suppress firing in cone-nonopponent neurons in area V1.

We recorded from 52 L – M opponent and 61 L + M nonopponent V1 neurons from Monkey K during random visual stimulation. Cone inputs were estimated and cells classified on the basis of an analysis of spike-triggered stimulus averages (STAs; Horwitz et al., 2007). Neurons were stimulated with a 10 × 10 checkerboard stimulus in which the color of each 0.1 × 0.1° element changed independently at 75 Hz (details of the stimulus and analysis can be found in Horwitz et al., 2007). The mean receptive field location of the recorded neurons was at (−3.1, −4.8°), which is closely matched to the location of the lower left Gabor stimulus in the contrast detection task. The sample of neurons was thus representative of those available for mediating performance on the detection task.

STAs are shown for 16 nonopponent neurons in Figure 7A and for 16 cone-opponent neurons in Figure 7C. For each cell, we aligned spike rasters to microsaccade initiations and averaged across them to derive a microsaccade-triggered spike-density function (SDF). SDFs are displayed in Figures 7B and 7D at the corresponding position of each STA. Some neurons were suppressed after microsaccades, others were excited, and others were suppressed and then rebounded. We found no clear relationship between SDFs and the spatial structure of STAs.

Nor did we find a clear relationship between SDFs and cone opponency. Figure 8A shows SDFs for each of the 113 neurons represented as a stack of vertically concatenated grayscale raster lines. To compensate for differences in firing rate across cells, each SDF was normalized by division by its maximum. On average, microsaccades evoked a brief suppression and then a rebound in spiking activity in both cone-opponent and nonopponent neurons (Figure 8B). Comparison of normalized SDFs from the dip (50–100 ms post-saccade) or rebound (120–300 ms post-saccade) in activity did not differ significantly between opponent and nonopponent neurons (dip: $p = 0.17$, rebound: $p = 0.32$, unpaired *t*-tests).

To express suppression in percent change in firing rate, we divided the firing rate during the dip by the maintained firing rate over the 250 ms preceding microsaccade initiation. Figure 8C shows how suppression quantified this way varies with maintained firing rate. The geometric mean suppression across all neurons (dashed line) was

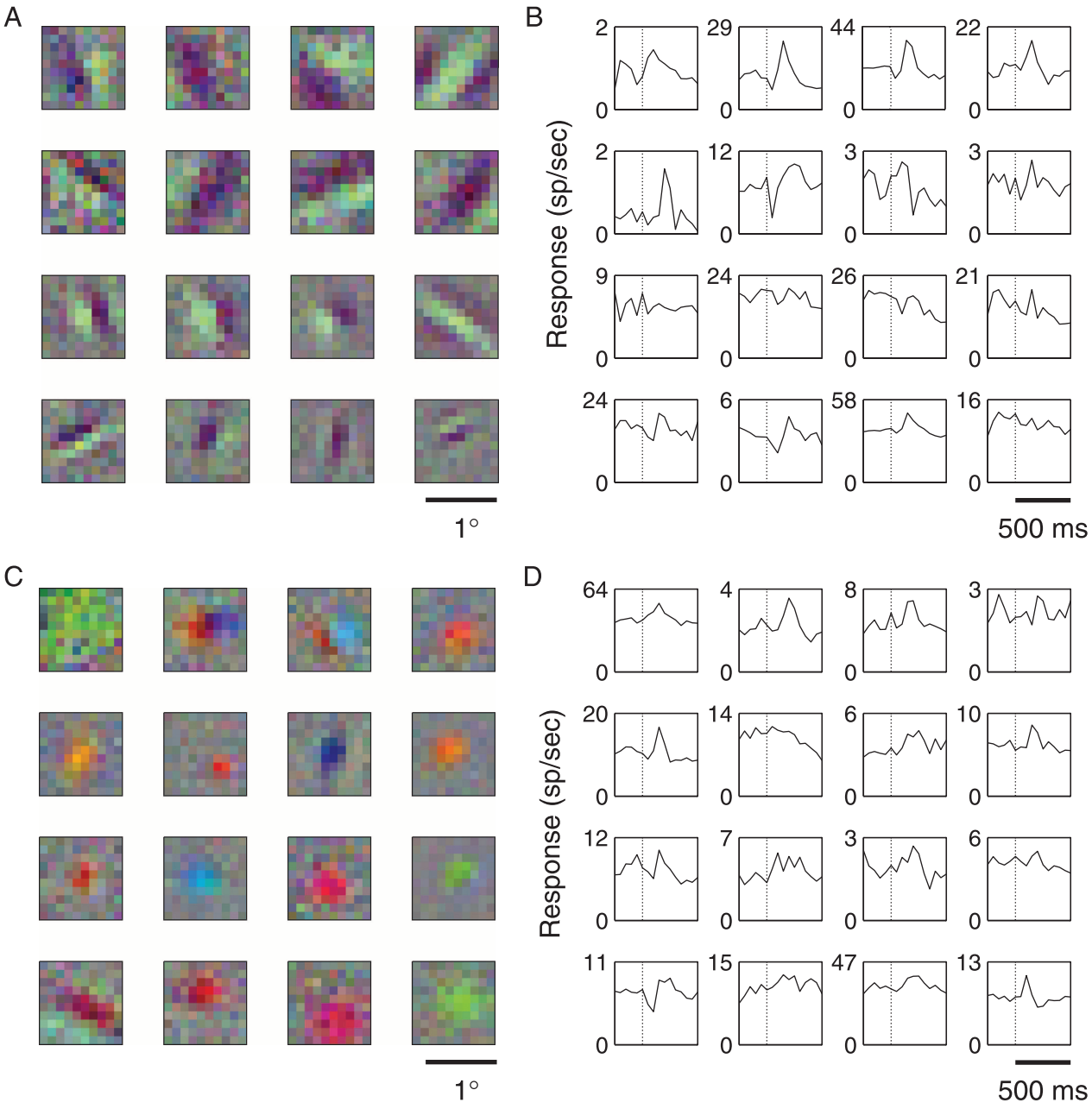
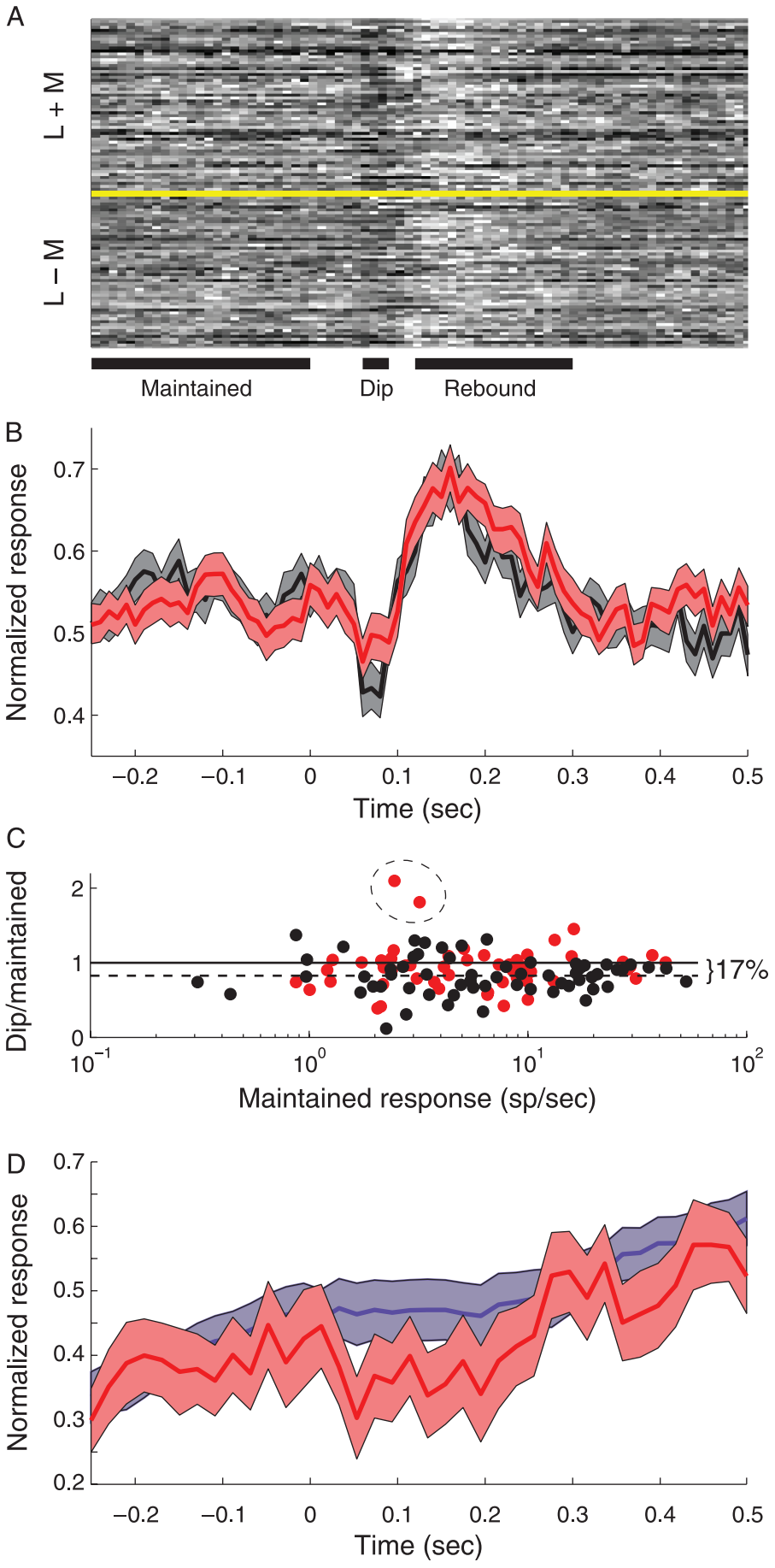


Figure 7. Spike-triggered averages (STAs) for (A) 16 neurons with nonopponent L- and M-cone weights and for (C) 16 neurons with opponent L- and M-cone weights. (B, D) Microsaccade-triggered spike-density functions. Dotted lines indicate the time of microsaccade initiation. Data from single neurons appear at corresponding locations in (A) and (B) (and similarly in (C) and (D)).

0.83, implying a 17% reduction in firing rate ($p < 0.0001$, one-sample t-test). Suppression was not significantly related to the maintained firing rate (Pearson's $r = 0.028$, $p = 0.77$) but was significantly greater for nonopponent neurons (22%) than cone-opponent neurons (11%; two-sample t-test, $p < 0.05$). This difference between cone-opponent and nonopponent neurons is consistent with the greater effect of microsaccades on the detection of achromatic relative to red–green stimuli but was contingent on the inclusion of two cone-opponent neurons (circled in Figure 8C) that responded above baseline during the 50–100 ms following a microsaccade. Omitting these two neurons from the analysis eliminated the difference between cone-opponent and nonopponent neurons (two-sample t-test: $p = 0.17$).

To determine whether the suppression of V1 responses following microsaccades depended on saccade amplitude, as perceptual suppression does, we computed for each neuron the correlation between microsaccade amplitude and the firing rate during the 50–100 ms following a microsaccade. The average correlation coefficient was 0.003, which was not significantly different from 0 ($p = 0.57$,


A
L + M
L – M
Maintained
Dip
Rebound
B
Normalized response
0.7
0.6
0.5
0.4
−0.2
−0.1
0
0.1
0.2
0.3
0.4
0.5
Time (sec)
C
Dip/maintained
2
1
0
}17%
10^{-1}
10^{0}
10^{1}
10^{2}
Maintained response (sp/sec)
D
Normalized response
0.7
0.6
0.5
0.4
0.3
0.2
−0.2
−0.1
0
0.1
0.2
0.3
0.4
0.5
Time (sec)

one-sample *t*-test). Our data thus provide no evidence that microsaccadic suppression of V1 responses depends on microsaccade amplitude.

The white noise stimulus had the same average luminance and chromaticity as the Gabor stimulus, but it differed in spatiotemporal contrast. The possibility therefore remains that microsaccades modulate the activity of V1 neurons when they are stimulated with white noise stimuli but not low contrast, red–green Gabor stimuli. To address this possibility, we recorded V1 responses during performance of the 2AFC detection task. In these experiments, the square frames were not shown, and the stimulus contrasts, which spanned psychophysical threshold, were determined by the Method of Constant Stimuli. Many of the neurons we screened responded weakly or not at all to these near-threshold red–green stimuli, but 21 neurons were sufficiently responsive to be included in this analysis (14 from Monkey K and 7 from Monkey S). As shown in Figure 8D, microsaccades suppressed the responses of these neurons as well. The time course of the suppression was protracted relative to the time course observed with white noise stimulation (Figure 8B), and a clear rebound was not observed.

The normalized firing rate 500 ms after a microsaccade exceeded the normalized firing rate 250 ms before a microsaccade. This change in firing rate was not due to microsaccades but rather the fact that most microsaccades preceded visual responses to the Gabor stimulus. To isolate the effects of microsaccades from other influences on firing rate, we shuffled microsaccade times and spike times across trials. The purple curve in Figure 8D shows the average SDF calculated from randomly shuffled data across the 21 neurons. A gradual increase in firing rate is common to both the shuffled (purple) and unshuffled (red) SDFs and therefore is not time-locked to the occurrence of microsaccades. In contrast, an ~200-ms-long post-saccadic dip in activity is visible in the unshuffled (red) SDF only, indicating that this suppression is time-locked to microsaccades. We conclude that the differential effect of microsaccades on the detection of achromatic and red–green stimuli is not due to a privileged immunity of red–green responsive V1 neurons from microsaccadic suppression.

Figure 8. (A) Normalized microsaccade-triggered spike-density functions displayed as grayscale raster lines. Normalization was achieved by dividing each cell's spike-density function by the largest value of that function over the time domain shown. Data from 61 nonopponent L + M cells and 52 opponent L – M cells appear above and below the yellow line, respectively. Solid lines at the base of (A) delimit epochs over which spikes were counted for quantitative analyses. (B) Normalized microsaccade-triggered spike-density functions averaged across nonopponent neurons (black) and opponent neurons (red). (C) Scatter plot of suppression (firing rate during dip divided by maintained firing rate) as a function of the maintained firing rate for nonopponent neurons (black) and opponent neurons (red). The dashed line shows the geometric mean of this ratio across neurons (0.83 = 17% suppression). The dashed ellipse encloses two cone-opponent neurons whose firing rate was above baseline during the dip interval. (D) Normalized microsaccade-triggered spike-density function (red) averaged across 21 V1 neurons that were responsive to near-threshold red–green modulations during the 2AFC detection task. The spike-density function shown in purple was computed from data in which microsaccade and spike times were randomly permuted across trials. Width of band in (B) and (D) indicates ±1 *SEM* across neurons.

Discussion

We report that microsaccades increase luminance contrast detection thresholds, but not red–green contrast detection thresholds, in rhesus monkeys. We tested the hypothesis that this chromatic selectivity results from a suppression of L + M, but not L – M, V1 neurons. Contrary to this hypothesis, microsaccadic modulation of spiking activity was similar in the two groups of neurons.

Below, we consider and reject an alternative interpretation of the suppression effect: that detection of the stimulus inhibited microsaccade production. We then discuss the influence of color and spatial frequency and discuss clues that these effects provide regarding the mechanisms of microsaccadic suppression. We describe the microsaccadic behavior of our monkeys during task performance and compare our results to those obtained with human subjects. We conclude with the implications of our results for the neurophysiological investigation of contrast detection in monkeys.

Direction of causality

We found that microsaccades were correlated with increased detection thresholds. Logically, this correlation could be due to an elevation of detection thresholds by microsaccades or to an inhibition of microsaccades by the appearance of the stimulus (Cui, Wilke, Logothetis, Leopold, & Liang, 2009; Rolfs, 2009). We favor the former explanation for three reasons. First, the color specificity of the microsaccadic suppression we observed matched the color specificity of suppression observed in human subjects making large amplitude saccades (Burr & Morrone, 1996; Burr et al., 1994; Diamond et al., 2000), whereas the inhibition of microsaccades by visual transients in humans is not color-specific (Rolfs, 2009). Second, large amplitude saccades produce suppression in monkeys (Mohler & Cechner, 1975) and it is likely that microsaccades do too, as they do in humans. Third, if stimulus visibility inhibited microsaccades, we would expect microsaccades to be suppressed strongly on high

contrast trials and weakly on low contrast trials. This was not the case; stimulus contrast was higher on *saccade* trials than on *no saccade* trials on average.

Effects of color

Large amplitude saccades exert greater effects on the perception of achromatic than chromatic stimuli (Bridgeman & Macknik, 1995; Burr et al., 1994; Diamond et al., 2000; Uchikawa & Sato, 1995). Similarly, we found greater microsaccadic suppression for achromatic stimuli than for red–green chromatic stimuli. These observations suggest that saccadic and microsaccadic suppression invoke common mechanisms and may be related to the recent finding that large amplitude saccades and microsaccades are produced by largely overlapping neural circuits (Hafed, Goffart, & Krauzlis, 2009).

One explanation for the chromatic specificity of saccadic suppression is that nonopponent neurons somewhere in the visual system are suppressed preferentially around the time of a saccade. We looked for, and failed to find, differences in peri-microsaccadic modulation of cone-opponent and nonopponent neurons in V1. Our results are consistent with those of Kleiser, Seitz, and Krekelberg (2004) who found comparable suppression in V1 BOLD response when subjects made saccades across luminance or isoluminant red–green gratings. We propose that the chromatic specificity of saccadic suppression occurs largely downstream of V1. The fact that V4 neurons are more strongly suppressed by saccades when stimulated with achromatic than chromatic stimuli suggests that a critical locus for saccadic suppression may lie between V1 and V4 (Han, Xian, & Moore, 2009). Transient visual neurons in the superficial superior colliculus, which are activated weakly by color (White, Boehnke, Marino, Itti, & Munoz, 2009) and suppressed by microsaccades (Hafed & Krauzlis, 2010), may play a particularly important role.

Effects of spatial frequency

Microsaccades elevated detection thresholds for low spatial frequency stimuli more than for high spatial frequency stimuli, consistent with results from Burr et al. (1994). Although this relationship has been reported before, seeing this trend in our data was surprising for two reasons. First, the range of spatial frequencies we tested was not optimal for observing this effect. Burr et al. found that saccadic suppression was maximal at spatial frequencies below ~0.12 cpd. The lower end of our spatial frequency range, 0.25 cpd, was above this value. Second, retinal image blur, which affects high spatial frequencies more than low spatial frequencies, played a greater role in our study than in the study of Burr et al. Burr et al. instructed subjects to make saccades parallel to the bars of an extended grating, a configuration that minimizes the contribution of retinal blur to saccadic suppression. The saccades we studied had a variety of directions and amplitudes, some of which smeared the stimulus on the retina. For example, one half-cycle of the 3.2-cpd stimulus is 0.16°, so a vertical microsaccade of typical amplitude could reverse the spatial phase of this stimulus in ~14 ms (the duration of a microsaccade of 0.16°). Such a shift would cause individual photoreceptors to encounter both a stimulus peak and trough within one integration period thereby reducing or nulling the response (Schneeweis & Schnapf, 1999). A microsaccade could not exert the same effect on the 0.25-cpd stimulus, one half-cycle of which is larger than the largest microsaccade in our study.

Microsaccades did not significantly impair the detection of 0.25 cpd, achromatic stimuli for Monkey S but did for Monkey K. We considered the possibility that Monkey S performed the task poorly in this condition as poor behavior could have caused subtle effects of microsaccades on stimulus detectability to go unnoticed. Instead, we found that Monkey S's performance in this condition was similar to that of Monkey K, and when we analyzed only those sessions from Monkey S in which detection thresholds were lowest, the suppression effect changed minimally and remained insignificant. The absence of a saccadic suppression effect in this condition is therefore unlikely a product of poor task performance.

Another possibility is that this difference between monkeys may result from differences in their microsaccadic behavior. Monkey K made more large amplitude microsaccades than Monkey S, and large amplitude microsaccades increased detection thresholds more than small amplitude microsaccades. These observations are consistent with the larger microsaccadic suppression in Monkey K than Monkey S. On the other hand, microsaccades exerted a greater effect on detection for Monkey K than for Monkey S even when matched for amplitude (Figure 5). We are thus unable to explain this difference between monkeys on the basis of microsaccade amplitudes. Data from additional animals could help resolve this issue.

Dynamics in microsaccade frequency

The variations in microsaccade frequency over the course of the trial (Figures 3A and 3B) can be understood in terms of the influences of visual stimulation and task demands on microsaccades. Our monkeys exhibited a biphasic modulation of microsaccade rate following the presentation of the stimulus frames. This modulation may be related to the transient suppression and rebound of microsaccades made by human observers following a flash in the retinal periphery (Engbert & Kliegl, 2003). The monkeys made relatively few microsaccades during the

Gabor stimulus presentation, which may be related to the temporary allocation of resources to a demanding visual task (Bridgeman & Palca, 1980; Winterson & Collewijn, 1976). The increase in saccade frequency at the end of the trial may be related to the allocation of covert spatial attention or the preparation of the saccade to a peripheral target (Engbert & Kliegl, 2003; Hafed & Clark, 2002; Horowitz, Fine, Fencsik, Yurgenson, & Wolfe, 2007). The effect was greater on correct trials, consistent with the idea that these movements represent a (barely) suppressed urge to make a saccade to the identified correct target.

The magnitude of microsaccadic suppression

Large amplitude saccades increase detection thresholds by 0.5 to 1 log unit (Burr et al., 1994; Diamond et al., 2000; Mohler & Cechner, 1975; Volkmann, Riggs, White, & Moore, 1978). Spontaneous microsaccades (of unreported amplitude) increase detection threshold by ~0.3 log unit (Beeler, 1967). The changes in threshold that we observed (0.1–0.5 log unit) span this benchmark.

Our stimulus was on the screen for 667 ms, which is much longer than the typical microsaccade duration (10–30 ms), the subsequent perceptual suppression (<100 ms; Beeler, 1967), or stimulus presentations used in previous studies (<1–32 ms; Diamond et al., 2000; Kleiser et al., 2004; Krauskopf et al., 1966; Schutz, Braun, & Gegenfurtner, 2007; Uchikawa & Sato, 1995; Volkmann et al., 1978; Zuber & Stark, 1966). Given this long stimulus duration, the fact that suppression was modest in some of the conditions we studied is perhaps less surprising than the fact that it was observable at all. For many purposes, microsaccadic suppression in monkeys may be sufficiently small as to be negligible. One context in which it might not be negligible is in the interpretation of neurophysiological experiments of contrast detection in monkeys, which is discussed below.

Microsaccades and choice probability

Choice probability is a metric that quantifies the covariation between neuronal activity and behavioral performance during psychophysical tasks (for a review, see Parker & Newsome, 1998). Significant choice probabilities have been taken as evidence that a sensory neuron's spiking activity contributes causally to the animal's perceptual judgment (Shadlen, Britten, Newsome, & Movshou, 1996), but microsaccades may contribute to choice probability in the absence of a causal relationship.

Herrington et al. (2009) found that microsaccades contribute to choice probability in some tasks. Monkeys more readily detect changes in visual motion when the change modulates the activity of motion-selective neurons in the visual cortex strongly. Part of this relationship can be attributed to the fact that microsaccades increase detection thresholds for changes in visual motion and suppress the responses of motion-sensitive visual neurons. A correlation between task performance and neuronal responses thus follows.

Our study extends the findings of Herrington et al. (2009) to luminance contrast detection. We found that microsaccades impair contrast detection and modulate the spiking activity of V1 neurons (see also Kagan et al., 2008; Leopold & Logothetis, 1998; Martinez-Conde et al., 2000; Snodderly et al., 2001). A monkey's ability to detect a near-threshold, achromatic stimulus is therefore expected to be correlated with V1 response to that stimulus. On trials without a microsaccade during the stimulus presentation, we would expect low behavioral thresholds and vigorous stimulus-locked neural responses. On trials including a microsaccade during the stimulus presentation, we would expect elevated behavioral thresholds and diminished neural responses. In the absence of a visual stimulus, we would expect no relationship between perceptual reports and neural responses. These effects have been reported in monkeys performing a seen/not seen contrast detection task (Palmer et al., 2007).

Our study shows that microsaccades play a small but potentially important role in determining contrast detection thresholds of rhesus monkeys. We found clear evidence for microsaccadic suppression when the animal was detecting achromatic but not red–green stimuli. We found that microsaccadic modulation of L – M and L + M V1 neurons was similar in amplitude and time course, suggesting that the locus of chromatic specificity is downstream of V1. Our study does not address whether microsaccade-induced modulation of V1 activity is causally related to detection performance. It remains possible that activity modulations in V1 are unrelated to behavioral microsaccadic suppression. An important future direction is to determine at what stage of the visual system microsaccade-related changes in activity can account quantitatively for changes in visual performance.

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