Stronger premicrosaccadic sensitivity enhancement for dark contrasts in the primate superior colliculus

Wenbin Wu[1,2] and Ziad M. Hafed[1,2]

1. Werner Reichardt Centre for Integrative Neuroscience, University of Tübingen, Tübingen, Germany

2. Hertie Institute for Clinical Brain Research, University of Tübingen, Tübingen, Germany

Correspondence: ziad.m.hafed@cin.uni-tuebingen.de

Abstract

Microsaccades are associated with enhanced visual perception and neural sensitivity right before their onset, and this has implications for interpreting experiments involving the covert allocation of peripheral spatial attention. However, the detailed properties of premicrosaccadic enhancement are not fully known. Here we investigated how such enhancement in the superior colliculus depends on luminance polarity. Rhesus macaque monkeys fixated a small spot while we presented either dark or bright image patches of different contrasts within the recorded neurons' response fields. Besides replicating premicrosaccadic enhancement of visual sensitivity, we observed stronger enhancement for dark contrasts. This was especially true at moderate contrast levels (such as 10-20%), and it occurred independent of an individual neuron's preference for either darks or brights. On the other hand, postmicrosaccadic visual sensitivity suppression was similar for either luminance polarity. Our results reveal an intriguing asymmetry in the properties of perimicrosaccadic modulations of superior colliculus visual neural sensitivity.

Introduction

Microsaccades are small saccades that occur during periods of maintained gaze fixation [1-4]. The generation of these rapid eye movements is associated with strong motor bursts in the superior colliculus (SC) [2,5-7], and these motor bursts are similar in character to those associated with larger saccades [8]. As a result, microsaccades are additionally known to be accompanied by robust perimovement alterations in visual neural sensitivity and perception, just like larger saccades are [2,7,9].

Perimicrosaccadic alterations in SC visual responses include premicrosaccadic enhancement of neural sensitivity [10], postmicrosaccadic suppression of neural sensitivity [11-13], as well as perimovement shifts in foveal visual response field (RF) locations [7,14]. These alterations are consistent with known perimicrosaccadic changes in perception [9,15,16]. For example,

premicrosaccadic enhancement in perception occurs for peripheral stimuli, and this also happens in covert attentional cueing tasks [15]. This suggests that perimicrosaccadic alterations in visual processing (whether in the SC, other brain areas, or perceptually) can have a substantial impact [9] on the interpretation of experiments containing sensory transients, such as in the case of cueing paradigms for the study of covert attention [9,15-17]. In fact, coupled with the intrinsic rhythmicity of microsaccades and the resetting of such rhythmicity by exogenous peripheral or foveal visual stimuli [9,18,19], it is possible [9,15,16,20] (at least theoretically) to fully account for classic Posner-style [21-23] cueing effects solely by the known perimicrosaccadic alterations in vision. This makes it necessary to keep studying additional properties of perimicrosaccadic effects.

Intriguingly, perisaccadic changes in visual sensitivity can exhibit different asymmetries as a function of different visual scene parameters. For example, even though visual perception can be worse in the upper rather than lower retinotopic visual field in the absence of saccades [24-31], perisaccadic perceptual performance is actually better in the upper visual field (during the strong perisaccadic suppression epoch) [13]; this is an opposite asymmetry from the no-saccade case, but it is one that nonetheless matches the one present in SC visual neural sensitivity [32]. Moreover, the strength of perceptual suppression associated with microsaccades can vary as a function of the visual appearance of foveated images [33,34], and different image properties can modulate the strength of perisaccadic suppression both perceptually and neurally [12,34-37].

Here we uncovered an additional asymmetry in perimicrosaccadic visual sensitivity, this time with a focus on SC visual responses to different luminance polarities, and specifically for premicrosaccadic stimulus onsets. We previously found that SC visual responses occur slightly earlier for dark than bright luminance contrasts, regardless of whether individual neurons prefer (in terms of their visual response strengths) darks or brights [38]. Importantly, there was no clear bias in the SC population to prefer either dark or bright luminance polarities. In addition, it was not studied how visual response strength for dark and bright stimuli was altered by microsaccades. This is what we did here. We found a specific asymmetry in the premicrosaccadic interval: premicrosaccadic enhancement of peripheral SC visual neural sensitivity was stronger for dark than bright stimuli, particularly at 10%-20% contrast levels, and this was independent of whether individual neurons intrinsically preferred one or the other luminance polarity. Postmicrosaccadic suppression of visual neural sensitivity was generally similar for either luminance polarity. These results add to the growing evidence of asymmetries in visual processing, and they extend them to the case involving concomitant eye movement generation.

Results

Our overall goal in this study was to look at how the visual response strength of extrafoveal SC neurons is altered if the visual stimulus appearing within the neurons' RF's occurs before or after tiny microsaccades. Thus, our monkeys fixated a small fixation spot, and we presented a disc of 0.51 deg radius within extrafoveal SC RF's (Methods). Across trials, the disc could be brighter than the gray display background (positive luminance polarity) or it

could be darker (negative luminance polarity). We varied the Weber contrast of the disc (relative to the background luminance) from trial to trial (Methods). In post-hoc analyses, we separated trials according to when the stimulus onset occurred relative to a given microsaccade (Methods). Baseline trials were those in which there were no microsaccades occurring within less than +/- 150 ms from stimulus onset. This was a sufficiently long interval to avoid at least the largest modulatory effects of microsaccades on vision [39]. For a given stimulus luminance polarity and contrast, we then measured the peak visual response in baseline trials, and we related it to trials in which the stimulus onset occurred within different time epochs around microsaccade onset.

Stronger premicrosaccadic enhancement for dark contrasts

Figure 1a-d shows the visual responses of an example SC neuron to two different contrasts (Fig. 1a, b versus Fig. 1c, d) and two different luminance polarities (Fig. 1a, c versus Fig. 1b, d). The dark and light gray curves show the baseline firing rates of the neuron within each luminance polarity condition in the complete absence of microsaccades near stimulus onset (Methods). As can be seen, this example neuron preferred the dark luminance polarity, as evidenced by the stronger visual responses for the negative luminance polarity at both shown contrast levels (compare the dark and light gray curves). The curves with the different shades of blue in the figure show the same neuron's visual response strengths, but now when the stimulus onsets occurred within <100 ms before microsaccade onset. The microsaccades had an average amplitude of only approximately 0.45 deg, whereas the neuron preferred an extrafoveal eccentricity of approximately 6.3 deg. For the dark stimuli (negative luminance polarity), premicrosaccadic enhancement in visual sensitivity of the neuron was evident (Fig. 1b, d). On the other hand, for the bright stimuli (positive luminance polarity), the enhancement was weak or absent (Fig. 1a, c). Similar observations were also made for a second example neuron from the same monkey; this time, the neuron (which was recorded simultaneously with the other neuron) preferred bright rather than dark stimuli in the absence of microsaccades (Fig. 1e-h, gray baseline curves). Once again, the neuron exhibited robust premicrosaccadic enhancement of visual sensitivity, and particularly for the negative luminance polarity (see, for example, the 100% contrast condition for this neuron; we later elaborate on the quantitative enhancement strengths as a function of different contrasts, luminance polarities, and neuron preferences for darks and brights). Therefore, regardless of neural preference for either darks or brights, premicrosaccadic enhancement was present in both of these example SC neurons, consistent with our earlier findings [10]. More importantly, premicrosaccadic enhancement seemed to be more robust for the dark stimuli in these two neurons. We next inspected these observations in more detail across the entire population.

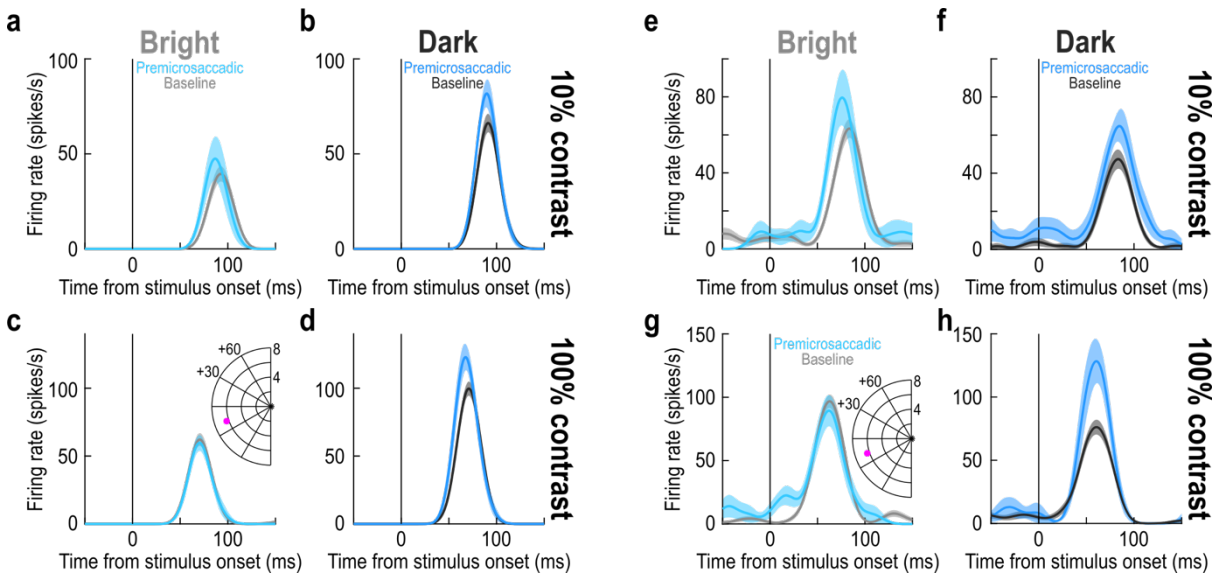

Figure 1 Stronger premicrosaccadic enhancement of superior colliculus (SC) visual sensitivity for dark contrasts. (a, b) Visual responses of an example SC neuron to 10% contrast stimuli of either positive (**a**) or negative (**b**) luminance polarity. The gray curves (dark in **b** and lighter shade in **a**) show the stimulus-evoked visual responses of the neuron in the absence of any microsaccades within +/- 150 ms from stimulus onset. The neuron preferred the dark stimulus. The colored curves show the same neuron's visual responses when stimulus onset occurred within <100 ms before microsaccade onset. Premicrosaccadic enhancement occurred, consistent with our earlier results [10], but it was stronger for the dark contrast. **(c, d)** The same neuron's responses for 100% contrast stimuli. This time, there was no premicrosaccadic enhancement for bright stimuli (**c**), but it was clearly present for dark stimuli (**d**). The inset shows the retinotopic preferred RF location of the neuron (approximately 6 deg eccentricity in the lower left visual field). **(e-h)** Another example neuron from the same SC site. This neuron preferred bright stimuli in its baseline curves. Once again, it exhibited premicrosaccadic enhancement. However, the enhancement was weak or absent for bright stimuli, and more robust for dark stimuli. Thus, there was stronger premicrosaccadic enhancement of SC visual sensitivity for negative luminance polarity stimuli. Error bars denote SEM. n = 24-33 trials in the baseline curves across panels; n = 5-9 trials in the premicrosaccadic curves across panels (Methods).

Across the population, we first confirmed that we obtained consistent premicrosaccadic enhancement [10] (and postmicrosaccadic suppression [10-12]). To do so, we used an approach similar to the one we used previously [11] (particularly because of the limited numbers of trials containing microsaccades within a given time epoch, as we discuss in more detail below and in Methods). Specifically, we normalized each neuron's firing rate curves across all trials, before pooling across neurons. The normalization factor was the peak of the average firing rate curve of the condition evoking the highest response in the absence of peristimulus microsaccades (Methods). That is, we found the condition in which the baseline (no-microsaccade) response of a neuron was the highest (typically, the 100% contrast condition of either luminance polarity), and we then divided the firing rates of all trials by this peak visual response. Then, for each time interval in which a stimulus onset occurred relative to a given microsaccade onset, we plotted the average normalized peak response across all data (Methods). This resulted in time courses of perimicrosaccadic modulations of visual neural sensitivity for all stimulus conditions (Methods), which we summarize in Fig. 2. In this figure, each horizontal dashed line shows the average normalized response of the population in the

baseline situation of no microsaccades within +/- 150 ms from stimulus onset (Methods). The correspondingly-colored solid curve shows the same response as a function of when a stimulus appeared relative to a given microsaccade onset (each sample on the x-axis shows an averaging window of within +/- 25 ms from the sample center). As can be seen, we always observed premicrosaccadic enhancement and postmicrosaccadic suppression of SC visual sensitivity, consistent with our earlier observations [10-12], and this was additionally the case for different contrasts and different luminance polarities. Specifically, the dashed horizontal lines were rank-ordered with increasing contrast, consistent with the contrast sensitivity curves of the neurons [38]. And, for each contrast, the perimicrosaccadic modulation involved a characteristic pattern of premovement enhancement relative to the corresponding baseline dashed line, followed by a subsequent postmovement suppression.

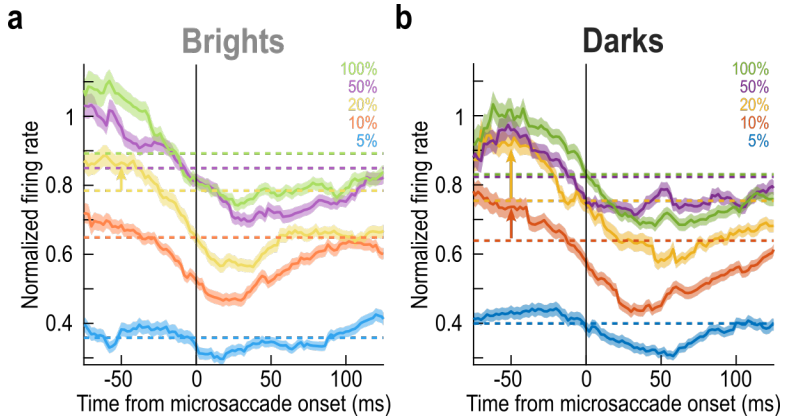

Figure 2 Stronger premicrosaccadic response gain enhancement for moderate dark contrasts in the primate SC. (a) For positive luminance polarity stimuli (brights), each dashed horizontal line shows the average normalized population firing rate (visual response strength) for trials in which stimuli of a given contrast appeared in the absence of any microsaccades within +/- 150 ms from stimulus onset (baseline trials). For each correspondingly-colored solid curve, the x-axis value indicates the center of a measurement window of +/- 25 ms from the given value, and the y-axis value indicates the normalized population visual response if stimulus onset occurred within the specific time window relative to microsaccade onset indicated by the x-axis value. For example, -50 ms on the x-axis indicates that we were measuring visual response strengths (y-axis value) for all trials in which stimulus onsets occurred within the interval from -75 ms to -25 ms from microsaccade onset. As can be seen, there was clear premicrosaccadic enhancement and postmicrosaccadic suppression, consistent with our earlier results [10-12]. **(b)** Same as **a** but for negative luminance polarity stimuli. Note how there was stronger premicrosaccadic enhancement than in **a** for the 10% and 20% contrast levels (upward pointing arrows at the -50 ms time point; see the corresponding comparison in **a**); also see Figs. 3-5. Error bars denote SEM.

Notably, relative to the no-microsaccade baseline sensitivity, premicrosaccadic enhancement was systematically stronger for the dark contrasts, especially at the contrast levels of 10%-20%. Consider, for example, the 20% contrast conditions in Fig. 2. For positive luminance polarity stimuli (brights), the no-microsaccade baseline sensitivity of our population was at a normalized firing rate of 0.784 (Fig. 2a). At the center of the -50 ms time bin (that is, for

stimuli occurring within the interval from -75 ms to -25 ms relative to microsaccade onset), this population firing rate was 0.866; thus, the SC population response was enhanced by 10.5% in the case of premicrosaccadic extrafoveal stimulus onsets (Fig. 2a). On the other hand, for dark stimuli of the same Weber contrast level, the no-microsaccade sensitivity was 0.754, and the premicrosaccadic sensitivity was 0.916, a 21.5% increase (Fig. 2b). Therefore, there was stronger premicrosaccadic enhancement of visual sensitivity for the negative luminance polarity condition across our population. As a second example, for 10% contrasts, the premicrosaccadic enhancement for bright stimuli was 5.4% at -50 ms (Fig. 2a), whereas it was 15.2% for dark ones (Fig. 2b). Importantly, these measures were made from the very same neurons under different luminance polarity conditions. Therefore, the observations cannot be explained by arguing that different SC neurons were differentially recruited by either the positive or negative luminance polarity stimuli; rather, within the very same neurons, premicrosaccadic enhancement was stronger if the appearing stimuli were dark. In other analyses that we will document shortly below, we also normalized the no-microsaccade reference lines for each condition individually, to achieve an even easier visualization of the stronger premicrosaccadic enhancement for dark stimuli than in Fig. 2, and we reached the same conclusions.

We assessed the observations of Fig. 2 further by plotting the data at the time bin -50 ms (+/- 25 ms) relative to microsaccade onset as full contrast sensitivity curves (Fig. 3a, b). We chose this time bin because it ensures stimulus onsets before microsaccade onset, while still having close temporal proximity to the movement onset event [10,15] (naturally, Fig. 2 indicates that other nearby time points would have yielded similar conclusions). The black data points in Fig. 3a, b show the baseline, no-microsaccade population firing rates as a function of stimulus contrast (same values as the horizontal dashed lines in Fig. 2); the continuous curves show computational fits to a monotonically increasing mathematical function (Methods). The data and fits demonstrate expected contrast sensitivity functions of primate SC neurons [10,38,40]. In the same panels of Fig. 3a, b, the colored data points and fits are those for only the trials in which the stimulus onset occurred within the interval from -75 ms to -25 ms relative to microsaccade onset (that is, the -50 ms x-axis point of Fig. 2). For both luminance polarities, there was clear premicrosaccadic enhancement in the population response [10] (Fig. 3a, b). However, the enhancement was stronger for the negative luminance polarity stimuli (darks), as also shown in Fig. 3c, in which we plotted the difference between the premicrosaccadic and baseline contrast sensitivity curves for either dark or bright stimuli.

Specifically, at 10% stimulus contrast, there was no substantial enhancement for bright stimuli relative to the no-microsaccade baseline sensitivity. This is indicated by the leftmost oblique arrow in Fig. 3a, c, and by the fact that a t-test of baseline versus premicrosaccadic firing rates at 10% contrast (Fig. 3a) yielded a p-value of 0.1741 ($t(6263) = -1.3594$; 5557 baseline trials; 728 premicrosaccadic trials; also see below for further statistical tests at the level of individual-neuron measurements). On the other hand, for 10% contrast dark stimuli (leftmost oblique arrow in Fig. 3b), there was a statistically significant difference between baseline and premicrosaccadic firing rates ($p = 6.3651 \times 10^{-6}$; t-test; $t(6310) = -4.5177$; 5692 baseline trials; 639 premicrosaccadic trials). These statistical observations were also consistent with the fact that the 95% confidence intervals associated with the difference measures of Fig. 3c at 10% contrast (which could be inferred from the shown SEM ranges)

overlapped with the zero-difference line for the bright stimuli but not for the dark ones (Fig. 3c).

Similarly, at 20% contrast, the enhancement for dark stimuli was already at the plateau level of enhancement in the difference curve of Fig. 3c, whereas it was not at the plateau enhancement level for the bright stimuli. Indeed, in Fig. 3a, baseline and premicrosaccadic normalized firing rates at 20% were 0.7844 (+/- 0.0278 SEM) and 0.8573 (+/- 0.0068 SEM), an enhancement of 9.293 % ($p = 0.0013$; t-test; $t(6212) = -3.219$; 5615 baseline trials; 613 premicrosaccadic trials); on the other hand, the values in Fig. 3b were 0.7541 (+/- 0.0067 SEM) and 0.9175 (+/- 0.0298 SEM), respectively, corresponding to an enhancement of 21.668% ($p = 5.4314 \times 10^{-13}$; t-test; $t(6066) = -7.2298$; 5486 baseline trials; 590 premicrosaccadic trials). Thus, there was stronger premicrosaccadic enhancement of SC visual neural sensitivity for negative luminance polarity stimuli, especially at 10-20% contrast levels.

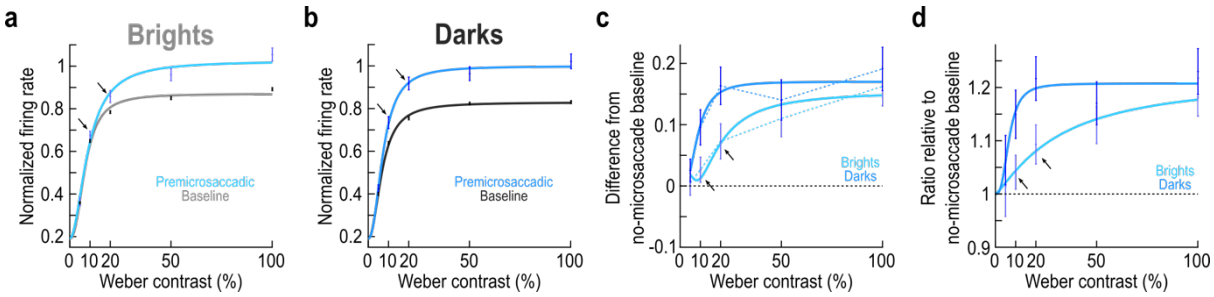

Figure 3 Stronger premicrosaccadic SC response gain enhancement for dark than bright stimuli at 10%-20% contrasts. (a) The black data points show the normalized population visual response firing rates as a function of Weber contrast for positive luminance polarity stimuli. We normalized all neurons' firing rates before averaging across them (Methods and ref. [11]). The gray continuous curve shows a curve fit of a monotonically increasing function (Methods). In color, we show the results and curve fit for stimulus onsets within the interval from -75 ms to -25 ms relative to microsaccade onset. There was clear premicrosaccadic enhancement [10]. However, at 10% and 20% contrast (black oblique arrows), the enhancement was small and not significant. **(b)** Same as **a** but for the negative luminance polarity stimuli being presented to the very same neurons. The enhancement at 10% and 20% contrast levels was now much more convincing (black oblique arrows). **(c)** For either brights or darks, we plotted the difference between premicrosaccadic and baseline data points and curves in **a**, **b**. The difference curve for darks was systematically higher than that for brights. The black oblique arrows show that for brights, the difference between premicrosaccadic and baseline curves was not significantly larger than zero at 10% contrast (error bars denote SEM); moreover, the difference was smaller in magnitude at 20% contrast when compared to the dark stimuli. **(d)** We also took the ratio of the premicrosaccadic to baseline data points in **a**, **b**. Here, we fitted the individual-point ratio measures to a mathematical function of the same form as that used in **a**, **b** for easier visualization (Methods). Again, at 10% and 20% contrast levels, there was stronger premicrosaccadic enhancement for dark stimuli. Error bars denote SEM.

We also plotted the ratio of the premicrosaccadic to baseline curves instead of the difference between them (Fig. 3d) (Methods). We found that the dark ratio curve plateaued by 20% contrast, whereas the bright curve did not, again suggesting that there was indeed an effect of luminance polarity on premicrosaccadic enhancement of SC visual sensitivity. To

confirm this statistically, for each neuron, we measured the visual response strength for the trials with premicrosaccadic stimuli (stimulus onsets within the interval from -75 ms to -25 ms relative to microsaccade onset), and we divided it by the visual response strength on the baseline trials of the very same condition (thus obtaining a ratio measure for each condition in each neuron). Then, we performed a two-way ANOVA across all neurons, using the two factors of stimulus contrast and stimulus luminance polarity as the experimental conditions. As expected, there was no statistical significance for the ANOVA factor of stimulus contrast ($F = 0.78$; $p = 0.5405$), since we normalized the firing rates at each contrast level by the visual response strength on the baseline trials of the same condition. However, for stimulus luminance polarity, there was indeed a statistically significant effect ($F = 8.08$; $p = 0.0045$). Therefore, we systematically observed stronger premicrosaccadic enhancement of extrafoveal SC visual neural sensitivity for negative luminance polarity stimuli, and this was particularly the case for the range of 10% to 20% contrasts.

In the above analyses, the neurons' firing rates were expectedly lower for lower contrasts (even on no-microsaccade baseline trials), and the population firing rates on no-microsaccade trials were additionally slightly different for dark and bright contrasts (for example, compare the correspondingly-colored dashed horizontal lines in Fig. 2 across the two luminance polarities). This latter observation is due to slight variability in the preferences of individual sampled SC neurons to either darks or brights in our population [38]. As a result, and as stated above, it might have been difficult to visually compare premicrosaccadic enhancement time courses between darks and brights in Fig. 2. Thus, we next normalized the firing rates within each experimental condition individually (Methods). Specifically, we compared all neurons' visual responses within a given contrast and luminance polarity to the no-microsaccade activity in the very same condition. This way, regardless of the contrast or luminance polarity, and regardless of the absolute response strength to a given stimulus contrast, a normalized population firing rate of 1 would be the no-micoraccade population peak visual response for the given condition and luminance polarity; deviations from this value would indicate either an enhancement or suppression in visual sensitivity (Methods). Using this approach, we obtained the time courses shown in Fig. 4. Each row represents one contrast level, and the two columns separate the two luminance polarities; error bars denote SEM. In each row, the leftward red arrow across the two columns indicates the peak value of the negative luminance polarity curve (in the premicrosaccadic interval), and it helps to compare the peak premicrosaccadic enhancement value to the value observed for the corresponding positive luminance polarity stimuli in the left column. As can be seen, especially for the contrast levels of 10% and 20%, there was substantially stronger premicrosaccadic enhancement of SC visual neural sensitivity with dark than bright contrasts. This is consistent with the results of Figs. 1-3 above. Therefore, we found a specific asymmetry in premicrosaccadic enhacement of SC visual neural sensitivity, particularly favoring dark stimuli of moderate contrast levels.

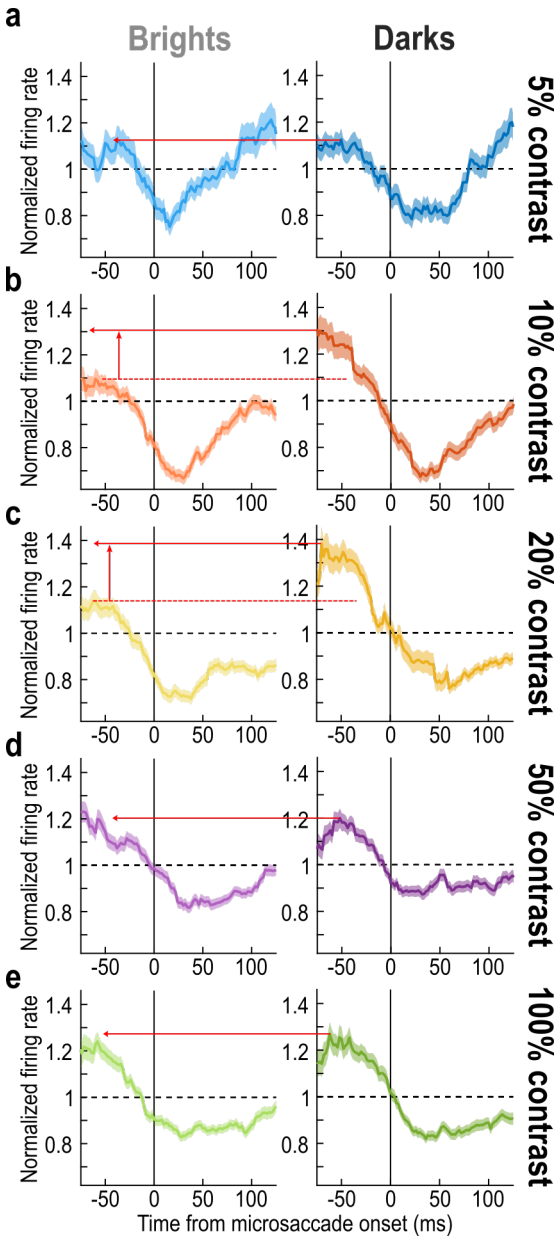

Figure 4 Time courses of perimicrosaccadic modulations relative to the no-microsaccade sensitivity within each contrast level and luminance polarity condition. (a) Each panel is similar, in principle, to what we showed in Fig. 2. However, the difference is that all firing rates were now normalized to the no-microsaccade population sensitivity of the exact condition (contrast level and luminance polarity) being analyzed by the panel. Thus, a value of 1 on the y-axis value indicates the no-microsaccade sensitivity level of the population (for a particular stimulus contrast and luminance polarity) in the absence of peristimulus microsaccades. The panel on the left shows the time course for bright stimuli, and the panel on the right shows the time course for dark stimuli. The leftward red arrow connects the peak of the curve on the right (in the prestimulus interval) to the left panel; this allows comparing the strengths of premicrosaccadic enhancement in the two panels. In this case, there was similar premicrosaccadic enhancement between dark and bright stimuli. **(b, c)** However, for 10% and 20% contrast levels, there was clearly stronger premicrosaccadic enhancement for dark stimuli, consistent with Fig. 3. **(d, e)** This effect dissipated again for higher contrasts. Error bars in all panels denote SEM.

Finally, our analysis approach in Figs. 2-4 above relied on pooling normalized responses across neurons, like we did earlier [11]. We had a good reason for doing so: since each experiment had ten different conditions (5 contrasts and 2 luminance polarities), we were limited in the numbers of trials that we could collect (Methods). As a result, there were even fewer numbers of trials having a stimulus onset within a specific premicrosaccadic time window. Nonetheless, our results above of robust premicrosaccadic enhancement in general (independent of luminance polarity) (Figs. 1-4) were directly consistent with those that we obtained earlier [10]. This increases our confidence in the specific effects of the current study, which we attribute to negative luminance polarities, especially because our earlier experiments purposefully amplified the numbers of trials collected to increase confidence in the observations [10]. Therefore, we are convinced of the robustness of premicrosaccadic enhancement in general, and of the asymmetry associated with dark contrasts that we document in the current study. Having said that, we still decided to inspect the raw firing rates of the individual neurons in our population, with and without microsaccades. We did so by measuring, for each neuron, the visual response strength on baseline no-microsaccade trials and plotting it together with the visual response strength when stimulus onsets occurred within the interval from -75 ms to -25 ms relative to microsaccade onset (Methods).

The results for each contrast and luminance polarity are shown in Fig. 5a-e. For each condition in the scatter plots, the x-axis value shows the baseline no-microsaccade visual response strength of a single neuron, and the y-axis value shows the response strength with premicrosaccadic stimulus onset. The p-value in each scatter plot indicates the result of a pairwise t-test across the population of neurons (comparing microsaccade to no-microsaccade firing rates). Even though we could not always assess significance on a within-neuron basis, due to the limited numbers of microsaccade trials as mentioned above (Methods), we could still assess the population patterns from the raw visual burst firing rates. As can be seen, there was always premicrosaccadic enhancement with the dark luminance polarity stimuli (right column in Fig. 5a-e). For the bright stimuli, significance at the $p<0.05$ level could not be reached for the lowest three contrast levels (left column in the same panels), suggesting that the enhancement was indeed more robust for the dark stimuli. We also performed a three-way ANOVA on all of the data in Fig. 5a-e with the following factors: stimulus onset epoch relative to microsaccades (premicrosaccadic versus baseline), stimulus contrast, and stimulus luminance polarity. There was a significant effect of both stimulus onset epoch ($F = 159.32$; $p = 0.0000$) and stimulus contrast ($F = 314.12$; $p = 0.0000$). These results make intuitive sense since all of our earlier analyses demonstrated a dependence of firing rates on stimulus contrast and also the existence of premicrosaccadic enhancement of SC visual neural sensitivity. Importantly, there were additionally significant interactions between stimulus epoch and stimulus contrast ($F = 4.81$; $p = 0.0007$) as well as between stimulus epoch and stimulus luminance polarity ($F = 5.67$; $p = 0.0173$), implying an interaction between dark versus bright contrasts and the amount of premicrosaccadic enhancement that we observed. Thus, consistent with the results of Figs. 1-4 above, premicrosaccadic enhancement was more robust for dark than bright contrasts in our SC neurons.

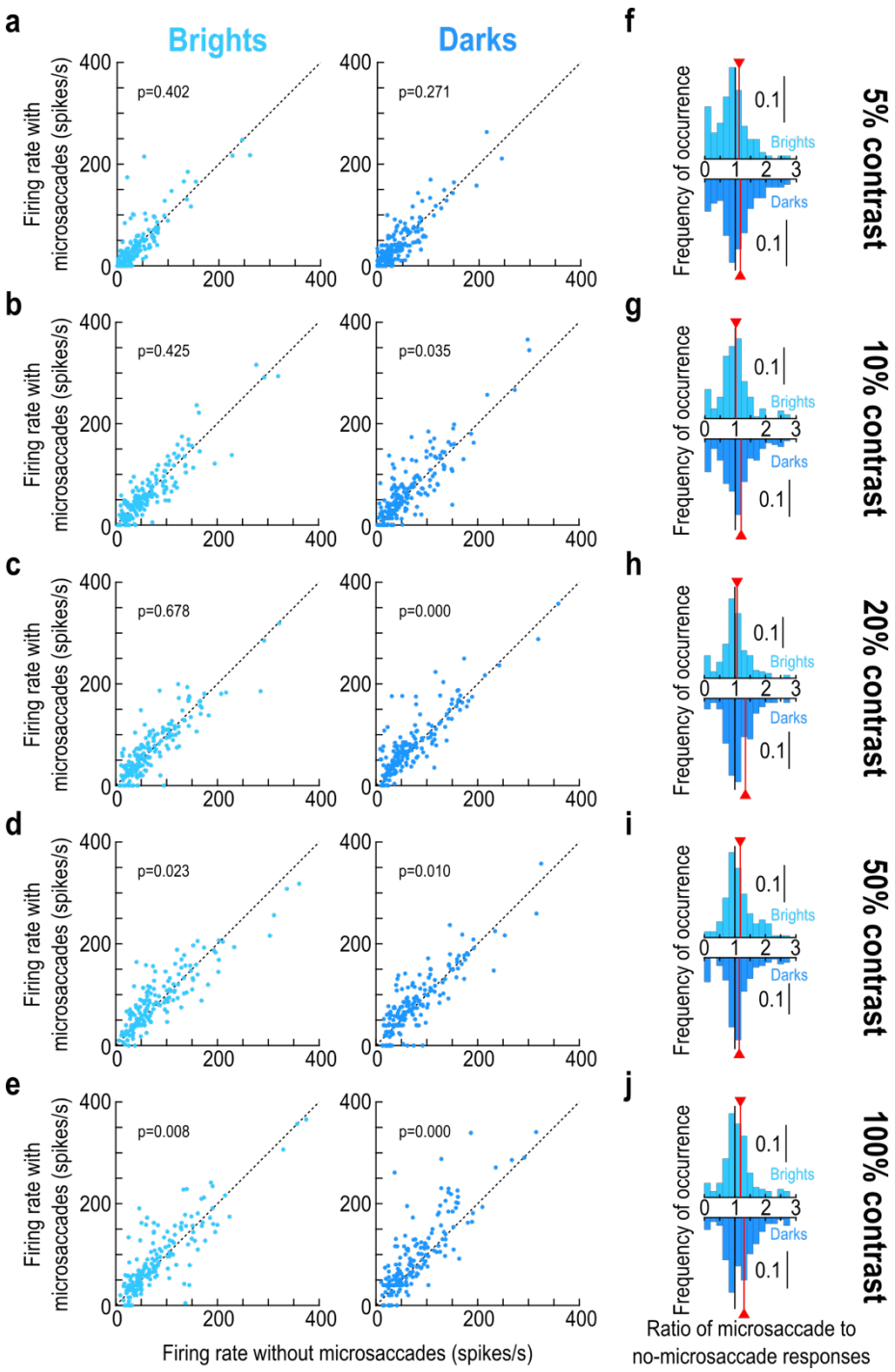

Figure 5 Robustness of the premicrosaccadic enhancement results at the individual neuron level. (a) In each panel, we plotted, for either brights (left) or darks (right), the peak visual response of neurons in baseline (x-axis) and with premicrosaccadic stimuli (y-axis; stimulus onsets within the interval from -75 ms to -25 ms relative to microsaccade onset). Each dot is an individual neuron. The indicated p-value is for a paired t-test across the population. For 5% contrast stimuli, there was barely any premicrosaccadic enhancement, consistent with the contrast sensitivity curves of Fig. 3a, b. **(b-e)** However, at higher contrasts, premicrosaccadic enhancement was robust, especially for the negative luminance polarity stimuli. **(f-j)** For each contrast level, we computed the ratio of the y- to x-axis values in **a**-**e** for each neuron and each luminance polarity. The upward pointing bar plots show the distributions of ratios for bright stimuli, and the downward pointing bar plots show the distributions of ratios for dark stimuli. In each distribution, the vertical red line indicates the mean across the population. As can be seen, there was stronger premicrosaccadic enhancement for darks than brights at 10% and 20% contrast levels (and also at 100% contrast in the bottom panel). n = 166-192 neurons per panel and condition (Methods).

We also inspected the enhancement amplitudes in the scatter plots of Fig. 5a-e more closely. We did so by taking, for each neuron, the ratio of premicrosaccade to no-microsaccade visual response strength, and then plotting the resulting population distributions (Methods) [10]. The histograms of Fig. 5f-j summarize these measurements for each contrast level and luminance polarity, and the red vertical lines indicate the mean values in each histogram across the population. As can be seen, especially for 10% and 20% contrast levels, premicrosaccadic enhancement was stronger for dark than bright contrasts. For example, at 10% contrast, the mean enhancement ratio for bright contrasts was 1.015, whereas it was 1.191 for dark contrasts (Fig. 5g). Similarly, at 20% contrast, the premicrosaccadic enhancement ratio was 1.073 for brights and 1.346 for darks (Fig. 5h). These values are both qualitatively and quantitatively similar to those obtained with our earlier analyses above (Fig. 4). Therefore, all results so far support our interpretation of stronger premicrosaccadic enhancement of SC visual response sensitivity for dark than bright luminance polarities, especially at moderate contrast levels.

As for the question of why brights did not have significant t-test results in three out of the five contrast levels in Fig. 5a-e, this was likely because we pooled across all microsaccade directions and neuron preferred eccentricities in these analyses. In fact, when we checked for an effect of neuron preferred eccentricity, similar to our earlier tests in the original demonstration of premicrosaccadic enhancement [10], we did indeed observe a decreasing trend of sensitivity enhancement with higher preferred eccentricities of the neurons. For example, for 20% contrast bright stimuli, the ratio of microsaccade to no-microsaccade visual response strength exhibited a negative correlation with preferred eccentricity ($r = -0.19$, $p = 0.01$, $n = 188$ neurons), and this was also true for dark contrasts ($r = -0.15$, $p = 0.05$, $n = 171$ neurons); in both cases, the preferred eccentricities ranged from 1 to 19 deg (mean: 5.7 deg, SEM: 0.1 deg). Therefore, even the eccentricity dependence of premicrosaccadic enhancement replicated our original observations [10].

Stronger enhancement independent of neuron preference

Because SC neurons can individually prefer either dark or bright stimuli [38], we next considered the possibility that the results above were restricted to only a subset of SC neurons (for example, those only preferring dark stimuli even in the absence of microsaccades). While this is highly unlikely given the roughly equal numbers of neurons preferring darks and brights across our population [38], we checked this explicitly by repeating the above analyses for only neurons preferring bright stimuli or only neurons preferring dark ones. We labeled the former neurons as “bright-sensitive neurons” and the latter one as “dark-sensitive neurons”.

Figure 6a, b shows the contrast sensitivity curves (similar to Fig. 3a, b) of only the bright-sensitive neurons in our population. The baseline curves (light and dark gray colors) confirm that these neurons did indeed prefer the bright stimuli; this is because the plateau firing rates were higher for bright stimuli (Fig. 6a) than for dark ones (Fig. 6b). Interestingly, the neurons had minimal premicrosaccadic enhancement for bright stimuli of 10% and 20% contrast (black oblique arrows in Fig. 6a), but they had clear enhancement for dark stimuli of

the same contrasts (black oblique arrows in Fig. 6b). Thus, the observations of Figs. 1-5 above still held for only the bright-sensitive neurons of our population: there was stronger premicrosaccadic enhancement at 10% and 20% contrasts for negative than positive luminance polarity stimuli.

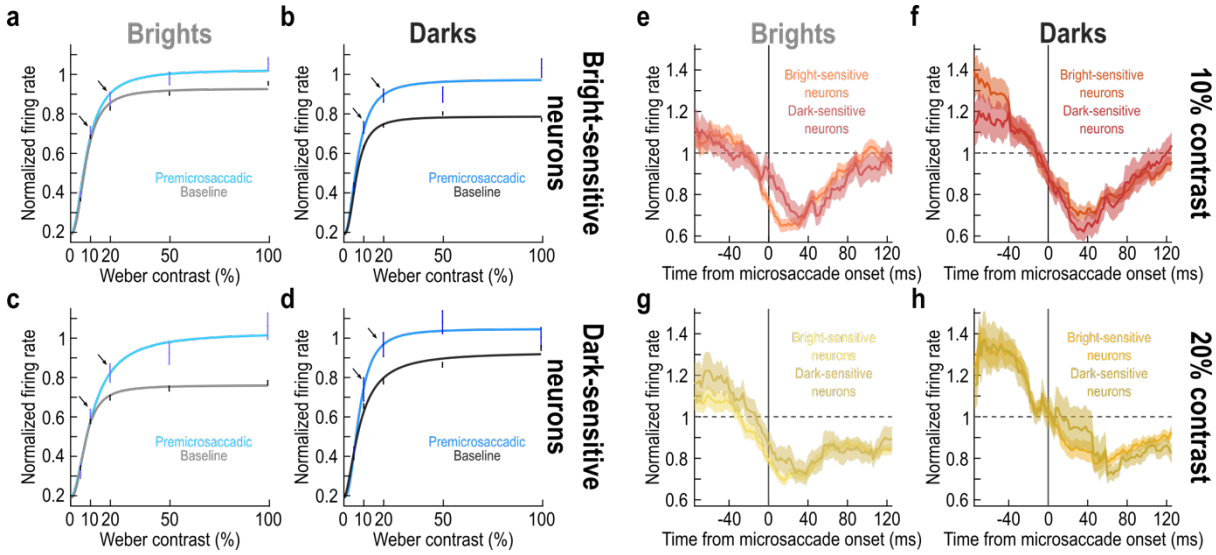

Figure 6 Stronger premicrosaccadic enhancement of SC visual sensitivity for moderate dark contrasts, independent of individual neuron preferences for either darks or brights. (a, b) Similar analyses to Fig. 3a, b, but now for only neurons preferring bright stimuli in their baseline activity. This preference can be seen by comparing the light and dark gray data points and curves across the two panels. In the premicrosaccadic interval, there was still stronger enhancement for dark stimuli at 10% and 20% contrast (black oblique arrows). Note that there was also stronger enhancement at 100% contrast (right panel), suggesting that the preference for brights in the left panel might have caused a ceiling effect on premicrosaccadic firing rates. **(c, d)** Similar analyses for dark-sensitive neurons. Now, there was more of a ceiling effect with dark contrasts than bright ones, and this makes sense because the neurons' baseline responses were now intrinsically stronger for darks. Nonetheless, and despite this fact, there was still stronger premicrosaccadic enhancement for dark contrasts at 10% and 20% contrast levels (black oblique arrows). Thus, the effects of Figs. 1-5 above persisted independent of neuron preference. **(e-h)** Similar analyses to Fig. 4 but after separating neurons according to their preference for either dark or bright stimuli. Neuron preference did not alter the general observation that there was stronger premicrosaccadic enhancement for the dark stimuli. Error bars denote SEM, and all other conventions are similar to Figs. 3, 4.

It is also interesting to note that there was generally stronger premicrosaccadic enhancement for the dark stimuli in the bright-sensitive SC neurons. For example, at 100% contrast, these neurons exhibited a sensitivity enhancement relative to baseline of only 10.15% with the preferred bright stimuli, but they showed a 34.14% premicrosaccadic enhancement for the non-preferred dark stimuli. This could reflect a ceiling effect associated with the preferred bright stimuli. In other words, the neural responses were already almost as strong as could be for the preferred stimuli (by virtue of the stimuli being preferred). As a result, there was less room for premicrosaccadic enhancement on top of the already strong visual responses. Consistent with this, in the dark-sensitive neurons instead, we now

observed generally stronger premicrosaccadic enhancement for the non-preferred bright stimuli than for the preferred dark ones (Fig. 6c, d: 37.19% enhancement for 100% contrast bright stimuli but only 5.63% enhancement for the preferred 100% contrast dark stimuli).

Critically, even in the dark-sensitive neurons (Fig. 6c, d) in which premicrosaccadic enhancement was generally stronger for bright stimuli (especially at 100% contrast), there was still stronger premicrosaccadic enhancement for dark stimuli at the 10% and 20% contrast levels (black oblique arrows in Fig. 6c, d). Thus, even though the firing rates in these neurons were approaching a ceiling effect due to their preference for dark stimuli, at 10% and 20% contrast levels, when there was still room for response strength enhancement compared to 100% contrasts, the dark-sensitive neurons still experienced stronger premicrosaccadic enhancement for dark than bright stimuli. This is all consistent with the results of Figs. 1-5 above. In fact, when we ran the same three-way ANOVA associated with Fig. 5a-e above but now only on the bright-sensitive neurons, all factors and interaction terms were statistically significant ($p<0.0095$ across all comparisons). And, for the dark-sensitive neurons, all main factors (stimulus onset epoch, stimulus contrast, and stimulus luminance polarity) were statistically significant ($p = 0.0000$).

Therefore, at moderate contrast levels, stronger premicrosaccadic enhancement for dark-contrast responses in the SC is a general property of all tested neurons, and it cannot be explained by the intrinsic preference of individual neurons for either bright or dark luminance polarities. However, for 100% contrast stimuli, the absolute value of premicrosaccadic enhancement for either brights or darks can depend on neuron preferences, likely reflecting ceiling effects associated with the preferred luminance polarities.

Consistent with the above, full time courses of perimicrosaccadic modulations in visual sensitivity showed very similar trends in the bright- and dark-sensitive neurons of our population. For example, Fig. 6e-h shows the time courses for 10% and 20% contrasts, in a format identical to that shown in Fig. 4. The only difference is that we now plotted the time courses separately for either the dark-sensitive or bright-sensitive SC neurons. As can be seen, the same trends as those observed in Fig. 4 could be seen, and this happened regardless of neural preferences for darks or brights. Of course, and consistent with Fig. 6a-d, the difference in the premicrosaccadic interval between darks and brights was quantitatively smaller for the dark-sensitive neurons than for the bright-sensitive neurons (due to the potential ceiling effects mentioned above); however, the overall time courses were qualitatively the same for dark and bright stimuli. Thus, premicrosaccadic enhancement of extrafoveal SC visual sensitivity is indeed stronger for dark stimuli at the contrast levels of 10%-20%. Note that we did not show the time courses of the other contrasts just for simplicity, but they were all consistent with the descriptions made above and in Fig. 6. Indeed, when we repeated the two-way ANOVA on the ratio measures of the neurons' firing rates (like we did in association with Fig. 3d above), we found a significant main effect of stimulus luminance polarity ($F = 12.65$; $p = 0.0004$) and a potential interaction between stimulus contrast and stimulus luminance polarity ($F = 2.2$; $p = 0.0671$) for the bright-sensitive neurons. For the dark-sensitive neurons, there was an interaction between stimulus contrast and stimulus luminance polarity ($F = 2.17$; $p = 0.0698$).

Therefore, all results so far indicate that SC neurons robustly exhibit premicrosaccadic enhancement in their visual sensitivity (Figs. 1-3) [10], that such enhancement is specifically stronger for dark luminance polarities of moderate contrast levels (Figs. 4, 5), and that the enhancement is independent of the intrinsic preferences of individual SC neurons for either bright or dark luminance polarities (Fig. 6).

Similar postmicrosaccadic suppression for darks and brights

For the sake of completeness, and because our experiments automatically also provided us with postmicrosaccadic stimulus onsets as well, we close this study by documenting our observations on microsaccadic suppression of SC visual sensitivity. Already in the time courses of Figs. 2, 4, 6, we could clearly see that stimulus onsets after microsaccade onset were systematically associated with suppressed SC visual sensitivity, rather than enhanced sensitivity like in the premicrosaccadic interval. This is consistent with our earlier observations [10-12], and it was additionally evident at the individual neuron level. For example, in Fig. 7a, b, we plotted the responses of the same example neuron of Fig. 1g, h, but this time for postmicrosaccadic stimuli. As can be seen, microsaccadic suppression of visual sensitivity was evident. However, intriguingly, it was not stronger for the dark stimulus onsets. Thus, at least in this example neuron, there was no stronger suppression for dark stimuli.

Across the population, we repeated the analyses of Fig. 3, but this time finding stimulus onsets within the interval from +25 ms to +75 ms from microsaccade onset (Methods). These times were in the same range as the premicrosaccadic time intervals that we tested above, but they were sufficiently long after microsaccade onset to allow correctly probing postmicrosaccadic visual sensitivity. The contrast sensitivity curves of the neurons were expectedly suppressed (Fig. 6c, d) [10], but there was no evident difference between the suppression strengths for brights and darks. Indeed, both the difference (Fig. 6e) and ratio (Fig. 6f) curves demonstrated generally similar suppression effects between darks and brights at 10% and 20% contrasts, which were the contrasts in which we observed the biggest differences between darks and brights in the premicrosaccadic enhancement interval. We also confirmed this statistically by repeating the two-way ANOVA on the underlying individual neuron data of Fig. 6f (just like we did in association with Fig. 3d for premicrosaccadic epochs). That is, for each neuron, we measured the peak visual response on postmicrosaccadic trials relative to the peak visual response on baseline trials of the same condition, and we then related this measure (across neurons) to stimulus luminance polarity and stimulus contrast. There was now no main effect of stimulus luminance polarity across the population ($F = 0.13$; $p = 0.7206$).

We also noted in Fig. 6e, f that at 50% contrast, there seemed to be much weaker suppression for dark than bright stimuli. We are not sure why this is the case. In part, it could be due to the particular choice of time window of analysis that we used. Specifically, the time courses in Figs. 2, 4 revealed a transient increase in visual sensitivity, particularly at 50% contrast, at the +50 ms time point. Whether this is just due to data variability or a real feature of postmicrosaccadic SC visual sensitivity remains to be investigated in the future. However, what is clear is that the differential effects that we observed earlier at 10% and 20% contrasts for premicrosaccadic enhancement were not present at the same contrasts

for postmicrosaccadic suppression (this is also evident by inspecting the postmicrosaccadic intervals in the time courses presented in Fig. 4 above).

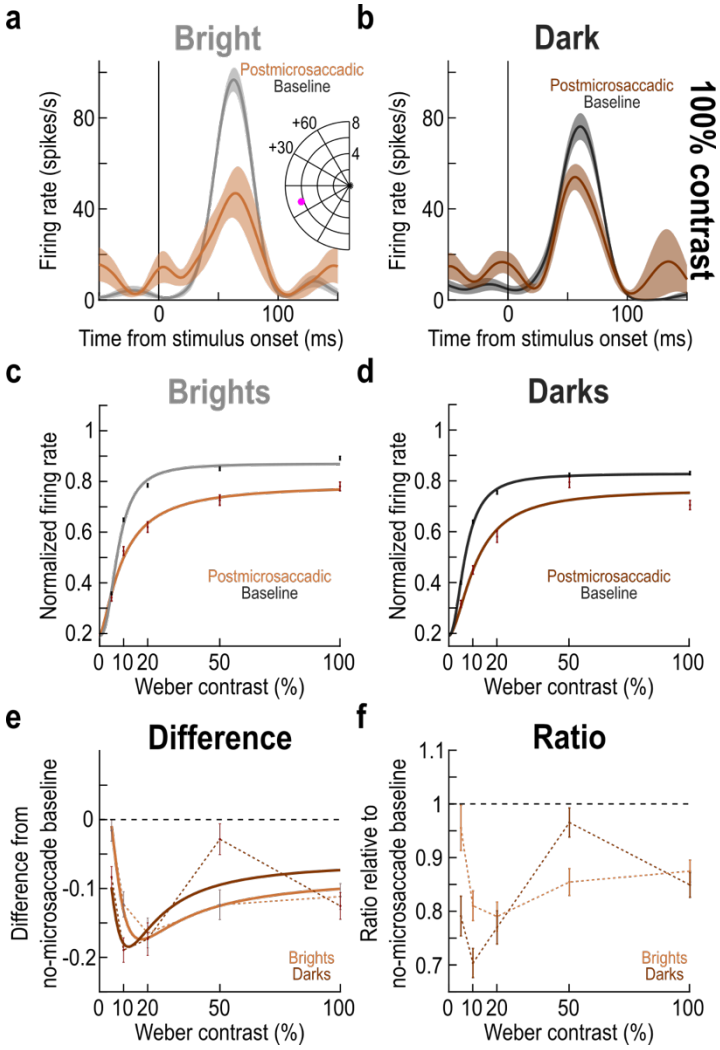

Figure 7 Similar postmicrosaccadic suppression of SC visual sensitivity for positive and negative luminance polarities. (a, b) Visual responses of the same example neuron as that shown in Fig. 1e-h. However, here, we show the responses for postmicrosaccadic stimuli instead of premicrosaccadic ones (note that only in these two panels, we included all trials with stimulus onsets within less than +100 ms from microsaccade onset, just to stay consistent with the example neuron measurement intervals of Fig. 1). As can be seen, there was expected suppression of SC visual sensitivity. n = 31-33 baseline trials and 8-10 postmicrosaccadic trials. **(c, d)** Contrast sensitivity curves like in Fig. 3a, b. Here, we compared baseline curves to those obtained with stimulus onsets occurring within the interval of +25 ms to +75 ms from microsaccade onset. There was similar suppression across luminance polarities (compare **c** and **d**). **(e, f)** Same analyses as in Fig. 3c, d, but for the postmicrosaccadic stimulus trials. At 10% and 20% contrast, there was similar suppression, unlike in the premicrosaccadic intervals in Figs. 1-6. Note that in **f**, we did not apply a fit like in Fig. 3d, because the ratio measure was now not monotonic. However, the data points still show that there was no clear differential effect at 10% and 20% contrasts like in the premicrosaccadic intervals. Error bars denote SEM, and all conventions in this figure are similar to those in Figs. 1, 3.

We also plotted the individual neuron results, like we did for Fig. 5. As expected, we observed robust postmicrosaccadic suppression (Fig. 8), but there was again less of a differential between darks and brights than with premicrosaccadic enhancement. For example, at 10% contrast, the mean population suppression ratio for brights and darks was 24.33% and 23.24% respectively (Fig. 8g), demonstrating a smaller difference across luminance polarities than in the premicrosaccadic enhancement effect. Similarly, the suppression ratio was 17.12% and 21.69% for brights and darks, respectively, at 20% contrast (Fig. 8h), which was again a smaller difference between luminance polarities than in the premicrosaccadic enhancement effects of Figs. 1-6.

Therefore, we observed robust postmicrosaccadic suppression of SC visual sensitivity, but this suppression was generally similar for both dark and bright stimuli. This is also consistent with our observations in human perceptual experiments that saccadic suppression strength was similar for either bright or dark luminance probes [36].

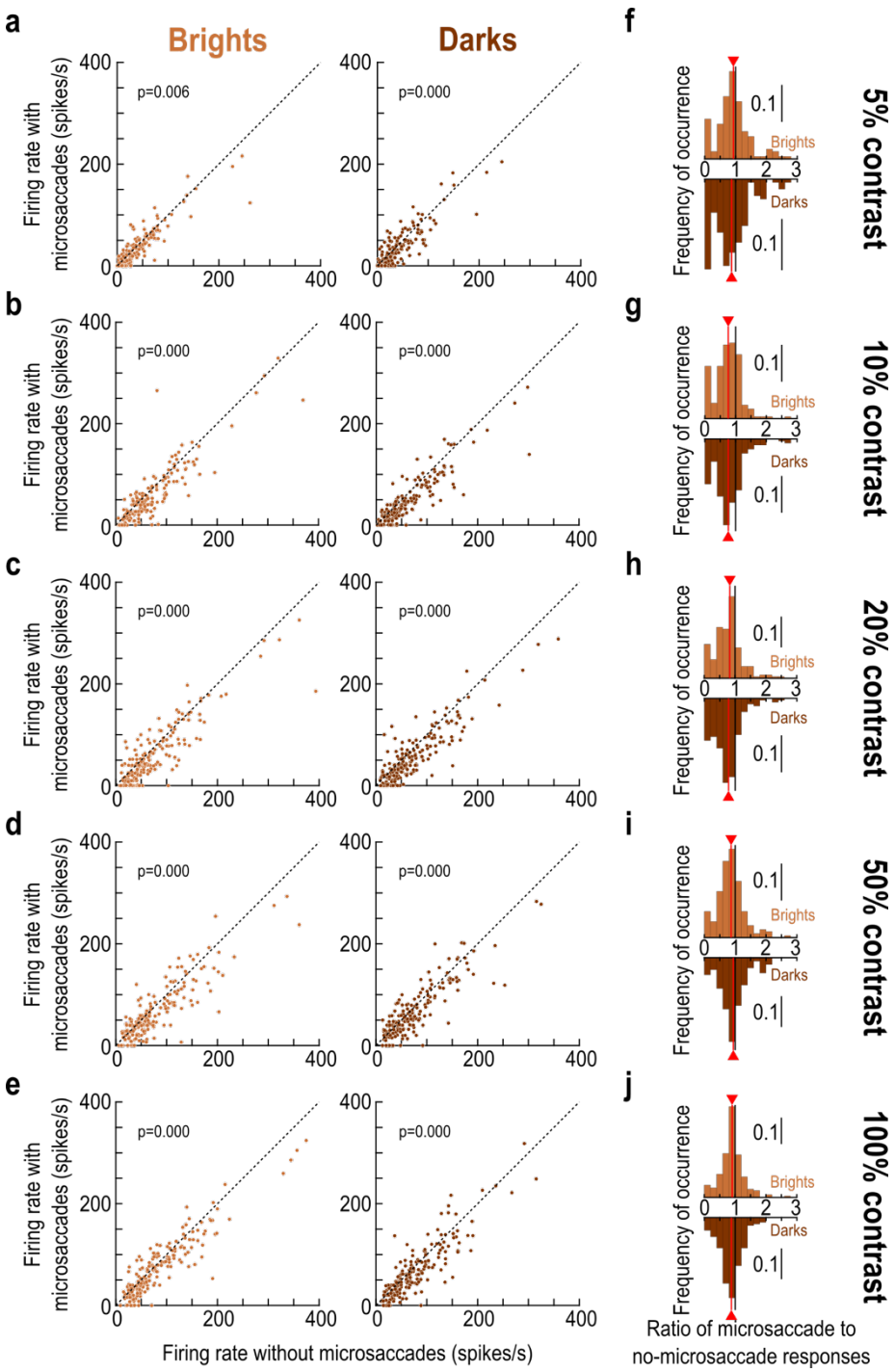

Figure 8 Similarity of postmicrosaccadic suppression between positive and negative luminance polarity stimuli. This figure is formatted similarly to Fig. 5. The only difference is that we now measured visual responses of individual neurons for stimulus onsets occurring within the interval from +25 ms to +75 ms from microsaccade onset. We noticed robust microsaccadic suppression of visual sensitivity. Critically, the histograms in **f-j** show that there were similar suppression strengths across all contrasts for the positive and negative luminance polarity stimuli (compare the red lines in each panel). Thus, the differential effect at 10% and 20% contrast levels in the premicrosaccadic interval was specific to premicrosaccadic enhancement and did not extend to postmicrosaccadic suppression. n = 175-193 neurons per condition and panel (Methods).

Discussion

In this study, we investigated the dependence of premicrosaccadic visual sensitivity enhancement in the SC on luminance polarity. We found stronger premicrosaccadic enhancement for dark versus bright stimuli having contrast levels of 10%-20%. This effect persisted even when we specifically looked at either only dark-sensitive or only bright-sensitive SC neurons. On the other hand, postmicrosaccadic suppression of extrafoveal visual sensitivity was generally similar for both dark and bright stimuli. Our results add to a body of evidence that ON- and OFF-channel visual processing can be different in different parts of the brain's visual processing hierarchy [41-46], and these results also demonstrate that such asymmetries can even appear in the oculomotor system and during the highly specific transient phase of presaccadic vision.

From a visual processing perspective, we find it intriguing to see a dependence of premicrosaccadic visual sensitivity enhancement on luminance polarity in the SC. For one, this helps reinforce the idea that visual processing in the SC is a reformatted version of its inputs [47], and may not simply inherit all of its properties from the primary visual cortex (V1), a major source of anatomical connection to the SC. That is, even if V1 neurons are dominated by dark-sensitive neurons [46], this is not necessarily the case in SC neurons [38]. Indeed, we recently found that it was roughly equally likely to encounter dark- or bright-sensitive neurons in the SC [38], suggesting that it should not be automatically assumed that luminance polarity asymmetries in the cortex would be identically observed in the SC. Our current results add that any asymmetry that may exist in the SC is further sculpted and modified specifically during the premicrosaccadic interval, and we think that this time specificity of the effect is noteworthy. We also think that this effect is unlikely to be similarly observed, whether qualitatively or quantitatively, in V1 perimicrosaccadic visual neural sensitivity.

Even in terms of the direct anatomical projections from V1 to SC, while V1 is indeed dominated by a preference for dark stimuli [46], this asymmetry is less prevalent in the deeper V1 layers directly projecting to the SC [46]. Thus, it is, again, not trivial to suggest that a luminance polarity asymmetry should be observed in the SC, let alone in the premicrosaccadic interval. This interval is very brief (~100 ms), and it is interesting that vision (at least at the level of the SC) during such a transient phase can preferentially favor dark stimuli. At the very least, we may safely conclude that at a specific time point in relation to microsaccades, the strong alterations in vision that are already known to take place in the SC [9-12,15] must also include a preferential enhancement of extrafoveal sensitivity for negative luminance polarity stimuli. It will be important in future studies to consider the functional implications of this observation. For example, there could be ecological benefits to differential processing of negative luminance polarity stimuli in general [48-51], and we can now begin to ask whether ecological and functional benefits may also exist in the premicrosaccadic interval as well.

We can already think of one potential functional implication of our results. Specifically, microsaccades are related to peripheral covert visual attention [20,52]. Moreover, we showed

709 previously that there could be an almost deterministic link between the occurrence of
710 individual microsaccades and the observation of attentional performance alterations in any
711 individual given trial [15,20]. Indeed, we also showed that this link between individual
712 microsaccades and perceptual performance in attentional cueing tasks is specifically
713 mediated by premicrosaccadic enhancement of visual sensitivity in different sensory-motor
714 brain areas [9,10], and that premicrosaccadic enhancement (including in perception [15]) is
715 actually entirely sufficient (at least theoretically) to explain classic peripheral attentional
716 cueing effects [15,16]. If that is the case, then our current results suggest that one can further
717 enhance attentional cueing effects in covert visual attention experiments by using negative
718 luminance polarity perceptual discrimination stimuli of intermediate contrast. To our
719 knowledge, classic attentional studies generally either use Gabor-like gratings [53] or bright
720 targets [15,53-55] instead.
721
722 Irrespective of covert visual attention, our results also add to the increasing evidence that
723 microsaccades can have a long-ranging effect on extrafoveal vision [7]. That is, eccentricities
724 approximately one or two orders of magnitude larger than individual microsaccade
725 amplitudes are significantly affected by microsaccade occurrence [9], and it would be
726 important to understand how this happens. One possible mechanism underlying this is
727 lateral interactions in the SC. For example, there are indications that different layers of the
728 SC can exhibit different amounts of lateral excitation and lateral inhibition across the SC
729 topographic map [56-58]. Intriguingly, in the deeper layers, there may be long-range excitation
730 effects [57], which, coupled with the large foveal magnification of neural tissue that is present
731 in the SC topographic map [14], could potentially mediate an excitatory influence of
732 microsaccades on extrafoveal SC visual sensitivity. Having said that, our work is still
733 correlative. That is, it could be the case that it is the enhanced extrafoveal visual sensitivity
734 itself (for whatever reason) that gives rise to a microsaccade. Thus, in future work, it is
735 important to causally test this. Specifically, one can create an experimental condition that
736 exclusively influences microsaccade generation in the absence of peripheral stimulation, and
737 then see what happens in the peripheral SC representation as a result of this. The
738 experimental condition itself could be to control foveal motor error, since it can very
739 potently alter microsaccade directions in monkeys [16,59]. Ongoing experiments in our
740 laboratory along these lines are suggesting that it is indeed sufficient for microsaccade
741 generation itself to cause enhanced peripheral visual sensitivity. Thus, the direction of
742 causality can indeed flow from the act of microsaccade generation to a concomitant
743 influence on peripheral visual sensitivity.
744
745 At the other end of the spectrum, we observed postmicrosaccadic suppression. This is
746 something that we routinely observe in the SC [10-12]. Nonetheless, we find it surprising that
747 there was no clear difference in suppression strength between bright and dark stimuli. In
748 fact, we recently found that in the retinal origin of saccadic suppression [36], there is a highly
749 substantial difference in the suppression of ON- and OFF-channel retinal circuits [60]. While
750 this observation could reflect species differences, we think that it may additionally be
751 consistent with the idea that SC visual responses are distinctly reformatted versions of visual
752 responses in other early visual areas [47]. To test this, one needs to repeat the current
753 experiments, but in V1 (and ideally using the very same animals that are used for the SC
754 recordings). The prediction is that there should be different patterns of saccadic suppression
755 in V1 between bright and dark luminance polarity stimuli. Demonstrating this would support

our intuition that SC visual responses, and how they are affected by saccade generation, are not merely inherited from visual responses in early cortical visual areas [47].

In all, our results show that active vision entails strong interactions between retinal and extra-retinal consequences of eye movements, and that such interactions must also involve intrinsic asymmetries in visual and motor representations of the surrounding environment.

Competing interests statement

The authors declare no competing interests.

Data availability statement

The databases generated and/or analyzed during the current study are available from the corresponding author on reasonable request. The source data for all plots will be uploaded upon publication.

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Methods

Experimental animals and ethical approvals

We analyzed neural data from the same database described recently [38]. Therefore, we did not perform any additional experiments for the current study; rather, we analyzed perimicrosaccadic modulations in the SC from the earlier neural database. These modulations were not documented previously.

Briefly, the experiments involved recording SC visual responses from two (M and A) adult, male rhesus macaque monkeys (*macaca mulatta*) fixating a small spot.

All of the original experiments were approved by ethics committees at the regional governmental offices of the city of Tübingen [38].

Laboratory setup and animal procedures

The monkeys were prepared previously for behavioral and neurophysiological experiments in our laboratory [38,59,61-63], and we tracked eye movements using the magnetic induction technique [64,65]. The laboratory setup was described in detail previously [38]. Briefly, the monkeys sat comfortably in front of a display having a gray background. They fixated a fixation spot, and they received fluid reward for maintaining gaze fixation on the spot until trial end. We recorded SC activity using single- or multi-electrode arrays inserted acutely into the SC and removed after the experiments [38].

Experimental procedures

The original main task of the earlier publication [38], and which we focused on here, was a gaze fixation task. Briefly, the monkeys initially fixated a small, black fixation spot. After several hundred milliseconds, a disc of 0.51 deg radius appeared at the recorded SC response field (RF) location, and it remained on for at least 500 ms. The monkeys were trained to maintain fixation on the fixation spot, regardless of the eccentric disc appearance. Across trials, the disc could have a Weber contrast of 5%, 10%, 20%, 50%, or 100%, with Weber contrast being defined as $100*|I_s-I_b|/I_b$ (I_s refers to the disc's luminance value, and I_b refers to the display background's luminance value). Moreover, the disc could either be brighter than the background (positive luminance polarity) or darker (negative luminance polarity). In the previous publication [38], we only accepted trials into the analysis that did not have any microsaccades occurring near stimulus onset. Here, we specifically analyzed these trials, and we used the trials without nearby microsaccades at stimulus onset as the baseline trials (see below).

Data analysis

Microsaccades were detected in the previous publication [38] using our previously established criteria [66,67].

For every neuron, trials in which there were no microsaccades within less than +/- 150 ms from stimulus onset were considered to be our baseline trials. These were expected to be the trials in which visual responses were least affected by either past or upcoming microsaccades [10,11].

In the current study, we only considered neurons in which stimulus onset resulted in an elevation of a neuron's firing rate in any of our stimulus conditions. Therefore, we excluded the few suppressed neurons from the original study. This gave us a database of 193 SC neurons. We considered all visually-responsive neurons in the SC because our previous work showed that premicrosaccadic enhancement occurs for either visual-only or visual-motor SC neurons [10].

To assess the visual sensitivity of any given SC neuron, we measured the peak stimulus-evoked response of the neuron. We did this by searching, in every trial, for the maximum value of the firing rate curve of the neuron within the interval from +10 ms to +100 ms relative to stimulus onset. This interval was large enough to allow for finding the peak response even when this response was slightly delayed for the lower contrasts [38]. We then

averaged across trial repetitions of a given condition. As mentioned above, for the baseline (no-microsaccade) response, we measured the peak visual response from only the trials without any nearby microsaccades to stimulus onset (within +/- 150 ms). To identify enhancement or suppression in visual sensitivity in association with microsaccades, we measured the peak firing rate (from the same measurement interval relative to stimulus onset), but now for only subsets of trials according to the timing relationship between stimulus onset and the nearest microsaccade onset. For example, to measure premicrosaccadic enhancement, we picked only the trials in which stimulus onset occurred within the interval from -75 ms to -25 ms relative to microsaccade onset. We then found the peak, stimulus-evoked visual response strength from only these trials. If it was larger than the baseline measurement, then the neuron experienced premicrosaccadic enhancement in its visual sensitivity [10]. The example neurons of Fig. 1 illustrate the logic of this analysis (for the interval from -100 ms to 0 ms from microsaccade onset in this particular exemplary case). Also note that our neurons typically had little or no pre-stimulus activity (see the examples of Fig. 1), and, especially because there was no RF stimulus in the perimicrosaccadic intervals that we considered, there was no direct reafference [68] by the microsaccades itself. Thus, we were always measuring the stimulus-evoked visual response, as we also confirmed with several control analyses in our original study [10].

Because perimicrosaccadic modulations in sensitivity have distinct time courses associated with them [10-12], in various analyses, we repeated the above approach, but this time with different time ranges of stimulus onsets relative to microsaccade onsets. In particular, for time course analyses like in Fig. 2, we used a moving time window approach. In each time sample, we only took trials in which flash onset occurred within +/- 25 ms from the chosen time sample. For example, if the time sample was -50 ms, then we took only trials with flash onset occurring from -75 ms to -25 ms relative to the onset of a microsaccade. Then, we measured the peak visual response on those trials, as mentioned above. In the full time course plots (such as in Fig. 2), we moved the time sample in steps of 2 ms, in order to assess the temporal evolution from premicrosaccadic enhancement to postmicrosaccadic suppression.

Because the above approach of time window analysis is data intensive (only small subsets of trials are included in each sampling window), we combined measurements from across neurons in our population analyses, in order to increase robustness of the analyses. This is because we did not have too many trials collected per session; as a result, there were even fewer trials containing microsaccades within a given time window relative to stimulus onset (see, for example, the numbers of trials in Fig. 1). Thus, we combined data from multiple neurons. However, since different neurons have different visual sensitivities, we had to first normalize measurements before combining data from the different neurons. We used an approach similar to the one that we used previously [11]. Specifically, we normalized the firing rate of every trial of every neuron by the neuron's baseline, no-microsaccade, peak visual response measurement. In one approach (such as in Figs. 2, 3), we normalized by the peak response of the neuron from the condition causing the strongest visual burst by the neuron. For example, if a neuron had the strongest baseline visual response for 100% dark stimuli, then we divided the neuron's firing rates in all trials (regardless of condition) by the response for 100% dark stimuli. Similarly, if the neuron was most sensitive to 100% bright stimuli, then the normalization factor for that neuron was the peak visual response for 100% bright

stimuli. The advantage of this approach is that we maintained the relative relationships of response strengths across contrasts, which in turn allowed us to obtain full contrast sensitivity curves (such as in Fig. 3). Moreover, because the population was roughly equally split between preferring dark and bright contrasts, the resulting baseline contrast sensitivity functions were roughly similar for darks and brights with this approach (see, for example, the gray curves of Fig. 3a, b). In the other approach (such as in Fig. 4), we normalized each condition individually. For example, when analyzing enhancement and suppression for 10% contrast dark stimuli, the normalization factor was the baseline, no-microsaccade, peak visual burst measurement from only trials with 10% contrast dark stimuli. After normalizing each neuron's firing rates, we then combined trials from across all neurons [11].

As mentioned above, the first normalization approach of firing rates allowed us to plot full contrast sensitivity curves (such as in Fig. 3a, b) because it did not mask relative response strengths for different stimulus contrasts. To visualize these contrast sensitivity curves, we fit the five contrast measurements of each luminance polarity to a continuous monotonic function of the form

$$f.r.(c) = R * \frac{c^n}{c_{50}^n + c^n} + B \qquad \text{(Equation 1)}$$

where c is stimulus contrast and B, R, n, and c_{50} are the fit parameters [10]. We obtained fits either for the baseline, no-microsaccade, trials, or for trials in which stimulus onset occurred within a given time window relative to microsaccade onset.

When visualizing premicrosaccadic contrast sensitivity curves in relation to the baseline, no-microsaccade, curves, we also took the difference between the two curves, as well as their associated data samples (separately for each luminance polarity; Fig. 3c). The continuous curves in Fig. 3c represented the difference between the continuous curves obtained from the fitting equation above.

We also took the ratio between the premicrosaccadic and baseline visual sensitivity measurements at each contrast (Fig. 3d). We then visualized the ratio for each luminance polarity by fitting the ratio measurements using the same fitting equation above.

We also repeated the above analyses but only for dark-sensitive or bright-sensitive neurons. We defined dark-sensitive neurons as those in which the condition causing the highest visual response strength in baseline, no-microsaccade, trials was from a dark contrast. That is, the neurons preferred dark contrasts in their visual response sensitivity. For example, if a neuron had the highest visual response for 100% dark contrasts, then we classified it as being dark-sensitive. Similarly, we defined bright-sensitive neurons as those in which the condition causing the highest visual response in baseline trials was from a bright contrast. That is, the neurons preferred positive luminance polarity stimuli.

To confirm that our results above held even at the individual neuron level, we additionally plotted raw firing rates premicrosaccadically and in the baseline (no-microsaccade) data. We did this at each contrast level and for each luminance polarity (Fig. 5). We then performed pairwise statistical tests across the population of neurons (using the raw firing rates), and we also plotted distributions of the raw ratio measurements (Fig. 5).

Statistically, we also checked for an effect of premicrosaccadic enhancement and luminance polarity on our neurons using ANOVA's. In one approach, we measured, for each neuron, the peak visual response on premicrosaccadic trials (trials with stimulus onset within the interval from -75 ms to -25 ms relative to stimulus onset), and we divided it by the peak visual response on baseline trials from the very same condition (contrast and luminance polarity). This way, all measurements within a given contrast were relative to the baseline visual sensitivity of that contrast. Then, we input these ratio measurements of all neurons into a two-way ANOVA with stimulus contrast and stimulus luminance polarity as the ANOVA factors. We expected the contrast factor to yield non-significant results because we were normalizing firing rates within each contrast level individually. However, we included it as a factor in the ANOVA anyway as a sanity check, and to also look for interactions between luminance polarity and contrast.

In the second approach, we stuck with raw firing rate measurements (without any normalization), and we included measurements from both baseline trials and premicrosaccadic stimulus trials of a given condition (contrast and luminance polarity). In this case, we used a three-way ANOVA with the following factors: stimulus presentation epoch relative to microsaccade onset (premicrosaccadic or baseline), stimulus contrast, and stimulus luminance polarity. With this kind of ANOVA, a significant main effect of stimulus presentation epoch would confirm the presence of premicrosaccadic enhancement of SC visual sensitivity, and other main and interaction terms would help in evaluating the dependence of premicrosaccadic enhancement on stimulus luminance polarity.

Finally, we repeated all of the above analyses for trials in which stimulus onset occurred postmicrosaccadically (Figs. 7, 8). The only difference was the choice of interval dictating which trials to include. Now, we only included trials in which stimulus onset occurred shortly after, rather than shortly before, microsaccade onset. Specifically, for the statistical tests and for the summary figures not showing full time courses, we included trials in which stimulus onset occurred from +25 ms to +75 ms relative to microsaccade onset. This allowed us to assess postmicrosaccadic suppression strength, rather than premicrosaccadic enhancement, and we used similar statistical tests to the ones that we described above. In the time courses including epochs of postmicrosaccadic suppression, the analysis windows were just the same as those described above for the premicrosaccadic time courses (that is, each time sample on the x-axis involved a measuring interval within +/- 25 ms from that sample point, and the sampling points were stepped in 2 ms steps).

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