Intrasaccadic suppression is dominated by reduced detector gain

Jon Guez
Center for Molecular and Behavioral Neuroscience, Rutgers, The State University of New Jersey, Newark, NJ, USA

Adam P. Morris
Center for Molecular and Behavioral Neuroscience, Rutgers, The State University of New Jersey, Newark, NJ, USA
National Vision Research Institute, Australian College of Optometry, Carlton, Victoria, Australia

Bart Krekelberg
Center for Molecular and Behavioral Neuroscience, Rutgers, The State University of New Jersey, Newark, NJ, USA

Human vision requires fast eye movements (saccades). Each saccade causes a self-induced motion signal, but we are not aware of this potentially jarring visual input. Among the theorized causes of this phenomenon is a decrease in visual sensitivity before (presaccadic suppression) and during (intrasaccadic suppression) saccades. We investigated intrasaccadic suppression using a perceptual template model (PTM) relating visual detection to different signal-processing stages. One stage changes the gain on the detector's input; another increases uncertainty about the stimulus, allowing more noise into the detector; and other stages inject noise into the detector in a stimulus-dependent or -independent manner. By quantifying intrasaccadic suppression of flashed horizontal gratings at varying external noise levels, we obtained threshold-versus-noise (TVN) data, allowing us to fit the PTM. We tested if any of the PTM parameters changed significantly between the fixation and saccade models and could therefore account for intrasaccadic suppression. We found that the dominant contribution to intrasaccadic suppression was a reduction in the gain of the visual detector. We discuss how our study differs from previous ones that have pointed to uncertainty as an underlying cause of intrasaccadic suppression and how the equivalent noise approach provides a framework for comparing the disparate neural correlates of saccadic suppression.

Introduction

Saccades are the fast and frequent eye movements that we make in order to direct the high-resolution fovea at regions of visual interest. We remain almost completely unaware of the sweeping visual input that the moving retina induces during saccades, a perceptual phenomenon termed *saccadic omission* (Campbell & Wurtz, 1978). This omission of self-induced retinal motion is crucial for perceptual stability (Wurtz, 2008). Saccadic suppression, defined as a decrease in visual sensitivity in the ~75 ms (Ross, Morrone, Goldberg, & Burr, 2001) leading up to and during saccades, is thought to contribute to this omission. This study focused on decreased sensitivity to visual stimuli presented during the saccadic eye movement (intra-saccadic suppression).

The computational and neural mechanisms that underlie intrasaccadic suppression remain debated. Some researchers emphasize a retinal origin (Castet, Jeanjean, & Masson, 2001; Richards, 1969), others an extraretinal, or central, origin (Ibbotson, Crowder, Cloherty, Price, & Mustari, 2008; Wurtz & Goldberg, 1972), or the combination of central and retinal mechanisms (Ibbotson & Krekelberg, 2011). One long-standing theory states that brain areas mediating eye movements send a corollary discharge that modulates activity in visual brain areas during saccades (for review, see Ibbotson & Krekelberg, 2011). An alternative view

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holds that spatial uncertainty induced by saccades can explain suppression (Greenhouse & Cohn, 1991; Matin, 1974). Which of these mechanisms is dominant and how these mechanisms interact (if at all) during intrasaccadic suppression have not been established.

We expanded on previous work (Watson & Krekelberg, 2011) that focused on *pre*saccadic suppression by applying Lu and Dosher's (1998) perceptual template model (PTM) to intrasaccadic suppression. The PTM describes a visual detection process in stages. A template stage attempts to filter out irrelevant sensory inputs, such as those arising from locations in space or time that do not contain signal; a gain stage scales all inputs (i.e., signal and noise), a multiplicative noise-injection stage adds noise in a stimulus-dependent manner, and an additive noise-injection stage adds noise in a stimulus-independent manner. Specifically, the PTM allows us to establish whether intrasaccadic suppression arises as a consequence of a change in the template stage, a decrease in gain, or an injection of noise into the visual system.

Watson and Krekelberg (2011) have shown that saccadic suppression of stimuli presented just *before* a saccade is best explained by a gain reduction mechanism. Here we applied the PTM to intrasaccadic suppression. We expected that an uncertainty mechanism could play a larger role than it does in presaccadic suppression. This expectation came from known factors that could lead to spatial uncertainty during saccades, such as perisaccadic changes in receptive fields (Duhamel, Colby, & Goldberg, 1992; Krekelberg, Kubischik, Hoffmann, & Bremmer, 2003; Tolias et al., 2001), perisaccadic mislocalization (Binda, Cicchini, Burr, & Morrone, 2009; Honda, 1989; Lappe, Awater, & Krekelberg, 2000), and a temporarily inaccurate internal representation of eye position (Dassonville, Schlag, & Schlag-Rey, 1992; Honda, 1989; Morris, Kubischik, Hoffmann, Krekelberg, & Bremmer, 2012). Contrary to our hypothesis, the results showed that gain reduction is the dominant mechanism in intrasaccadic suppression.

Methods

Participants

A total of eight subjects (ages ranging from 20 to 30 years) participated in three experiments (wide-signal, narrow-signal, and high-noise). All subjects had normal or corrected-to-normal vision. All subjects, except one author (S1), were naïve as to the experiment's purpose. Four subjects (two women) participated in the wide-signal experiment. Four subjects (one woman) participated in the narrow-signal experiment. Four subjects (one woman) participated in the high-noise experiment. The author (S1) participated in all three experiments. The experiments were in compliance with the protection of human subjects as outlined in the Declaration of Helsinki and were approved by Rutgers University's Institutional Review Board.

Stimuli

One challenge in intrasaccadic experiments is presenting a stimulus such that it will appear the same to a fixating and a moving retina. The effect of a (world-referenced) stationary grating moving across a translating retina is called smear. Although smear certainly contributes to lowered visibility during saccades, we were interested in the internal mechanisms leading to suppression. To minimize the effects of retinal smear, our task involved a horizontal saccade across a horizontal grating (Burr, Morrone, & Ross, 1994; Volkmann, Riggs, White, & Moore, 1978). Another factor that contributes to lowered visibility during saccades in everyday vision is forward and backward masking of the intrasaccadic scene by the structured pre- and postsaccadic scenes. By presenting a uniform gray background before and after the stimulus, we minimized this influence.

The stimulus was a horizontal grating (to be detected) embedded within a noise pattern. Stimuli were presented on a 30- $\times$ 40-cm Sony FD Trinitron (GDM-C520) CRT monitor with a resolution of 1024 $\times$ 768 pixels and a refresh rate of 120 Hz. The target grating was oriented horizontally, had a spatial frequency of 0.1 cycles per degree, and was vignetted by a Gaussian contrast envelope with a standard deviation of 2° in the vertical direction (Figure 1b). The peak of the Gaussian envelope was located 4° above or below fixation. The contrast of the sine-wave component was varied from trial to trial according to a Bayesian-adaptive method (Kontsevich & Tyler, 1999) to optimize threshold estimation. The stimulus was flashed for one frame (8 ms).

The external noise pattern consisted of horizontal bars. The noise pattern was added to both the target grating and the background, extending across the entire monitor (Figure 1b). Each bar's luminance offset was chosen from a normal probability distribution with a mean of zero and a standard deviation (σ_e) expressed in terms of percentage background luminance.

In the wide-signal experiment, the target grating's horizontal extent covered the entire monitor. The vertical extent of each noise bar was 0.2°, and the external noise level was varied over the range 10%, 15%, 30%, 60%, and 90%.

In the narrow-signal experiment, the target grating's horizontal extent was 12°. The external noise was the same as in the wide-signal experiment.

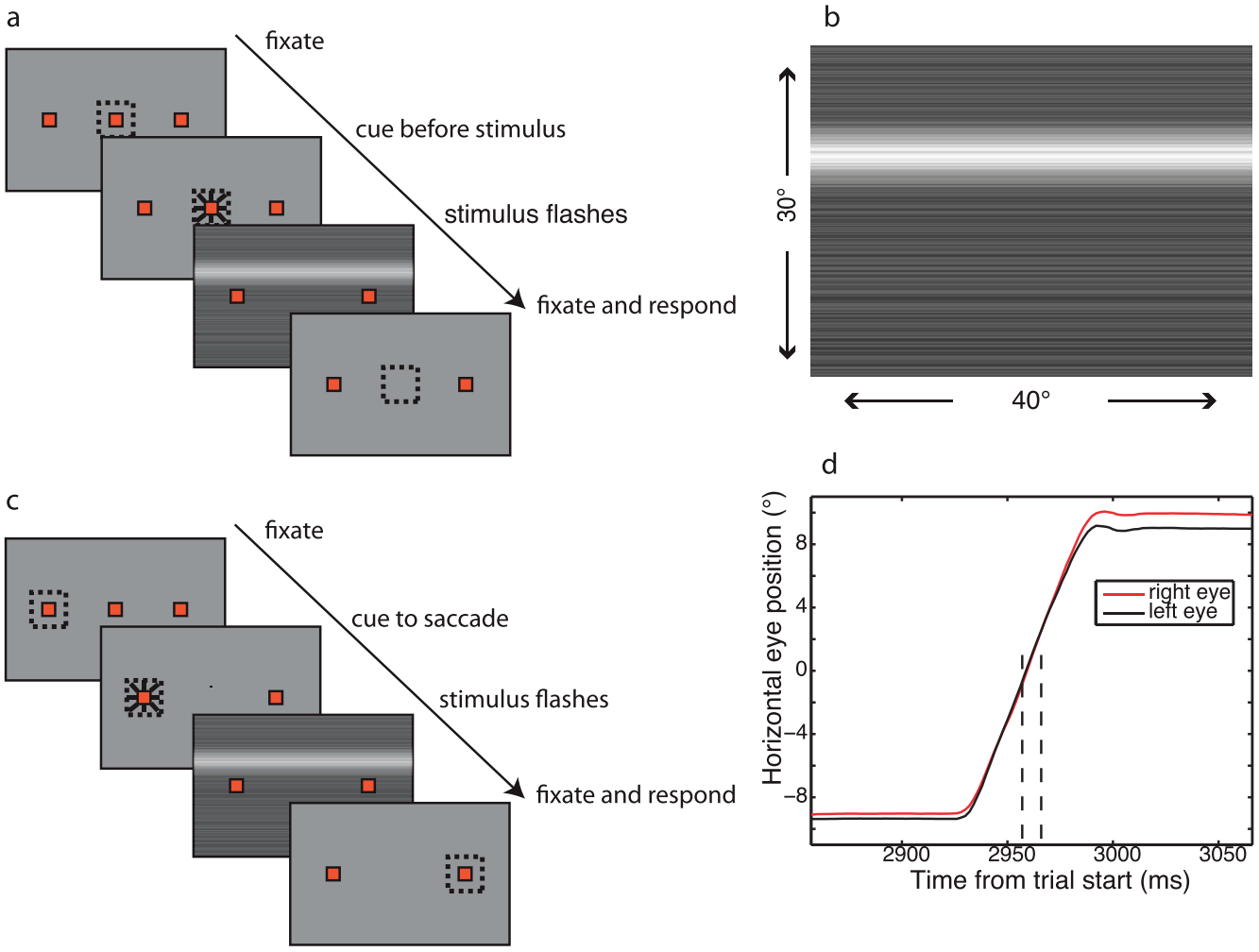

Figure 1. Stimulus and procedure. (a) The fixation condition began with subjects fixating a central dot, which disappeared before a horizontal grating flashed on the screen for one frame. (b) A horizontal grating was used. The vertical coarseness of the entire stimulus (grating and noise) was chosen according to the subject's ability to execute horizontal saccades (see the Methods section). (c) The saccade condition began with subjects fixating a left dot, which blinked, cuing the subject to make a rightward saccade. When the subject's gaze crossed an invisible trigger, the stimulus flashed on the screen for one frame. Subjects completed the saccade and fixated the rightmost dot before responding with a key press. (d) The eyes' horizontal position versus time, illustrating the cued, 18° saccade. The dashed, vertical lines denote the grating stimulus's on- and offset.

In the high-noise experiment, the target grating's horizontal extent covered the entire monitor (as in the wide-signal experiment). The vertical extent of individual bars of the external noise was 0.4°, and the external noise was set to 2% or 60%.

Procedure

Subjects were seated 57 cm from the monitor in a dark booth with a molded bite bar used to restrict head movement. An Eyelink II (SR Research) camera was used to monitor eye movements at a sample rate of 500 Hz and a nominal spatial resolution of 0.1° of visual angle. Trials in which eye position strayed beyond the windows specified below were discarded from analysis.

In all experiments, subjects performed a two-alternative-forced-choice task in which they decided whether a horizontal grating was presented in the lower or upper half of the screen. The grating was presented either during fixation (fixation condition) or midway through a saccade (saccade condition). Subjects responded using a keyboard press and received auditory feedback (low-/high-pitched beep for wrong/right answer) after each response. For saccade conditions (Figure 1c), two red dots (0.3°) appeared on the screen: one 9° to the left, the other 9° to the right of the screen center on the horizontal meridian. Subjects fixated the left dot, which vanished between 640 and 960 ms (jittered) after fixation and reappeared 83 ms later, cuing the subject to make a

saccade to the right dot. To ensure that the stimulus was presented midway through the saccade, the stimulus was presented when the subject's gaze crossed a screen positional threshold. The trigger's position was adjusted such that the stimulus flashed during the middle of the saccade (Figure 1d). Fixation of the rightmost dot had to be maintained within a 2° × 2° window for 100 ms after the saccade.

For fixation conditions (Figure 1a), the same dots as in the saccade condition appeared on the screen (to create a comparable stimulus in both conditions) along with a central fixation dot. Subjects fixated the central dot, and after a jittered delay of 640 to 960 ms, the dot disappeared and the grating flashed approximately 160 ms later for one frame (approximately 8 ms). To ensure that the fixation condition and saccade condition had comparable temporal uncertainty (Morris et al., 2010), the delay between the central dot disappearing and the stimulus appearing was adjusted to match approximately the delay between the cue and the stimulus onset in the saccade condition. Trials from the five external noise levels and two eye-movement conditions (saccade and fixation) were randomly interleaved within the same run.

Analysis

The aim of our analysis was to first estimate an observer's contrast threshold at each level of external noise and then to estimate the parameters of the PTM that best explained the observed threshold-versus-noise (TvN) relationship. These steps were performed separately for the fixation and saccade conditions.

Using the adaptive threshold estimation algorithm (Kontsevich & Tyler, 1999), we determined the maximum a posteriori psychometric function for each external noise level. We chose the 75% correct performance level on these curves to define threshold contrast. The three free parameters of the PTM (β, w, σ_m) were fit to the thresholds across the different external-noise levels using a (inverse-variance) weighted least-squares curve-fitting algorithm (lsqnonlin in MATLAB). Once parameters for the fixation and saccade conditions were estimated, a Wilcoxon signed-rank test was performed to see if any of the parameters differed significantly between fixation and saccade conditions.

Results

Simulation

The PTM (Lu & Dosher, 1998) has been used extensively to model the effects of attention on contrast sensitivity. We apply it here to understand the signal-detection mechanisms underlying intrasaccadic suppression. The model includes an input stage (Figure 2a), a set of signal-processing stages (Figure 2b through d), and an output (Figure 2e) resulting in a decision variable (DV). The elegance of the PTM lies in the qualitatively distinct changes in TvN curves predicted for variations of the parameters that represent each of the processing stages (Figure 2b through d). We applied the PTM to detection thresholds obtained during saccades and during fixation with the goal of ascertaining which of the processing stages were responsible for intrasaccadic suppression.

The PTM implemented here consists of two identical spatial channels: one for the screen location that receives signal-plus-noise and one for the screen location that receives only noise (Figure 2a). The variable c represents signal contrast (or strength). The α parameter is a novel addition to the PTM, which we discuss below. Each of the spatial channels proceeds independently until the final stage, at which their outputs are subtracted (Figure 2e) to produce the DV. If the DV is greater than zero, then the model responds that the signal was in the upper screen position. If the DV is less than zero, then the model chooses the lower screen position. DV's distribution has a total variance (σ_t^2) that can be expressed as a function of the external noise (noise added to the signal before it enters the system) and the model parameters. The detector's sensitivity (d') is then given by the ratio of the signal, $\beta c/(1+\alpha)$, and the standard deviation of the DV, σ_t. Analogous to the derivation in Watson and Krekelberg (2011; their equations 1–3), this allows us to express the contrast threshold for a given level of performance (d') as a function of external noise (σ_e) and the model parameters

$$c = (1+\alpha)\sqrt{2\frac{(w\sigma_e)^2(1+\sigma_m^2)+\frac{\sigma_q^2}{\beta^2}}{\left(\frac{1}{d'^2}-\sigma_m^2\right)}}. \quad (1)$$

Figure 3 shows the shape of TvN curves that the PTM predicts if intrasaccadic suppression is determined by a change in only one of the parameters or dominated by that parameter. In other words, these are the quantitative hypotheses that our experiments tested.

The first processing stage is the perceptual template stage (Figure 2b). This stage's output depends on how well the template matches the input signal, in effect, modeling selectivity. The template's two features are a gain parameter (β) and a tuning parameter (w) controlling the width of the template. The gain (β) amplifies the input, which contains either signal plus external noise (Figure 2a, top) or noise only (Figure 2a, bottom). The template can be thought of as an exclusion term: In general, a tighter template (a smaller w) implies a more focused perceptual analysis on the

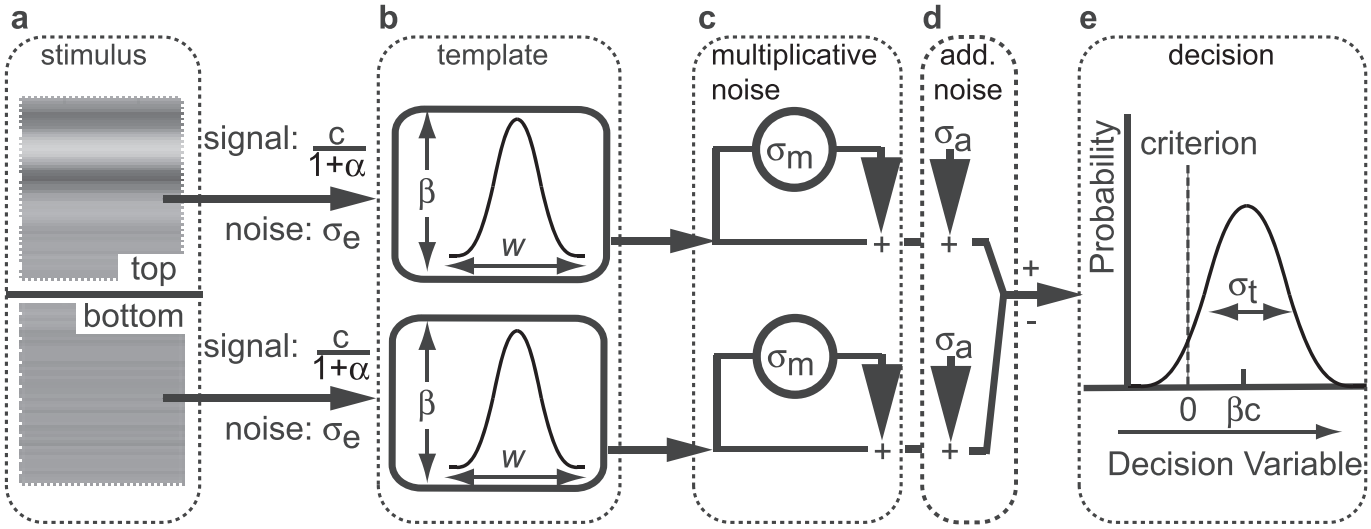

Figure 2. The perceptual template model (PTM). (a) Detectors for the top and bottom halves of the visual field are modeled as signal and noise processed through a series of stages. (b) The first template stage can be thought of as a signal gain and noise exclusion stage: Increasing β increases the output of the template; narrowing *w* will exclude noise (nonrelevant stimuli). (c) Multiplicative noise injects noise into the system in a stimulus-dependent manner. (d) Additive noise injects noise into the system in a stimulus-independent manner. (e) A decision variable is produced by summing the top and bottom detectors; it has a standard deviation, σ_t.

true timing, spatial position, or other characteristic (e.g., spatial frequency) of the stimulus (Lu & Dosher, 1998). In our model, an increase in the template stage (*w*) refers to an increase in spatial uncertainty about the signal. A narrow template (less spatial uncertainty) focuses the detector on only the relevant spatial location and thereby excludes any external noise appearing outside of the signal's location. Therefore, an

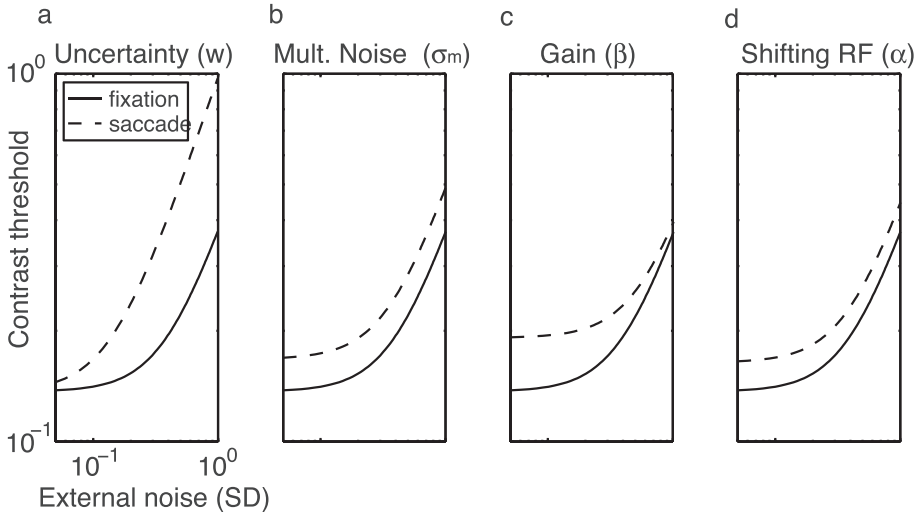

Figure 3. The trends of the PTM under four different parametric accounts of saccadic suppression. (a) An increase in uncertainty (i.e., a wider template) will lead to an increase in detector noise as external noise increases. This is because the increasing external noise will be allowed into (not excluded by) the system. At low external noise levels, this lack of exclusion will not play a critical role in determining thresholds. (b) An increase in multiplicative noise will increase the detector noise in a stimulus-dependent manner, leading to a constant separation (in log units) of thresholds as external noise is increased. (c) Gain reduction will lead to converging thresholds at high external noise. This is because the gain factor amplifies both signal and external noise; hence, it becomes ineffective when the input is dominated by external noise. (d) We extended the PTM with a term that reflects the influence of a shifting receptive field (α), which we modeled as a loss of signal. Our simulation reveals a trend that is qualitatively similar to multiplicative noise injection.

increase in external noise has a minimal effect on thresholds for a narrow template. Now consider a wide template: External noise existing outside of the signal's location would be allowed into the detector. At low external noise levels, this would have only a minimal influence on thresholds. However, at high external noise, the wider template's inclusion of noise would have a dramatic effect on thresholds (Figure 3a), allowing high levels of noise (outside of the signal's location) into the detector. Therefore, if saccadic suppression occurs because of an increase in spatial uncertainty, we expect saccade and fixation thresholds to diverge as external noise increases (Figure 3a).

The second processing stage is a multiplicative noise injection (Figure 2c). The noise added to each channel at this stage is stimulus dependent because the output of the template stage is scaled by the parameter (σ_m). Because this noise injection is scaled by both the signal and noise, the PTM predicts that detection thresholds will be influenced equally at both low and high external noise levels (Figure 3b). Therefore, if saccadic suppression is caused by a stimulus-dependent noise injection, we expect a constant amount of suppression (in log units) across external noise conditions.

The third stage is a stimulus-independent additive noise injection (Figure 2d). Noise with standard deviation σ_a is added to the system, independent of the signal or external noise. It follows that as the external noise is increased, this term will have less of an influence on the threshold. Therefore, the PTM predicts that varying this term will lead to different thresholds at low external noise, when the internal noise is dominated by this additive injection. As external noise is increased (thereby increasingly influencing the system's response) while the additive noise remains constant, the thresholds should converge (Figure 3c). Because this term is mathematically equivalent to gain (β) reduction (see Equation 1), we will consider its effects under the gain (β) parameter.

To model the possible influence of receptive field (RF) shifts that are known to occur at the time of saccades (Duhamel et al., 1992; Tolias et al., 2001), we introduced a novel parameter into the PTM. A spatially shifted detector should reduce the signal but not the external noise (assuming the shifted position is also somewhere on the display); hence, we introduced the term at the earliest stage (Figure 2a). The parameter (α) represents the amount of shift between the detector and the stimulus (when there is no shift, α equals zero, and the input to the detector is equal to c). Our simulation of the influence of α (Figure 3d) shows a trend similar to that of multiplicative noise injection (Figure 3b). Because it is qualitatively similar to multiplicative noise injection (σ_m), we do not consider α in the fits.

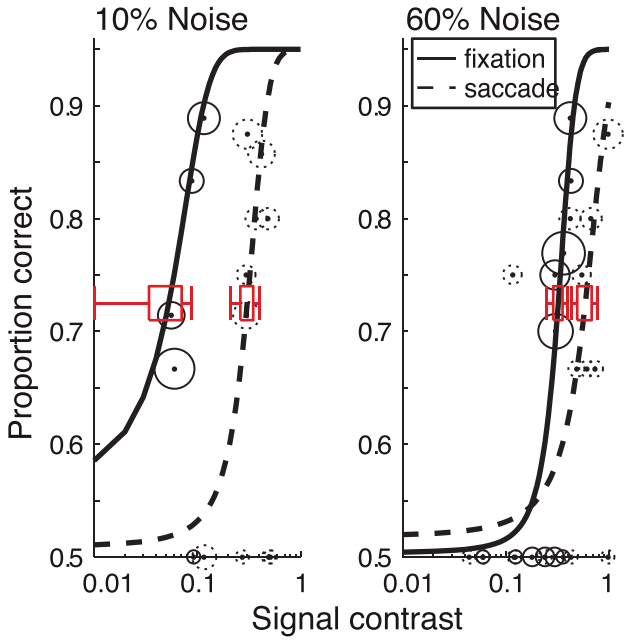

Figure 4. Psychometric curves for the 10% and 60% external noise conditions and for saccade (dashed line) and fixation (solid line) data for one subject (S5). The circle size around the data points represents the number of trials at the given contrast. The curves were obtained using the Bayesian-adaptive method for threshold estimation (see the Methods section). Ninety-five percent confidence intervals are plotted in red for the 75% performance threshold. A total of 1,621 trials were run for this subject, with at least 100 trials for each of the 10 conditions (five external noise conditions for both saccade and fixation).

Experiments

In the first—wide-signal—experiment, we measured contrast thresholds for horizontal gratings covering the entire width of the monitor in varying levels of contrast noise while subjects fixated and during 18° cued saccades. The psychometric curves of one subject obtained for two noise conditions (10% and 60%) are seen in the two panels in Figure 4. The 75% performance level thresholds were extracted for each level of external noise. In each panel, the dotted curve (saccade condition) is shifted to the right from the solid curve (fixation condition), indicating an increase in threshold during saccades (saccadic suppression) for both external noise conditions. In this study, we were concerned with how this saccadic suppression changed as a function of external noise level. This example shows that the increase in threshold induced by the saccade was larger at low (10%) than at high (60%) external noise.

Figure 5 shows thresholds across all external noise levels and for all subjects for the wide-signal stimulus. At low external noise levels, there was a clear

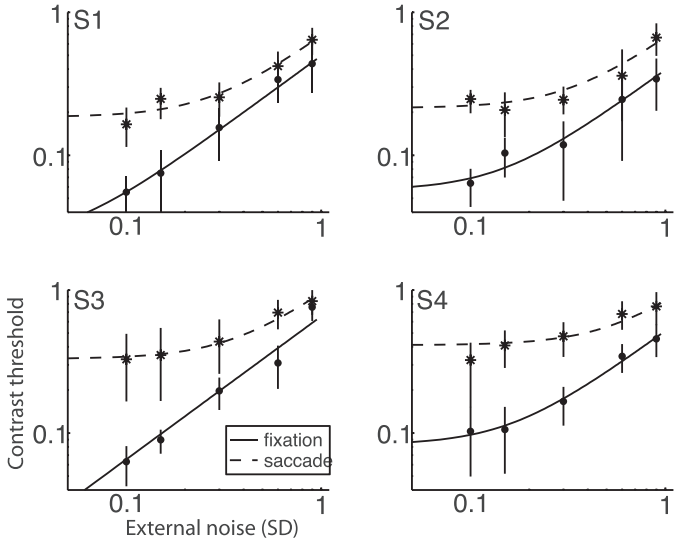

Figure 5. TvN data for the wide-signal stimulus with PTM fits. A horizontal grating extending the width of the monitor (40°) was flashed for one frame (8 ms) during fixation and cued saccades. Fixation data are shown as dots, with the PTM fits drawn solid. Saccade data are shown as asterisks with PTM fits drawn dashed. Each data point is drawn with 95% confidence intervals (thresholds and confidence intervals obtained as in Figure 4). The external noise levels were 10%, 15%, 30%, 60%, and 90%.

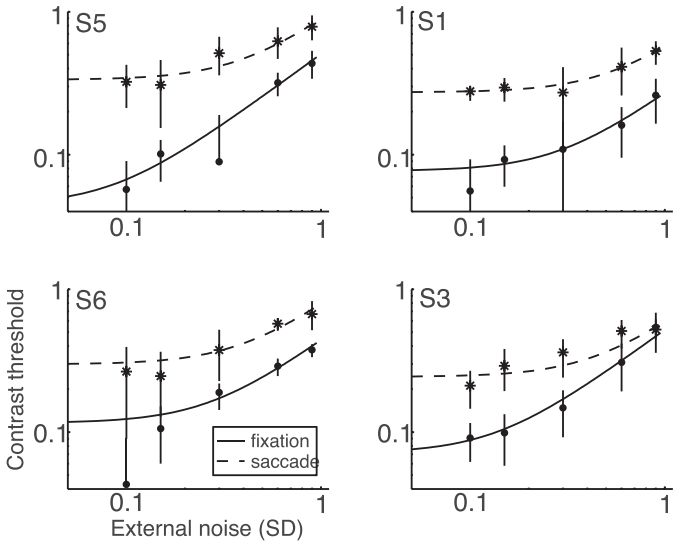

Figure 6. TvN data for the narrow-signal stimulus with PTM fits. A horizontal grating extending 12° was flashed for one frame (8 ms) during fixation and cued saccades. All plotting conventions are the same as in Figure 5.

separation between saccade and fixation thresholds (Figure 5), but this gap decreased as noise increased. This is consistent with a gain reduction mechanism (Figure 3c). However, the thresholds did not converge completely for all subjects.

The stimulus of the wide-signal experiment was chosen to minimize retinal smear and therefore was uniform along the horizontal meridian and extended horizontally 40° (the entirety of the display). One could argue that such a wide target stimulus limits the possible contributions of an uncertainty mechanism. Essentially, any variation in horizontal (but not vertical) extent or location of the template could go unnoticed because the stimulus does not vary in that dimension. To address this, we performed another experiment, now with a horizontally narrow grating (12°). The results of this narrow-signal experiment are shown in Figure 6. The trends were qualitatively similar to the results of the wide-signal experiment (Figure 5): Suppression decreased as external noise increased. Again, this is consistent with a gain reduction mechanism (Figure 3c). To analyze this possibility, we fit each of the TvN curves from the wide-signal and narrow-signal experiments with the PTM, separately for the fixation and saccade conditions (Figures 5 and 6, solid and dashed lines).

Figure 7 compares the best-fitting PTM parameters in the fixation and saccade conditions (wide-signal data points shown as squares, narrow-signal data points shown as triangles). A unity line is plotted for comparison purposes; points lying below the line represent a decrease in that parameter during saccades. The uncertainty parameter and the multiplicative noise parameter did not increase or decrease consistently during saccades, but the gain parameter decreased in seven of the eight subjects. One wide-signal data point (S3) is not displayed on the gain plot because it was two orders of magnitude greater during fixation (and therefore rendered the plot visually less informative); it still obeyed the trend of the other data, decreasing during saccade. A statistical analysis of these parameter changes across all subjects first confirmed the qualitative impression based on Figures 5 and 6: There was a significant intrasaccadic reduction in the gain (β) parameter (Wilcoxon $T = 3.00$, $p = 0.039$). Second, even though the multiplicative noise (σ_m) parameter appears somewhat reduced during saccades (Figure 7), this effect was not significant ($T = 8.00$, $p = 0.200$). We note furthermore that such an intrasaccadic multiplicative noise reduction would not be consistent with saccadic suppression (i.e., it predicts lower thresholds during saccades). Third, the uncertainty parameter (w) was not significantly different during saccades ($T = 16.00$, $p = 0.844$). The same statistical conclusions (a significant effect of gain, but not of multiplicative noise, or uncertainty) were also reached after excluding outliers from the statistical analysis.

Rather than relying solely on this population statistical analysis, we used the PTM to generate a prediction that more clearly disentangles gain, on one hand, and multiplicative noise and uncertainty mechanisms on the other. Notably, if saccadic suppression

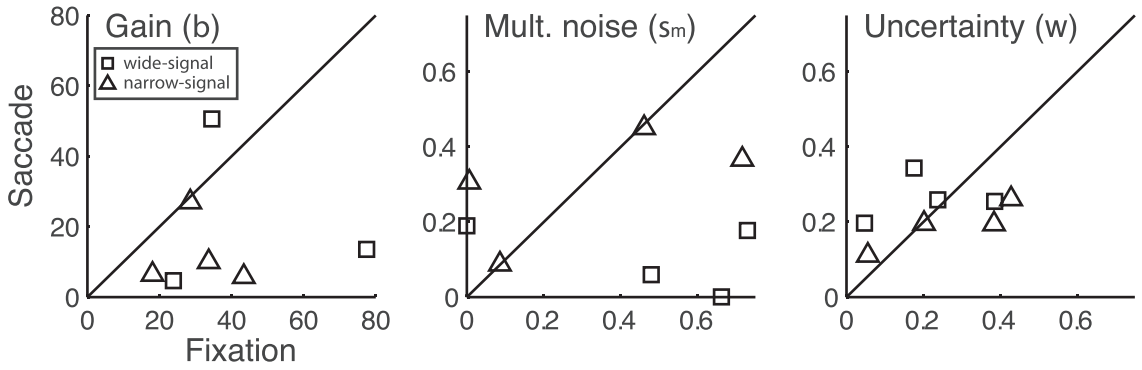

Figure 7. Changes in PTM parameters between fixation and saccade conditions. Each plot shows a PTM parameter's saccade versus fixation value. Each data point represents one subject (wide signal shown as squares; narrow signal as triangles). Points below the line represent a decrease in that parameter during saccades. A Wilcoxon signed-rank test revealed a significant difference in gain during saccades ($T = 3.00$, $p = 0.039$) but not in multiplicative noise ($T = 8.00$, $p = 0.200$) or uncertainty ($T = 16.00$, $p = 0.844$).

were dominated by gain, then TvN curves should converge at higher levels of external noise. To test this prediction, we performed a third experiment, high-noise, in which the size of the external noise bars was increased to match the spatial frequency of the target stimulus more closely (it was not possible to add further contrast noise because it was beyond the capability of the display). Because the template stage (acting as a matched filter) is presumably matched to the signal's characteristics, the external noise is more effective (less of it is filtered out) as its spatial frequency approaches the signal's spatial frequency. Figure 8 shows the results of the high-noise experiment: At low external noise, the saccadic thresholds were significantly higher than at fixation, but at high levels of external noise, the saccade had no significant influence. This is again consistent with a gain change (Figure 3c) but inconsistent with the other mechanisms: An active uncertainty mechanism (Figure 3a), multiplicative noise (Figure 3b), and a shift in the detector (Figure 3d) all predict suppression at high external noise. This confirms that the intrasaccadic suppression is dominated by a gain change.

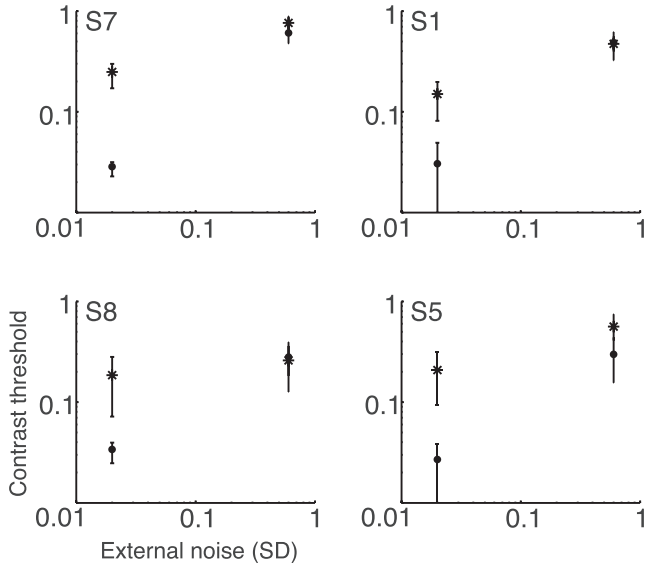

Figure 8. High noise experiment. Fixation thresholds are shown as dots, saccade thresholds as asterisks. The external noise levels were 2% and 60%, but the coarseness of the external noise was increased to more closely match the grating's spatial frequency, increasing the effectiveness of the noise. As predicted by a gain mechanism, the thresholds fully converged at high noise, indicating a gain reduction mechanism.

Discussion

We set out to provide a description of intrasaccadic suppression in terms of basic signal-detection mechanisms. The main finding of our experiment was that a gain reduction is the dominant mechanism in explaining an increase in contrast thresholds for stimuli presented during saccades, as it is for stimuli presented before saccade onset (Watson & Krekelberg, 2011). This conclusion is based on (a) consistency of gain reduction across subjects and (b) the convergence of fixation and saccadic contrast thresholds at very high levels of external noise. The fact that uncertainty about the spatial location of the stimulus contributed little to detection thresholds is surprising as it implies that phenomena such as perisaccadic receptive field shifts and mislocalization have at best a minor influence on intrasaccadic stimulus detection.

Signal-detection mechanisms

We found that gain reduction (β) was sufficient to explain an increase in contrast thresholds during saccades. Our conclusion is consistent with a previous

study using the PTM that found that gain reduction (β) was sufficient to explain presaccadic suppression (Watson & Krekelberg, 2011) and earlier proposals that gain reduction (Burr et al., 1994; Burr & Morrone, 1996) or (the formally equivalent) stimulus-independent noise injection (Diamond, Ross, & Morrone, 2000) in early visual areas may be responsible for increased thresholds during saccades. In addition, these findings are compatible with electrophysiological studies showing pre and intrasaccadic reduction of neural responses. (For review, see Ibbotson & Krekelberg, 2011.)

We set out to test the hypothesis that uncertainty about a stimulus's location is another mechanism that could contribute to intrasaccadic suppression. We considered spatial uncertainty in two qualitatively different ways. One way is a reduction in signal resulting from perisaccadic variability in stimulus localization, such as could arise from RF shifts (Duhamel et al., 1992; Kusunoki & Goldberg, 2003; Tolias et al., 2001) or from perisaccadic error in eye-position signals (Morris et al., 2012). We refer to this as signal mislocalization because the increased detection thresholds result purely from lost signal. Simulations of such a detector show that signal mislocalization predicts suppression effects at both high and low external noise (Figure 3d). We also considered spatial uncertainty as a change in the detection *process* that aims to compensate for the expected intrasaccadic uncertainty about the stimulus's location. For instance, a good detection strategy would be to widen the population from which a detector receives input. This change in the detection strategy is captured in the PTM by a widening of the search template (w). The widening, however, also allows more noise into the system, leading to increasing thresholds as external noise increases (Figure 3a). Hence, although these two uncertainty mechanisms predict quantitatively different TvN curves (Figure 3a vs. d), qualitatively they predict that threshold differences should be found even at high external noise. Our data did not match this prediction; hence, we reject the hypothesis that uncertainty is a dominant contributor to intrasaccadic suppression.

Our conclusion is opposite to that of Greenhouse and Cohn (1991), who found uncertainty to be sufficient to explain a decline in detectability at the time of saccades. It is important here to distinguish between the different characterizations of uncertainty used in our respective studies. Greenhouse and Cohn found reduced receiver-operating characteristic slopes for stimuli flashed during saccades, a result explained by an observer's uncertainty about stimulus parameters. Although their methods show that uncertainty plays a role, they do not necessarily show that uncertainty is a dominant mechanism or that it is sufficient to explain suppression. Although our use of the PTM allows us to compare the contributions of different mechanisms, the Greenhouse and Cohn study investigates only one mechanism (uncertainty) and cannot eliminate the possibility that another mechanism is *more* responsible for saccadic suppression. In an experimental variant designed to prove that uncertainty is sufficient to explain suppression, Greenhouse and Cohn did show a reduction of suppression by flashing a pedestal at the spatial location of the stimulus on both signal and noise (blank) trials. However, in this experimental variant, they also increased the signal intensity to achieve the same detectability. This implies that the pedestal effectively served as a source of external noise. Hence, their finding that the pedestal reduced suppression can be interpreted in the equivalent noise framework as a convergence of TvN curves at high external noise, which is consistent with our findings and points to gain reduction. In a final variant, Greenhouse and Cohn attempted to reduce uncertainty without increasing stimulus intensity by using spatial markers at the site that the stimulus would appear. However, the results of this variant were inconclusive as one of the two subjects tested continued to show significant suppression even when the markers were in place. In summary, Greenhouse and Cohn's results are not inconsistent with ours, but our application of the PTM allows us to compare between mechanisms and to conclude that gain is dominant.

Another main difference between our study and Greenhouse and Cohn's (1991) is that their stimulus was only 1°. This raises the interesting possibility that an uncertainty mechanism could play a more dominant role at a small spatial scale (and therefore in a brain region with smaller receptive fields) but that its influence on the overall detection process is diminished by selecting a sufficiently large stimulus size. We note, however, that a small stimulus necessarily has sharper edges, which move across the retina during the saccade. This provides a qualitatively different visual cue that complicates a fair comparison with the fixation condition. The larger stimulus with a smooth Gaussian edge in our wide-signal experiment greatly reduces this potential confound.

Our conclusion that uncertainty is not a dominant factor in intrasaccadic suppression appears at odds with the physiological findings that motivated our study. However, it is quite easily conceivable that RF shifts (Duhamel et al., 1992), eye position uncertainty (Morris et al., 2012), or RF widening (Tolias et al., 2001) are too small to affect detection relative to the effects of gain reduction (Ibbotson & Krekelberg, 2011). It is also possible that because not all neurons display perisaccadic changes such as RF shifts, the effect of cells that do shift is less critical for detection at the population level.

Neural mechanisms

Equivalent noise analysis can identify dominant mechanisms in terms of signal processing, but it cannot identify the underlying *neural* mechanisms. In the context of saccadic suppression, it is important to point out that it cannot distinguish between the contributions of so-called active and passive backward masking mechanisms. An active gain reduction of visual neurons is certainly compatible with the data (Bremmer, Kubischik, Hoffmann, & Krekelberg, 2009), but our data do not exclude a contribution from passive mechanisms as long as these result in a behavioral effect that is dominated by a gain change. Such neural mechanisms could include backward masking or the Stiles-Crawford effect. The Stiles-Crawford effect reflects an intrasaccadic tilt of the photoreceptors resulting in less light absorption; this scales both signal and external noise and therefore results in a reduction in gain at the earliest stage of the detection process. We note, however, that gain reduction has also been shown to account for presaccadic suppression (Watson & Krekelberg, 2011). In that study, stimuli were flashed just before the saccade, while the eyes were still stationary, so that the gain reduction could not be explained by the Stiles-Crawford effect. Considering the gradual time course of suppression—both behaviorally (Diamond et al., 2000) and in studies of its neural correlates—the most parsimonious explanation is that the same gain reduction mechanism that plays a role presaccadically also operates intrasaccadically. The importance of gain control is also supported by the work of Burr and Morrone (1996). They studied impulse response functions during saccades to stimuli processed predominantly by magno- or parvo-cellular pathways and showed that differences in contrast gain control in these pathways are compatible with behavioral differences in saccadic suppression.

Even though equivalent noise analysis cannot identify a neural mechanism, it can aid the search for such mechanisms by providing a quantitative target. In other words, our findings show that to qualify as an explanation of the behavioral effect of saccadic suppression, any proposed neural mechanism should result in a gain change at the behavioral level. A direct way to incorporate our findings into studies of neural mechanisms of suppression is to compare signal-to-noise ratios of neural responses to a target stimulus embedded in external noise with the predictions of the PTM. This could be a worthwhile extension of single-cell studies in sensory areas already known to show intrasaccadic modulation of activity (reviewed in Ibbotson & Krekelberg, 2011) but also in functional imaging studies (Kleiser, Seitz, & Krekelberg, 2004; Sylvester, Haynes, & Rees, 2005; Vallines & Greenlee, 2006) that have the ability to reveal changes in multiple brain regions at the same time. A quantitative investigation of the neural mechanisms of saccadic suppression within the equivalent noise framework may be able to decide which (if any) among the plethora of proposed neural mechanisms (Ibbotson & Krekelberg, 2011) is the dominant neural mechanism of saccadic suppression.

Conclusions

We employed a visual detection model to describe intrasaccadic suppression in terms of signal-processing mechanisms. We tested and rejected the hypothesis—based on physiological findings—that spatial uncertainty is the dominant factor explaining intrasaccadic suppression. Instead, we found that gain reduction was the dominant mechanism. This quantitative analysis of the dominant signal-processing mechanism is particularly useful as a guiding principle in the search for the neural correlates of saccadic suppression.

Keywords: saccadic suppression, perceptual template model, equivalent noise, eye movements, noise injection, gain reduction, spatial uncertainty

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Corresponding author: Jon Guez.
Email: jon@vision.rutgers.edu.
Address: Center for Molecular and Behavioral Neuroscience, Rutgers, The State University of New Jersey, Newark, NJ, USA.

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