The Influence of Gaze Control on Visual Perception: 
Eye Movements and Visual Stability

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Primates make several saccadic eye movements each second, and yet the retinal motion these movements generate goes unnoticed. Saccadic suppression is a profound loss of visual sensitivity occurring around the time of eye movements, and it is thought to contribute to visual stability by blunting the perception of self-generated motion. Neurophysiological studies have produced evidence that neurons throughout the visual system, including both the dorsal and ventral streams of extrastriate visual cortex, show a reduction in visual responses or sensitivity around the time of saccades. However, the source of this suppression remains unknown. We review evidence that oculomotor regions such as the superior colliculus and frontal eye field may play a role, as well as anatomical data that place constraints on possible mechanisms of suppression.

In spite of the constant shifting of our gaze from one stimulus to another, we perceive the visual world to be a stable and continuous whole (Fig. 1A,B). By comparison, a quick displacement of an eye with one’s finger results in a marked perception of visual motion. This difference suggests the existence of some form of active mechanism that contributes to perceived stability during centrally generated eye movements. Although substantial progress has been made in understanding the neural mechanisms of visual processing during passive fixation, saccadic eye movements (saccades) punctuate these fixations approximately two to four times each second during free viewing. The mechanisms by which the brain compensates for the visual disruption that eye movements introduce remain largely unknown. A recent body of evidence implicates the saccadic system in the modulation of visual cortical representations and of perception (e.g., Moore 2006; Squire et al. 2013), but large gaps remain in our understanding of the neural circuitry that could specifically underlie the illusion of stability during eye movements.

One psychophysical phenomenon thought to reflect an active mechanism in perceived stability during eye movements is saccadic suppression. Saccadic suppression is a profound loss of visual sensitivity that takes place around the time of saccadic eye movements (Fig. 1C). For example, when human observers are asked to report the presence or absence of a visual probe appearing just before, during, or after a saccade, their contrast detection thresholds show an approximately fivefold elevation above those measured when the probe occurs during fixation (Latour 1962; Zuber and Stark 1966; Bridgeman et al. 1975; Burr et al. 1982, 1994; Shioiri and Cavanagh 1989; Diamond et al. 2000; Knöll et al. 2011). Saccadic suppression has also been reported in nonhuman primates, both during the fast (saccadic) phase of optokinetic nystagmus (Mohler and Cechner 1975), as well as during microsaccades (Hass and Horwitz 2011).

Given the frequency at which primates make saccadic eye movements, these findings imply that primate vision is impaired much of the time. The benefit conferred by a reduction of self-generated visual motion during saccades evidently outweighs the disadvantages of frequent visual impairment. Consistent with this, some patients with cerebellar or brainstem lesions experience visual motion in conjunction with abnormal spontaneous eye movements that they report to be disabling and distressing (Corkill and Vijayan 1976; Tilikete and Vighetto 2011). Suppression of vision during saccades therefore appears to play a critical role in perceptual stability across saccades. Here, we review (1) psychophysical studies exploring possible mechanisms of saccadic suppression, (2) neural correlates in the primate visual system, (3) anatomical data that constrain possible mechanisms of saccadic suppression, and (4) functional evidence that the saccadic system plays a role in modulating perisaccadic visual representations in the visual system.

PSYCHOPHYSICAL EVIDENCE FOR A CENTRAL SOURCE OF SUPPRESSION

Psychophysical studies have examined two basic classes of potential mechanisms for saccadic suppression. Active mechanisms for suppression would likely incorporate some form of a centrally generated (extraretinal) signal coupled to the motor command to move the eyes, known as corollary discharge or efference copy. It has
long been argued that an active mechanism must be at play (Holt 1903; von Helmholtz 1925; Sperry 1950; von Holst and Mittelstaedt 1950). Passive mechanisms are those that would depend solely on visual feedback (reafference) generated by the saccade. These could include an inability of neurons early in the visual system to resolve high-speed visual stimuli and/or visual masking, in which the ability to perceive one visual stimulus is compromised by the appearance of another stimulus close by in space and time. Active and passive mechanisms are not mutually exclusive, and support has been found for both.

If passive mechanisms are responsible for saccadic suppression, then moving a visual stimulus across the eye at the high speeds achieved by saccades should reduce its visibility compared with stable snapshots. (C) A selection of psychophysical studies demonstrating that around the time of eye movements, contrast sensitivity (black y-axis) and the likelihood of detecting a visual stimulus (green y-axis) decline sharply.

Diamond et al. (2000) addressed the relative roles of passive and active mechanisms by comparing visual sensitivity when subjects made saccades over an image (the “active” condition) and when the image was moved over their eyes during fixation (the “passive” condition). When this image included high-contrast noise, they found that passive visual motion was sufficient to generate a suppressive effect equal to that observed in the active condition. However, when the image consisted solely of a horizontal sinusoidal grating that was swept horizontally across the eyes, such that there was little visual motion, suppression was observed in the “active” condition but not in the “passive” condition. Thus, saccadic suppression persisted even when passive mechanisms were excluded. This evidence is consistent with the idea that passive mechanisms may be sufficient to explain saccadic suppression under some visual conditions, but an active mechanism or mechanisms are also at play.

SACCADIC SUPPRESSION OF NEURONAL RESPONSES TO VISUAL STIMULI

Neural correlates of saccadic suppression have been observed in the responses of neurons throughout the primate visual system, including the lateral geniculate nucleus (LGN), the superior colliculus (SC), the pulvinar nucleus, primary visual cortex (V1), and extrastriate areas V2, V3, V4, MT, LIP, and VIP (Table 1; Fig. 2). Many of these studies have used strategies to minimize visual reaference and isolate any active mechanisms of saccadic suppression, such as presenting stimuli just before or after eye movements while the eye is still stable (e.g., Ibbotson et al. 2008; Brenmer et al. 2009; Han et al. 2009; Berman and Wurtz 2011), using spatially uniform stimuli (e.g., Mohler and Cechner 1975; Reppas et al. 2002; Sylvestre et al. 2005), and/or comparing responses to active and
passive visual motion (e.g., Robinson and Wurtz 1976; Reppas et al. 2002; Thiele et al. 2002; Ibbotson et al. 2007). Correlates of saccadic suppression are consistently observed under all of these conditions, supporting a role for an active, extraretinal mechanism of suppression.

In addition to these neural correlates, saccadic suppression of perception has also been observed in the macaque. Mohler and Cechner (1975) showed that macaques’ ability to detect briefly flashed stimuli is impaired by 0.5–0.8 log units during the fast (saccadic) phase of optokinetic nystagmus. Similarly to human saccadic suppression, this impairment stretches from ~50 msec before to 50 msec after eye movement initiation and is accompanied by a loss of visual responses in striate cortex. More recently, Hafed and Krauzlis (2010) observed perimicrosaccadic suppression of visual bursts in the SC accompanied by elevation of saccadic reaction times. Finally, Hass and Horwitz (2011) found that the occurrence of microsaccades during stimulus presentation impaired detection of achromatic stimuli.

Table 1. Summary of past studies of neural correlates of saccadic suppression in humans and nonhuman primates

<table>
<thead>
<tr>
<th>Year</th>
<th>Authors</th>
<th>Region</th>
<th>Saccadic suppression</th>
<th>% Suppression (max across time)</th>
<th>% Cells suppressed (if applicable)</th>
<th>Postsaccadic enhancement</th>
</tr>
</thead>
<tbody>
<tr>
<td>1975</td>
<td>Mohler and Cechner</td>
<td>V1</td>
<td>✓</td>
<td>[]</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td>1976</td>
<td>Bartlett, Doty, Lee, and Sakakura</td>
<td>Geniculostrate tract</td>
<td>✓</td>
<td>43% (Magnocellular); 17% (parvocellular)</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>1976</td>
<td>Robinson and Wurtz</td>
<td>SC</td>
<td>✓</td>
<td>[]</td>
<td>61%</td>
<td>✓</td>
</tr>
<tr>
<td>1991</td>
<td>Robinson, McClurkin, Kertzman, and Petersen</td>
<td>Pi/PI</td>
<td>✓</td>
<td>[]</td>
<td>75%</td>
<td>✓</td>
</tr>
<tr>
<td>2001</td>
<td>Ramcharan, Gnadt, and Sherman</td>
<td>LGN</td>
<td>✓</td>
<td>[]</td>
<td>45%</td>
<td>✓</td>
</tr>
<tr>
<td>2011</td>
<td>Hass and Horwitz</td>
<td>V1</td>
<td>✓</td>
<td>17%</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td>2002</td>
<td>Reppas, Usrey, and Reid</td>
<td>LGN</td>
<td>✓</td>
<td>20%</td>
<td>90% (Magnocellular); 21% (parvocellular)</td>
<td>✓</td>
</tr>
<tr>
<td>2002</td>
<td>Thiele, Henning, Kubischik, and Hoffman</td>
<td>MST</td>
<td>✓</td>
<td>[]</td>
<td>68.1%</td>
<td>✓</td>
</tr>
<tr>
<td>2004</td>
<td>Kleiser, Seitz, and Krekelberg</td>
<td>V4 (Human)</td>
<td>✓</td>
<td>50%</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td></td>
<td></td>
<td>V3 (Human)</td>
<td>✓</td>
<td>34%</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td></td>
<td></td>
<td>hMT + (Human)</td>
<td>✓</td>
<td>46%</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IPS (Human)</td>
<td>✓</td>
<td>14%</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LGN (Human)</td>
<td>✓</td>
<td>[]</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td></td>
<td></td>
<td>V1 (Human)</td>
<td>✓</td>
<td>[]</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td>2005</td>
<td>Sylvester, Haynes, and Rees</td>
<td>V2 (Human)</td>
<td>✓</td>
<td>[]</td>
<td>24%</td>
<td>✓</td>
</tr>
<tr>
<td>2006</td>
<td>Sylvester and Rees</td>
<td>V3 (Human)</td>
<td>✓</td>
<td>[]</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td></td>
<td></td>
<td>V1 (Human)</td>
<td>✓</td>
<td>[]</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td></td>
<td></td>
<td>V2 (Human)</td>
<td>✓</td>
<td>[]</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td>2007</td>
<td>Ibbotson, Price, Crowder, Ono, and Mustari</td>
<td>MT/MST</td>
<td>✓</td>
<td>I</td>
<td>83%</td>
<td>✓</td>
</tr>
<tr>
<td>2008</td>
<td>Ibbotson, Crowder, Cloherty, Price, and Mustari</td>
<td>MSTd</td>
<td>✓</td>
<td>81%</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td>2008</td>
<td>Kagan, Gur, and Snodderly</td>
<td>V1</td>
<td>✓</td>
<td>[]</td>
<td>26%</td>
<td>✓</td>
</tr>
<tr>
<td>2009</td>
<td>Bremmer, Kubischik, Hoffmann, and Krekelberg</td>
<td>MT</td>
<td>✓</td>
<td>19%</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td>2009</td>
<td>Hafed and Krauzlis</td>
<td>SC</td>
<td>✓</td>
<td>20%</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td>2010</td>
<td>Cloherty, Mustari, Rosa, and Ibbotson</td>
<td>MSTd</td>
<td>✓</td>
<td>22%</td>
<td>[]</td>
<td>✓</td>
</tr>
<tr>
<td>2010</td>
<td>Han, Xian, and Moore</td>
<td>V4</td>
<td>✓</td>
<td>43%</td>
<td>42.6%</td>
<td>✓</td>
</tr>
<tr>
<td>2010</td>
<td>Berman and Wurtz</td>
<td>P</td>
<td>✓</td>
<td>[]</td>
<td>30%</td>
<td>✓</td>
</tr>
<tr>
<td>2011</td>
<td>Berman and Wurtz</td>
<td>SC</td>
<td>✓</td>
<td>24%</td>
<td>[]</td>
<td>✓</td>
</tr>
</tbody>
</table>

Although paradigmatic differences make precise comparison between studies impossible, the strength of suppression is estimated here by the percent reduction in visual responses across all cells, at the time of deepest suppression with respect to the saccade (column five) and/or the percent of cells suppressed (column six). A number of studies report postsaccadic enhancement, usually following perisaccadic suppression, in the LGN, V1, MT, MST, and VIP (column seven).

[, exact value not reported; entry left blank if not applicable.}
et al. 1994). Evidence for maintenance of chromatic contrast sensitivity during saccades was also found in macaques (Hass and Horwitz 2011). This observation has prompted a number of experiments exploring the hypothesis that saccadic suppression first occurs in the color-insensitive magnocellular pathway of the LGN, sparing representations of color in the parvocellular and koniocellular layers. In support of this argument, suppression may be more common in magnocellular neurons than parvocellular neurons. However, identical patterns of suppression beginning \(\approx 100\) msec before saccades are present in magnocellular, parvocellular, and koniocellular LGN cells (Reppas et al. 2002; Royal et al. 2006). Furthermore, neuronal firing rate responses and blood-oxygen-level-dependent (BOLD) responses to both chromatic and achromatic stimuli appear similarly suppressed in human LGN and V1 (Kleiser et al. 2004; Sylvester and Rees 2006; Hass and Horwitz 2011). These studies also show that saccadic suppression in the LGN is followed by much stronger postsaccadic enhancement, a pattern that is not observed in psychophysical measurements (Burr et al. 1994; Diamond et al. 2000; Knöll et al. 2011) or in downstream visual areas (Bremmer et al. 2009).

Another potential explanation for the luminance selectivity of saccadic suppression could be that suppression is biased toward the relatively color-insensitive dorsal stream, though neurophysiological evidence does not unequivocally support this view. Visual responses are suppressed in dorsal areas V3, MT, and MST (Thiele et al. 2002; Kleiser et al. 2004; Ibbotson et al. 2007; Bremmer et al. 2009; Cloherty et al. 2010). However, strong luminance-selective suppression is present in the ventral stream as well (Kleiser et al. 2004). Han et al. (2009) measured V4 neuronal contrast sensitivity using a staircasing procedure to determine the threshold contrast that elicited a significant response from each neuron (Fig. 3). They found a 0.5 log-unit decrease in sensitivity to brief, luminance-contrast modulated presaccadic visual probes. In contrast to the LGN and V1, perisaccadic sensitivity to red-green modulated equiluminant stimuli was less reduced in V4, paralleling human perception. Although the luminance specificity of saccadic suppression may yet be an important clue to its neural implementa-

**Figure 2.** Summary of mono- and disynaptic pathways between oculomotor areas and visual cortex. Saccadic suppression (blue outlines) is observed in both the dorsal and ventral streams of visual cortex (Kleiser et al. 2004; Bremmer et al. 2009; Han et al. 2009) as well as in oculomotor structures including LIP (Bremmer et al. 2009), the superior colliculus (Robinson and Wurtz 1976; Hafed and Krauzlis 2010), and the frontal eye field (Krock and Moore, abstract in Society for Neuroscience 2014). The superior colliculus sends disynaptic projections (medium orange lines) to dorsal stream but not ventral stream visual areas (Lyon et al. 2010), whereas the FEF (red-orange lines) and LIP (yellow lines) are reciprocally connected with both ventral and dorsal visual cortex (Baizer et al. 1991; Shipp and Zeki 1995; Stanton et al. 1995; Ungerleider et al. 2008; Anderson et al. 2011). Extensive connectivity also exists between these oculomotor regions, including a corollary discharge pathway from the SC to FEF (Sommer and Wurtz 2000, 2002, 2006; Ferraina et al. 2002; Berman et al. 2009). FEF, frontal eye field; LIP, lateral intraparietal area; SC, superior colliculus; PI, inferior pulvinar; LGN, lateral geniculate nucleus; MD, mediodorsal thalamus; MST, medial superior temporal; MT, middle temporal.
tion, the existing data on the subject leave many questions unanswered.

ANATOMIC CONSTRAINTS ON POSSIBLE MECHANISMS OF SUPPRESSION

Although psychophysical and neurophysiological studies support a role for an active suppressive mechanism in saccadic suppression, exactly what form this mechanism might take is unclear. One type of mechanism that is frequently postulated to play a role is corollary discharge, defined as a copy of a motor signal that is used to alter sensory representations. A structure that could produce a corollary discharge would need to have information about impending saccades, implicating particular oculomotor structures. To date, the clearest evidence of a saccadic corollary discharge pathway involves the indirect pathway from the SC to the FEF. Sommer and Wurtz (2008) identified a pathway from the SC to the frontal eye field via the mediodorsal thalamus that appears necessary both for accurate behavioral updating of eye position (Sommer and Wurtz 2002) and for modulating eye movement signals within the FEF (Sommer and Wurtz 2006).

In addition, one might expect corollary discharge signals to directly convey information about impending saccades to the visual system to alter visual representations (Wurtz et al. 2011). Indeed, a simple implementation of a corollary discharge might consist of an inhibitory signal that originates in an oculomotor structure and suppresses visual representations. Yet because long-range cortical connections are overwhelmingly excitatory, it is unlikely that any saccade command signal directly inhibits neurons in visual cortex. For example, ~90% of the projections of FEF neurons to extrastriate area V4 terminate directly onto the dendritic spines of pyramidal cells, precluding the possibility that FEF neurons directly inhibit neurons in this area (Anderson et al. 2011). Another way that oculomotor structures could contribute to perisaccadic insensitivity of visual cortex is via a decrease in excitatory modulation. Berman and Wurtz (2010) used orthodromic and antidromic stimulation to identify a disynaptic relay from the superior colliculus to area MT via the inferior pulvinar. These pulvinar relay cells showed a robust suppression of activity around the time of saccades (Berman and Wurtz 2011). These data are consistent with the idea that the SC contributes to saccadic suppression of visual representations in MT via a reduction of excitation. In addition, recent evidence indicates that SC projection through the pulvinar and into extrastriate visual cortex is exclusively projected to areas of the dorsal visual stream (e.g., MT) and not to ventral stream areas (e.g., V4) (Berman and Wurtz 2010; Lyon et al. 2010). This suggests a possible basis for the relative sparing of chromatic and high spatial frequency vision during saccadic suppression (Burr et al. 1994; Han et al. 2009).

If the SC indeed transmits suppressed visual responses to visual cortex during saccades, where might suppression within the superior colliculus originate? The avian optic tectum (OT), a homolog of the primate SC, is part of a circuit that generates global competitive suppression and sharpens selection of saccade target representations (Mysore and Knudsen 2013; Goddard et al. 2014). The intermediate and deep layer neurons of the OT send topographic projections to the isthmi pars magnocellularis (Imc), the avian homolog of the lateral tegmental nucleus, which in turn sends global inhibitory projections back to the OT. The global inhibition mediated by the Imc is powerful even at great distances from the chosen target.

Figure 3. Saccadic suppression of luminance contrast sensitivity of V4 neurons. (A) The monkey was cued to make a visually guided saccade (red arrow, planned saccade vector; yellow cross, eye position.) A brief visual probe consisting of an oriented bar was presented inside the V4 receptive field at varying times with respect to the saccade onset. Luminance or chromatic contrast of the probe was varied across trials in a staircase procedure to determine the threshold contrast that elicited a neuronal response. (B) For luminance-contrast modulated probes, neuronal sensitivity declined by 50% just before movement onset. There was minimal change to neuronal sensitivity to red-green equiluminant modulated stimuli. (Modified from Han et al. 2009.)
representation. This network could generate global suppression of visual responses within the OT around the time of eye movements. In the future, it will be of interest to determine whether activity in the intermediate and deep layers of the primate SC is necessary for saccadic suppression of visual responses in the superficial and intermediate SC, in visual cortex, and for saccadic suppression measured behaviorally.

**MODULATION OF VISUAL CORTICAL SIGNALS BY THE FEF**

It is now evident that the visually driven responses of neurons within posterior visual cortex are significantly influenced by the activity of neurons within the FEF (for review, see Squire et al. 2013). Of particular importance are the observations that changes in FEF neuronal activity are sufficient to alter the gain of stimulus-driven responses in extrastriate visual cortex (Moore and Armstrong 2003; Armstrong et al. 2006; Ekstrom et al. 2009; Noudoost and Moore 2011a). The FEF is directly connected to neurons within extrastriate visual cortex, including extrastriate areas MT and V4 (Fig. 2; Stanton et al. 1995). The influence of FEF neurons on posterior visual cortex appears to be accomplished, at least in part, via direct excitatory connections from FEF neurons onto pyramidal neurons within retinotopically corresponding columns within extrastriate cortex (e.g., area V4) (Anderson et al. 2011). One current view posits that saccade-related activity within the FEF neurons participates in a recurrent excitatory circuit that can simultaneously amplify visual responses at prospective saccadic target locations and further specify the precise saccade vector needed to foveate particular visual targets (Moore et al. 2003; Schafer and Moore 2007; Hamker et al. 2008; Noudoost and Moore 2011b). In the proposed circuit, the recurrent excitatory interactions between the FEF and extrastriate cortex also facilitates competitive, local inhibitory interactions between cortical columns representing different locations in space, resulting in progressively greater gain disparities between target and nontarget locations (Hamker et al. 2008; Noudoost and Moore 2011b). Such a circuit is consistent with the observation that stimulation of FEF sites both facilitates the visually driven responses at retinotopically corresponding locations and suppresses the responses at noncorresponding locations (Fig. 4; Moore and Armstrong 2003). It is also consistent with the observation that visual receptive fields within visual cortex (Tolias et al. 2001) and within the FEF (Zirnsak et al. 2014) appear to converge toward the location of impending saccadic targets (Hamker et al. 2008). In addition, the proposed circuit also seems consistent with basic phenomenon of saccadic suppression in that it predicts a reduction in visual processing and sensitivity at all locations outside of the target location during saccadic eye movements. Indeed, both the psychophysical and the physiological data to date indicate that around the time of saccades, visual sensitivity at the target location is increased in exchange for large decrements in sensitivity elsewhere (Zirnsak and Moore 2014).

**CONCLUDING REMARKS**

We have reviewed evidence that although numerous psychophysical and neurophysiological studies have showed a role for an active suppressive mechanism in

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**Figure 4.** Modulation of visually driven responses of area V4 neurons with microstimulation of the FEF. (A) Sites within the FEF were electrically stimulated while recording from neurons in area V4. Cartoon shows a side view of the macaque brain. (Top) The locations of the FEF in the anterior bank of the arcuate sulcus and of area V4 in the prelunate gyrus and below the inferior occipital sulcus are shown (both shaded). Monkeys performed a fixation task while oriented bar stimuli were presented inside the recorded V4 neuron’s receptive field (RF) (dotted circle) and at another location outside the RF. The stimulation and recording sites in the FEF and area V4, respectively, could be chosen such that the FEF saccade vector (arrow) and the area V4 neuron’s RF overlapped spatially. (B) The stimulation and recording sites in the FEF and area V4, respectively, could be chosen for each experiment such that the FEF saccade vector (dotted red arrow) and the area V4 neuron’s RF (dotted circle) were either spatially overlapping (left) or nonoverlapping (right). In the overlapping configuration, FEF stimulation enhanced the V4 neuron’s response to visual stimuli appearing in the RF, and these enhancements mirrored the modulations observed during covert spatial attention in that more enhancement was seen for preferred than nonpreferred stimuli (left). In contrast, in the nonoverlapping configuration no response enhancement was produced.
saccadic suppression, many questions remain about the precise form this mechanism might take. Neural correlates of saccadic suppression have been observed throughout the visual and saccadic systems, but these data leave open the question of what the source(s) of this suppression could be. Although corollary discharge seems to be a likely mechanism, direct inhibition of vis-

cual cortex by oculomotor structures is unlikely; instead, structures such as the FEF and SC could contribute to saccadic suppression of visual cortical representations through a loss of excitatory feedback. Future studies are needed to determine whether the SC, the FEF, or other structures in the saccadic system play a causal role in the generation of saccadic suppression of visual neural representations and psychophysical contrast sensitivity.

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