

## REVIEW

Now and Then

## The inevitability of visual interruption

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## Abstract

For successful adaptive behavior, exogenous environmental events must be sensed and reacted to as efficiently as possible. In the lab, the mechanisms underlying such efficiency are often studied with eye movements. Using controlled trials, careful measures of eye movement reaction times, directions, and kinematics suggest a form of “exogenous” oculomotor capture by external events. However, even in controlled trials, exogenous onsets necessarily come asynchronously to internal brain state. We argue that variability in the effectiveness of “exogenous” capture is inevitable. We review an extensive set of evidence demonstrating that before orienting must come interruption, a process that partially explains such variability. More importantly, we present a novel neural mechanistic account of interruption, leveraging the presence of early sensory processing capabilities in the very final stages of oculomotor control brain circuitry.

*active vision; brainstem; omnipause neurons; saccadic inhibition; superior colliculus*

## INTRODUCTION

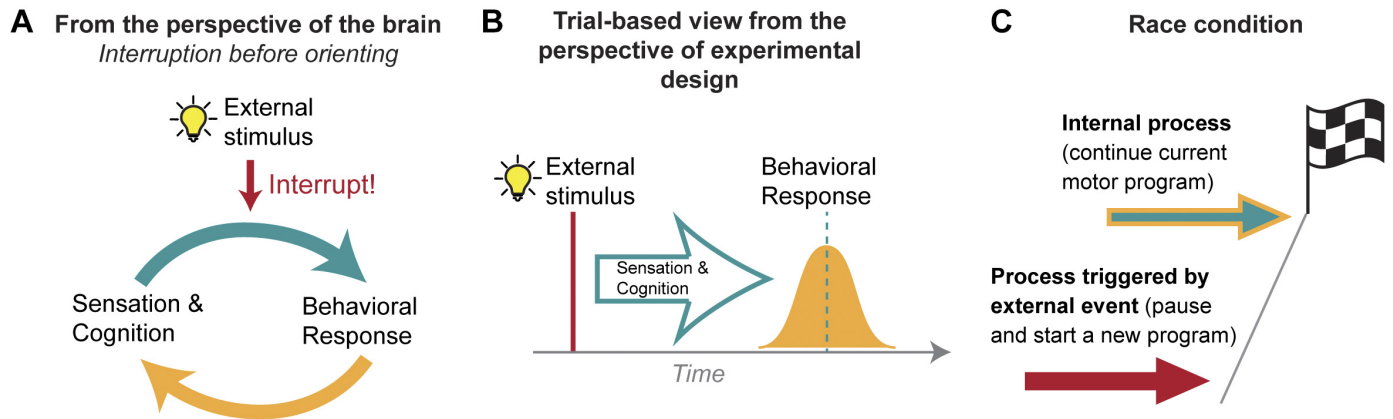
Exploring the surrounding environment is an essential behavior to guarantee survival. At any point in time, the central nervous system is tasked with processing, in parallel, a rich and dynamic stream of information to produce the most ecological behavior. To do so, organisms need to be equipped with “sensors,” enabling stimulus detection, and motor apparatus, executing behavioral responses. In many species, including primates, vision is one of the most important senses, and eye movements are one of the means to respond to changes in the surroundings. However, these two processes are not independent and sequential. They are, in fact, intricate parts of a perpetual loop in which sensation informs action and vice versa (Fig. 1), also referred to as active vision, the combination of sensing and moving the eyes to shape perception (2).

In most instances, the oculomotor response to a selected stimulus is in the form of overt orienting, and animals show a natural preference (often referred to as being “reflexive”) to direct their gaze toward onset stimuli. The orienting reflex (3–5) represents one of the most fundamental response mechanisms to environmental changes. Indeed, attention-

like mechanisms, processing intrinsic (shape, color, and so on) and extrinsic (location, time, and so on) stimulus characteristics, are present in animals as simple as insects (6), and attentional biases can be observed in fish (7). In primates, there is a seemingly strong preference to orient gaze toward peripheral sensory stimuli (8–13), whether they be visual, acoustic, or even tactile, suggesting a form of “exogenous” oculomotor capture by external events.

However, orienting is a costly behavior and as such requires control. Considering that the visual input arrives asynchronously to the active cycles of perception, do we reflexively orient to external stimuli equally well regardless of when a stimulus happens relative to our internal rhythm? Simple introspection would suggest otherwise. Consider, for example, the analogy of talking to a colleague while another colleague approaches mid-conversation. If you were about to start a sentence, you might interrupt and react to the new colleague. In this case, your reaction time to the new colleague is decidedly shorter than if he/she had come late in your sentence, in which case you would be more likely to complete the original sentence before reacting. Thus, reaction time to the same stimulus (the new approaching colleague) will vary greatly based on internal state (how far you





**Figure 1.** Different ways for conceptualizing the variability in orienting responses to identical external sensory events. *A*: most organisms are continuously immersed in a perception-and-action loop in which sensory information is processed to elicit a behavioral response and the results of the behavioral response inform the next steps for perception. While immersed in this loop, external novel stimuli in the environment (red “interrupt” arrow) necessarily come asynchronously to the internal brain state. *B*: historically, psychophysical as well as neurophysiological experiments have been interpreted from a trial-based regime in which experimenters carefully control external stimuli (red vertical line) and study responses to them. Interpreting data from such a trial-based perspective is agnostic of the internal brain state at stimulus occurrence. *C*: functionally, the situation in *A* is analogous to a so-called race condition (!) in electronic circuits: an internal process tries to continue the original motor program and vies for dominance over an asynchronous sensory-driven input that also needs to be oriented toward. Variability in the resulting orienting response to the external stimulus becomes, at least partially, an outcome of how the brain regularizes such a race condition. In our framework, we suggest that inhibition must always precede orienting, necessitating both very early stimulus detection as well as a potent influence on the motor control apparatus at the same time.

are in the sentence). Indeed, even in the most simple and controlled oculomotor orienting paradigms, starting with stable gaze fixation and a sudden jump in target location (Fig. 1*B*), it is well known that reaction times are much longer and more variable than the shortest possible conduction delays from the retina to the eye muscles (14).

The paradox posed by the observation that simple reflexes have longer than expected reaction times, which are also variable, suggests that internal processing does indeed play a role in modulating response timing to incoming sensory information. However, surprisingly little attention has been paid to the possibility that one source of such internal processing is related to the temporal asynchrony between external events and internal brain state (Fig. 1*A*). For example, much like in the conversation analogy above, if an exogenous stimulus comes at a phase in which the internal oculomotor program is still too early in its progression, then orienting to the exogenous stimulus will have a different reaction time than if the internal program was already well on its way. This leads to various observations like whether an express response is expected or not (15, 16).

How might such asynchrony of external sensory inputs be dealt with optimally in orienting? We suggest that a critical answer could be in the implementation of a fast resetting signal that can affect ongoing oculomotor activity and stop orienting before it is deployed, a concept that we refer to with the clause: “interruption before orienting” (Fig. 1*C*). We argue that such insight unifies a large range of observations about oculomotor control, attention, and perception.

In this article, we first clarify the need for a “brake” within the oculomotor system that can help optimize the orienting response to visual onsets in the environment. We then provide converging evidence that such a brake does indeed exist, in a phenomenon called saccadic inhibition, or the sudden momentary cessation of saccades after stimulus onsets (17–20). Importantly, this phenomenon has the same

characteristics in different species and across a variety of eye movement types, suggesting the need for rapid sensory processing even in the very final oculomotor control circuitry, and it also appears obligatorily in a wide range of situations in which abrupt stimulus onsets occur (e.g., a large fraction of experiments in systems neuroscience involving awake, behaving subjects). We end by describing the neurophysiological consequences of exogenous stimulation, and we propose a neural mechanism within the brainstem that can implement the inhibition process described above, effectively resolving a “race condition” (1) caused by conflicting needs to both finish an existing motor plan and orient to a new sensory-driven one (Fig. 1*C*). We conclude that visual onsets inevitably mean visual interruption, even in the most “reflexive capture” of scenarios.

## THE NEED FOR A “BRAKE” IN THE OCULOMOTOR SYSTEM

In an environment characterized by general spatial and temporal continuity, unexpected events are particularly salient, and a successful system exhibits behavioral flexibility to display the most appropriate response to the external input. In some circumstances, the planning of motor responses requires assessing the priority of the novel stimulus as quickly as possible to determine whether to orient to it or not. However, such deliberation cannot even begin before resetting the active perception cycle of Fig. 1*A*, which entails countermanding an internal motor program to allow reacting to the novel stimulus. A manifestation of such resetting is an almost-complete cessation of any new saccades until the active perception cycle is reinitiated (15). Thus, even in the most reflexive of scenarios, a momentary pause in saccade generation is to be expected. Such a pause, being a manifestation of a process resetting the active vision cycle (akin to phase resetting in general), is exactly what can help

to make an assessment on the external input feasible. Without such a “brake” to behavior, an organism might fall victim to uninformative external stimulation or fail to react appropriately. Indeed, without resetting the active vision cycle (Fig. 1A), the behavioral outcome will simply reflect which signal wins the race in Fig. 1C.

In nature, there are several solutions for pausing behavior, spanning from the universal fear response of “freezing” by prey animals (21) to the more sophisticated interruption of vocalization patterns showed by some nonhuman primates (22). However, in the present context, we focus on a specific type of inhibition that starts as a highly stereotyped low-level phenomenon, which can then (potentially) initiate a cascade of repercussions within the oculomotor system and beyond, even setting the stage for higher-level processes, such as urgent decision making (23, 24).

One classic scenario that has been studied for the past 50 years in psychological research is how the response changes when multiple target alternatives are possible. Early studies investigating oculomotor responses showed that such responses were delayed compared to conditions in which only one object was presented. The delay was attributed to the cognitive aspects associated with choosing between multiple simultaneously presented stimuli (25). In later years, interest instead shifted toward more low-level mechanisms in which cognitive factors were reduced by making targets more predictable (26, 27). This was evidenced by terms like “distracting” or “irrelevant” in the literature, with the idea being to characterize how properties of stimulus onsets translate into delayed saccades (28). A number of works followed suit, again mainly focusing on the characteristics of suddenly appearing stimuli (spatial location, shape, contrast, and so on) that influence the triggering of eye movements (29–35). Much less importance was given to the influences of stimulus timing relative to the internal state of oculomotor programming. This was a natural consequence of the way in which experimental protocols were (and are) carried out. We typically use highly precisely controlled stimulus timing relative to individual well-controlled experimental trial events, but the stimuli necessarily remain completely asynchronous with respect to internal brain states (Fig. 1, A and B). On the other hand, we suggest that what is critical for the final behavior to emerge is the time relation between the external exogenous stimulus and the (random) point at which it arrives relative to internal behavioral plans. As we describe in the next section, saccadic inhibition (17–20) recently became a model example of considering the temporal relationship between the outside world and internal state.

## A BRIEF HISTORY OF SACCADIC INHIBITION

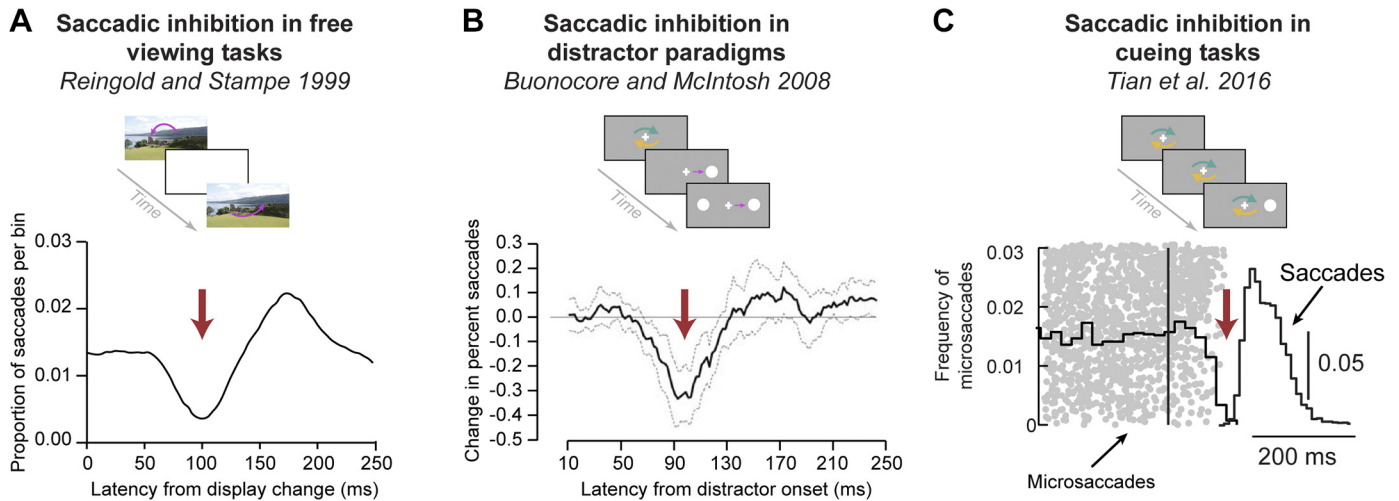
Among the first observations that visual onsets might cause a resetting of the active vision cycle were ones made during gaze-contingent experiments; in such experiments, a transient display change caused momentary changes in fixation duration distributions (e.g., Refs. 36–38). Nonetheless, it was not fully appreciated that the relative timing between the transient event and the stage of the current oculomotor plan was a critical variable for saccades to be inhibited. Only a few years later, saccadic inhibition was formally described by Reingold and Stampe (17), who pointed out that whenever

a subject was involved in a visual scanning task, be it reading or looking at a picture, the probability of making an eye movement dropped dramatically to almost zero ~90 ms after flicker onset. Shortly thereafter, ~120–150 ms following flicker onset, it was possible to see a rebound in the probability of making an eye movement (Fig. 2A). The phenomenon was named saccadic inhibition, and the label was descriptive of the characteristic saccade probability curve representing “one cycle” of inhibition followed by rebound. Later works explored in detail how this saccadic inhibition curve was modulated by the properties of the flicker, such as its size, location, and spatial relationship with the ongoing eye movement (40–42).

The most important aspect of this early research from the perspective of this article is that it stressed the relationship between ongoing active oculomotor behavior and the timing of an exogenous event. Making the display flicker at a random time during visual exploration rendered it possible to align the outcome of an internal process (saccade programming) with an external stimulus onset. This led to the observation that there was an almost deterministic and strict rule of inhibition following stimulus onsets; no matter what, almost no eye movement could be generated ~90 ms after the presentation of a visual transient (and even slightly earlier in monkeys). Indeed, even subjectively fleeting and imperceptible flashes are sufficient to cause saccadic inhibition. Thus, the most critical characteristic of an external stimulus was its temporal relationship with the ongoing oculomotor activity, causing a resetting of the active vision cycle of Fig. 1A (15).

In subsequent work, Reingold and Stampe (18) transformed their original visual exploration paradigm into a more classic trial-based one, as in most current research on eye movements. Similar to what they observed in their original experiments, they reported a consistent reduction in saccade frequency, starting as early as 60 ms after flash onset and reaching peak inhibition at ~90 ms. Because they used a different visual flash this time (two horizontal white bars, one covering the top third of the display and the other covering the bottom third), one of the main insights from this work was that potentially any transient used in classic oculomotor experiments might lead to saccadic inhibition. Nonetheless, this observation remained relatively unnoticed for a few more years.

Inspired by the work of Reingold and Stampe, Buonocore and McIntosh (19) applied the saccadic inhibition rationale to a well-known paradigm within the eye movement literature: the remote distractor effect (28). In this effect, it was found that if a saccade is being planned toward a recently appearing target, then a remote localized flash (called the distractor) occurring within a few milliseconds before or after the target onset could delay the onset of the eye movement by ~15–20 ms. The authors noted that the effect of the distractor was maximum at near simultaneity with the target and faded away for distractor presented later in time. However, instead of analyzing the time of the distractor onset relative to the time of the original target onset for the saccade, Buonocore and McIntosh (19) instead explored the time difference between the distractor onset and the saccade itself. By aligning saccadic reaction times to the onset of the distractor rather than the original target, they found a



**Figure 2.** Visual transients of any kind elicit saccadic inhibition with a similar temporal profile irrespective of the eye movement task. **A:** in a free viewing experiment, Reingold and Stampe asked their participants to search for four targets embedded in an image. At some random time, a full screen flash was presented for 33 ms. When aligning saccadic reaction times to the flash onset, a strong reduction in saccade frequency was observed about 90 ms after the flash. The phenomenon was named “saccadic inhibition.” Figure adapted with permission from Reingold and Stampe (17). **B:** Buonocore and McIntosh (19) applied saccadic inhibition analysis in a classic remote distractor effect paradigm (28) with a trial-based design. The authors observed that small visual distractors could generate saccadic inhibition with an identical time course as in the free viewing experiments, suggesting that trial-based paradigms (Fig. 1B) with small stimuli can still be understood from the perspective of saccadic inhibition. Figure adapted with permission from Buonocore and McIntosh (19). **C:** similarly to A and B, saccadic inhibition can also be seen in a classic covert attentional cueing task, such as the Posner paradigm (39). In this case, microsaccades (gray dots) that are generated while maintaining fixation are aligned to cue onset. In this context, a simple transient (cue) during fixation can lead to microsaccadic inhibition with an almost identical inhibition profile to the one observed for larger saccades. Importantly, this happens before the instructed orienting response (i.e. saccades). Thus, even simple, well-controlled experimental paradigms require coordination between ongoing behaviors (microsaccades) and exogenous events, and this coordination, in turn, requires inhibition happening before the goal-directed behavior. Figure adapted from Tian et al. (16) and reproduced under Creative Commons license CC-BY. In all panels, insets depict trial sequences of each experiment.

clear saccadic inhibition dip in the distribution (Fig. 2B). In other words, the remote distractor effect (a delaying of saccades by distractor onsets) was nothing more than an outcome of saccadic inhibition caused by the distractor onset. At around the same time, similar work was done by other groups applying different versions of distractor paradigms, involving, for example, memory-guided saccades (20).

This unification between distractor effects and saccadic inhibition was an interesting result for two primary reasons. First, it was now recognized that even small flashes (as opposed to large visual transients) cause saccadic inhibition in standard visually guided eye movement tasks; a similar conclusion was reached from the parallel field of microsaccadic research (43), as we describe in more detail below. Second, this was a clear demonstration that a potentially large array of classic paradigms used in vision and eye movement science (using onsets of small visual stimuli) were indeed ideal candidates for saccadic inhibition to occur in. Some examples are the brief probes presented in perceptual discrimination tasks (44, 45) or the visual cues used in classic attentional paradigms (43, 46, 47).

Driven by this intuition, a subsequent stream of articles followed, showing that different characteristics of the so-called distractor (such as size, location, and contrast) all caused saccadic inhibition, but to varying extents (48, 49). These modulatory effects were the first evidence that saccadic inhibition fundamentally reflects the properties of early sensory responses somewhere in the brain (50), although where exactly remained unknown. That is, a stimulus that was intuitively expected to cause strong and early visual

responses in the brain (e.g., high contrast) caused strong and early saccadic inhibition, whereas a weak stimulus caused delayed and weakened inhibition. Moreover, it was shown that such interruption process is an essential component of the “stop” signal studied in classic countermanding experiments (23, 51, 52). These countermanding paradigms are considered to be primary tasks for studying executive control (e.g., 53–55), and saccadic inhibition in them reflects a resetting signal to bring the system back in a regime in which it is able to respond to the external stimulus (whether via an express reaction time or a delayed one). Thus, a pause in saccade generation (Fig. 2) is not only a manifestation of a resetting mechanism of the active vision cycle, to temporally align the brain with the outside world (Fig. 1A), but also a very useful manifestation of such resetting; it allows a possibility for deliberation on whether to ignore the external stimulus or orient toward it (51).

## PARALLEL DISCOVERY OF SACCADIC INHIBITION IN MICROSACCADES

The original Reingold and Stampe experiments involved active visual exploration and helped demonstrate how saccadic inhibition is an obligatory first step in orienting responses in general. Although later experiments moved to trial-based paradigms, it is interesting that even in such paradigms, the active exploration component is still very much present (although often overlooked). Specifically, starting any trial with an “initial fixation” interval, as in a wide array of visual neuroscience experiments (like

the remote distractor effect experiment described above), implies the occurrence of tiny fixational eye movements. If we “zoom in” to such tiny eye movement ranges (typically less than 1° of visual angle), we would now see a constellation of eye movements that are very similar to the ones observed during free and voluntary visual exploration. Microsaccades are mechanistically similar to larger saccades (56), and the slow ocular position drifts that occur in between them have certain analogies with smooth pursuit eye movements (57–59). Given this, one should expect that the same resetting effects that we described so far for large saccades would also apply to these tiny eye movements.

Interestingly, the discovery of “microsaccadic inhibition” paralleled the one of saccadic inhibition, both of them being documented in the literature primarily during the first decade of the century. The first explicit documentation of microsaccadic inhibition was provided by Engbert and Kliegl (43), in which the authors explored microsaccade frequency modulations in classic covert attentional cueing tasks. Earlier, Hafed and Clark (60) had shown that microsaccade directions and times reflect covert attention shifts in such tasks, but the specific paradigms of Engbert and Kliegl (43), with less cue onsets per trial and long precue fixation periods, were more amenable to seeing the classic saccadic inhibition curve. Engbert and Kliegl (43) showed that microsaccade frequency is relatively constant during steady-state fixation, drops strongly after cue onset as early as 50 ms later, and then rebounds later (Fig. 2C). Later research demonstrated that the so-called pause period was modulated by changes in contrast (61) as well as the spatial frequencies of the flashes (50). Thus, again, it seemed that visual properties do matter for shaping the early inhibition, like with larger saccades. In addition, also like larger saccades, sensory signals could also be auditory rather than visual. In fact, auditory stimuli seem to cause, in some cases, microsaccadic inhibition more reliably than saccadic inhibition (42, 62, 63), although the reasons remain unknown.

The use of microsaccades for studying saccadic inhibition was additionally useful for several more reasons. First, there was an interest in learning the neurophysiological mechanisms for microsaccade generation. Therefore, microsaccades in rhesus macaque monkeys were analyzed, again while they maintained fixation during covert visual attention tasks. Hafed and colleagues (47) showed that the same monkeys training on the same paradigms for thousands of trials showed the same effects. Therefore, the inhibition does not really adapt with successive exposure.

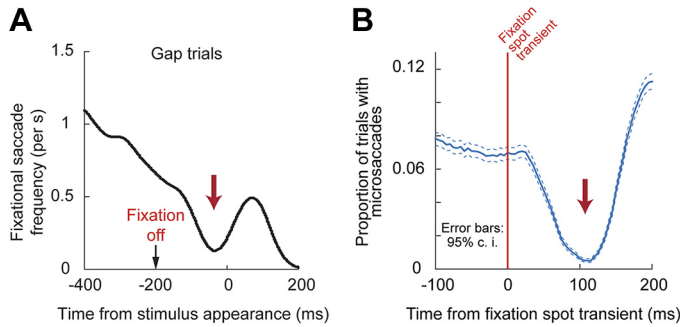
Second, because of the interest in the links between microsaccades and covert visual attention (15, 43, 60, 64, 65), the microsaccadic field explored microsaccade directions during and after the inhibition (15). This is an interesting characteristic of saccadic inhibition because it essentially reveals two components of the phenomenon, one temporal and one spatial. In the temporal domain, reviewed above, it is possible to see at which point following stimulus onset the eye movements are inhibited. In the spatial domain, we see that the saccades surviving the inhibition show a direction dependency with the location of the visual transient. Specifically, while the early microsaccades generated after cue onset are mostly attracted toward the location of the stimulus, the microsaccades that are triggered after the inhibition are biased away

from it (15, 16, 43). Another way of putting it is that microsaccades with directions congruent to stimulus location show a later start of the inhibition compared with the microsaccades opposite from it. As we describe later, these spatial effects have been attributed to different populations of neurons being active in the superior colliculus (SC) at the time of readout for saccade triggering, suggesting a simultaneous interaction between visual and motor signals in the oculomotor system. Importantly, these spatial effects also suggested a potential dissociation from the circuit initiating the inhibition itself, as we discuss in more detail later.

Interestingly, although the temporal profile of larger saccadic inhibition was deeply investigated both in free viewing (40–42) and in trial-based experiments (19, 20, 48, 49, 66), much less is known about the temporal dependency in the spatial domain of large saccades executed following the interrupting signal. Single saccade studies would hint that in some cases, saccades can curve toward the distractor location or even land in intermediate positions between the target and the distractor (also known as the “global effect”) (29, 35, 67, 68). Moreover, spatial dependencies have been reported for large saccades when multiple targets are available (69–71). It would, therefore, be interesting to study spatial interactions in saccadic inhibition in free-viewing paradigms. In this regard, computational models of microsaccades attribute the direction of oscillations, toward and away from the unexpected exogenous stimulus, to saccadic rhythmicity and corrections for foveal motor error (15, 16); extending these ideas to patterns of scanpaths in natural viewing, and to the likelihood of return saccades in such scanpaths, is important.

With the advent of the field of microsaccadic inhibition, it was also possible to now begin studying large saccades by including the full gamut of possible saccades (whether small or large) that occur in trial-based paradigms (Figs. 1B and 2C). Consider, for example, the classic antisaccade paradigm (e.g., Refs. 72, 73), in which subjects are supposed to suppress a reflexive saccade toward an appearing stimulus, and instead program a saccade in the opposite direction. This paradigm has saccades that are often inaccurate, have slower kinematics, and possess longer latencies (74–77). Other than the lack of the visual stimulus during saccade programming in these paradigms, ongoing microsaccade programs at fixation are in preparation when the antisaccade cue appears. Thus, the success or efficacy with which microsaccades are then reset by the “trial onset” necessarily influences whether the subsequent orienting response can take place (78).

Similarly, another classic saccade paradigm, the gap paradigm (79), is known to systematically reduce orienting saccade reaction times (80–83). The defining hallmark of this paradigm is the removal of the fixation spot ~200 ms before the appearance of the peripheral saccade target. It turns out that such fixation spot removal is itself an external sensory stimulus to the oculomotor system, and it is thus expected to cause resetting of the active vision cycle of Fig. 1A. Indeed, studies of the gap paradigm that have explored microsaccades during fixation have found that fixation spot removal was sufficient to cause microsaccadic inhibition (84) (Fig. 3A). What this means, in turn, is that the eccentric stimulus now appears when the oculomotor system is no longer



**Figure 3.** Foveal transients cause microsaccadic inhibition. *A*: in a gap paradigm, the fixation spot is removed approximately 200 ms before saccade target appearance. About 100 ms following fixation spot offset, a strong drop in microsaccade frequency is observed (red arrow). This reduction in microsaccade generation has an identical signature to the (micro)saccadic inhibition effects obtained with peripheral stimuli (Fig. 2C). Figure adapted with permission from Watanabe et al. (84). *B*: during fixation, if a tiny fixation spot undergoes a very brief and transient luminance change, the likelihood of microsaccades toward the fixation spot also drops dramatically (red arrow). This is remarkable because the sensory transient (fixation spot luminance change) is spatially coincident with the saccade goal location. Thus, data from *A* and *B* both suggest that foveal sensory transients, almost perfectly colocalized with the eye movement goals, can lead to strong saccadic inhibition. Figure adapted from Buonocore et al. (24) and reproduced under Creative Commons license CC-BY.

burdened by generating microsaccades, and this can cause the faster saccadic reaction times in the gap paradigm. Incidentally, even a transient change in fixation spot appearance, without its full removal, is also sufficient to cause microsaccadic inhibition (Fig. 3B).

Finally, studying microsaccadic inhibition also illuminated questions of whether saccadic inhibition, in general, is indeed imperative, as we suggest, or merely frequently observed in experiments. Specifically, many experiments with enforced fixation require difficult eccentric perceptual discrimination. As early as Hagedorn and Clark (60), it was clear that microsaccade rate can drop to near zero levels when a difficult exogenous event is expected. Even in monkeys, this was the case (60), and more recent studies on temporal expectation also reveal something similar (85, 86). However, critically, in all of these cases, microsaccadic inhibition still clearly occurs; it just occurs in an active vision cycle (Fig. 1A) that now has a much lower temporal frequency (see, for example, Fig. 3B of Ref. 47). In other words, saccadic inhibition is indeed inevitable, but this idea is not at all equivalent to saying that there is no cognitive control over the oculomotor system. Rather, resetting of the cycle (Fig. 1A) is an unavoidable fact of the asynchrony of the external world with internal oculomotor programs; high-level cognition and behavioral context can reduce the frequency of the active vision cycle enough to avoid excessive costs associated with such resetting. To invoke the conversation analogy above once more, if you expect an important interruption to your conversation, you would most likely reduce the likelihood of starting a new sentence with your colleague. But, if you happen to have already started a sentence because the expected event was not known with full certainty, then the same dilemma of interrupting or continuing your sentence is still present.

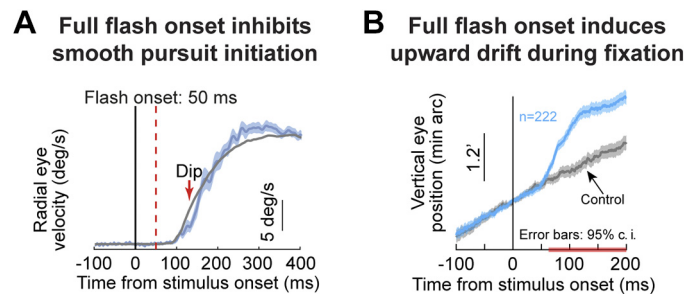
## SACCADIC INHIBITION BEYOND SACCADES

The idea that interruption is essentially unavoidable suggests that its underlying mechanisms could also manifest themselves, in various ways, with different types of eye movements, such as catch-up saccades, smooth pursuit initiation, and sustained gaze following (87, 88). As expected, the effects on catch-up saccades were identical to the classic effects described for regularly sized saccades, both in time and kinematics, including the directional effects seen with microsaccades. More interestingly, the effects on smooth pursuit showed that sensory information related to flash onsets can impact both the initial phases of visual tracking (Fig. 4A) (87) as well as the sustained pursuit phase (87, 88). Importantly, the modulations in velocity reflect the spatial relation between the stimuli and the position of the motion, unequivocally determining in which direction the eyes are pulled (87). Thus, there is a spatial component associated with saccadic inhibition, as revealed by the microsaccadic modulations described above (15).

Intriguingly, a newly discovered phenomenon suggested that even ocular position drift during fixation seems to also be modulated during the “inhibitory” time following stimulus onset, showing a smooth acceleration of the eyes in the upward direction for some stimulus types (89) (Fig. 4B). Although the mechanisms underlying this effect are still unknown, it is indeed suggestive that even the smallest of the eye movement repertoire show modulations in a time window compatible with saccadic inhibition. Again, this suggests a rapid access of sensory information to the very final stages of oculomotor control in the brain, an idea that we elucidate further below.

## NEUROPHYSIOLOGICAL CONSEQUENCES OF EXOGENOUS STIMULATION

The behavioral properties of saccadic inhibition have been explored in great detail. Introducing the concept of temporal relation between external stimulus onsets and internal state motivated developing models of saccade generation that



**Figure 4.** Saccadic inhibition generalizes to smooth pursuit and oculomotor drift during gaze fixation. *A*: a brief flash presented during the initial phase of a smooth pursuit eye movement (by a monkey) suddenly decreases eye velocity about 50 ms afterward (blue curve) relative to a condition in which no flashes are presented (gray curve). Figure adapted by Buonocore et al. (87) and reproduced under Creative Commons license CC-BY. *B*: flash onset can also impact the slowest of the eye movements, ocular drifts during fixation, increasing eye velocity about 50 ms after its presentation (note the different eye position scale bars in *A* and *B*). Figure adapted from Malevich et al. (59) and reproduced under Creative Commons license CC-BY.

could account for eye movement delays whenever a visual stimulus was presented ~80–100 ms before the expected saccade (23, 48, 52, 90). Nonetheless, as of today, there are virtually no studies approaching the question of saccadic inhibition from a neurophysiological perspective (but see Ref. 91 for an overview of the evidence so far). Moreover, the existing models are too specific to account for the large behavioral variety mentioned above. In this final part of our article, we provide new insights on the underlying physiological mechanisms of this incredibly powerful and pervasive behavioral phenomenon. This is important, because it calls for important modifications to current popular models of saccadic inhibition.

Visual input entering the brain from the retina immediately spreads in multiple and parallel visual pathways, with many cortical and subcortical areas receiving new visual spikes around the same time window. This massive and parallel visual input creates all the conditions for a competition between an ongoing process trying to do something, in our case move the eyes, and a new process asynchronous to the first one generated by the visual spikes entering the brain, and attempting to lead to a different behavioral outcome. Such a situation can, in fact, generate a conflict between spatial locations and the different types of motor action that might be taken. Although it is quite clear that the responses of visual neurons in oculomotor areas are heavily involved in exogenous capture (e.g., Refs. 69, 92), favoring reorienting behavior, an important unanswered question remains about which brain areas are mainly involved in the inhibitory part of the process, which might suddenly reset the active vision cycle.

In the next section, we discuss which are the main brain areas and mechanisms that are implicated in coordinating and stabilizing oculomotor behavior when multiple visual and motor signals compete to drive the response behavior.

## THE CLASSIC ROLE OF SUPERIOR COLLICULUS AND FRONTAL EYE FIELDS IN DRIVING ORIENTING BEHAVIORS

To date, neurophysiological studies of the impacts of exogenous stimuli on the oculomotor system have mainly focused on the SC and cortical areas such as the frontal eye fields (FEFs). Mirroring the classic view of “oculomotor capture” alluded to earlier, a great effort has gone into studying how these oculomotor areas drive orienting reflexes. Classic studies on the SC described neurons as being organized according to a retinotopic map and showing a rise in activity starting before the execution of an eye movement toward a particular location (93, 94). Similarly, FEF neurons were described to represent space and to be active before saccadic eye movements of a particular size and direction toward visually present or memorized targets (95). Moreover, in both the SC and FEF, the neurons firing for a specific motor vector often showed visual responses for the target onset, thus suggesting a viable functional route (visual to motor transformation) for exogenous stimuli to support the ideas of “capture” and “orienting reflexes.”

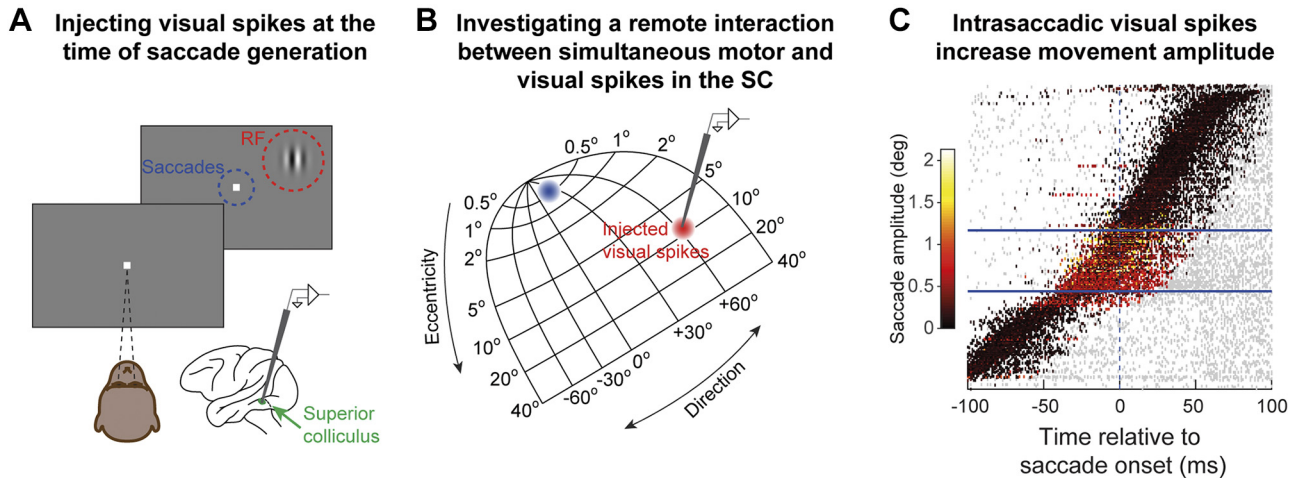
Following this basic principle sustaining oculomotor programming, one interesting question was whether additional visual stimuli other than the saccade target would modulate

premotor activity (e.g., a “distractor” effect like in early behavioral studies). In the SC, one main hypothesis was that the presentation of an exogenous stimulus during the preparation of an eye movement would cause an increase in activity at another location of the SC map, away from the current saccade goal (70). Consequently, this extra activity might alter the preparatory activity related to the planned saccade. In these experiments (70), the stimuli had a differential effect on the response outcome whether they were presented in close proximity of the target or away from it. In particular, pretarget activity of visuomotor neurons at the saccade goal was boosted by the presentation of nearby stimuli, similar to collicular microstimulation experiments that induced nearby excitation (96). On the other hand, pretarget activity was inhibited by the presentation of far ones via the lateral inhibition suggested to be present in the superficial and intermediate layers of the SC (97, 98). Similar observations were also made in the FEF. For example, when a visual distractor was presented simultaneously with a target, saccade trajectories could curve toward or away from it. Neuronally, FEF activity at distractor locations was higher when the trajectory veered toward the distractor and lower for trajectories veering away (68), supporting the hypothesis of spatial interactions between different locations.

Even though this framework can seem, at face value, ideal to explain the differential effects of exogenous stimuli on saccade behavior, similar to what was reported in the classic behavioral literature on distractors (e.g., Refs. 29, 99), it still presents with some strong limitations from the broader perspective of this article. First, we already know from behavioral studies that even a flash presented at the saccade target itself would cause saccadic inhibition (24, 49), which is decidedly different from the nearby distractor effects in the SC and FEF described above. No lateral inhibition exists in the SC and FEF for such small target-to-distractor spatial separations. In fact, even for microsaccades, a visual transient on the fixation spot (which is most frequently the saccade goal for microsaccades), causes strong microsaccadic inhibition (Fig. 3B) (84). This important discrepancy suggests that there is no prerequisite to have spatial competition for implementing an interruption signal. In fact, if anything, distractors in the SC can be strongly excitatory exactly at the time of saccade triggering, to the extent that they significantly misdirect the originally planned eye movements (Fig. 5).

Second, the assumption of a lateral inhibition mechanism locally within the SC network was found to be not as reliable as initially thought. Research using in vitro whole cell patch-clamp techniques failed to find clear evidence for nearby excitation or distant inhibition across the intermediate layers of the SC in rodents (100), which have similar motor maps to nonhuman primates (101), but see Kasai and Isa (98) for alternative views. Moreover, studies using in vivo work paired with pharmacological activation of the intermediate SC were able to demonstrate the facilitation of saccades associated with the affected site, but they failed to find evidence for distant inhibition (102). This might suggest that long-range lateral inhibition in the SC might be mediated by external inhibitory inputs from the basal ganglia (103).

Third and most important, both the SC and FEF are mainly excitatory circuits that trigger saccades rather than



**Figure 5.** Injecting peri-saccadic movement-unrelated spikes in the SC has an excitatory rather than inhibitory impact on triggered eye movements. **A:** in a study by Buonocore et al. (92), a monkey maintained fixation while an eccentric stimulus in the form of a vertical grating was presented in a recorded neuron's response field (RF; red). This allowed injecting movement-unrelated "visual" spikes into the SC map around the time of microsaccade generation (blue). **B:** the extra "visual" spikes were injected at more eccentric retinotopic locations (red) than the neurons that would normally exhibit motor bursts for microsaccades (blue) (analogous to so-called remote distractor effects). **C:** the generated microsaccades were enlarged when extra-foveal SC spiking (stimulus-driven visual bursts) happened right before and during the microsaccades (spikes are color-coded according to the observed movement amplitude on a given trial; the rest of the spikes are gray). The spike raster is sorted based on the time of the injected visual burst (peak firing rate after stimulus onset) relative to saccade onset (in the bottom left part of the raster, there are the trials with visual bursts earlier than microsaccades, and in the top right, the trials are with visual bursts later than microsaccades). Thus, SC activity induced by "distractors" can exert an excitatory, rather than inhibitory, effect on movement generation. Figure adapted from Buonocore et al. (92) and reproduced under Creative Commons license CC-BY. SC, superior colliculus.

inhibit them. For example, task-irrelevant sensory activity on the SC map at a location different from the saccade goal, but coincident with movement triggering time, can actually contribute to modifying individual saccade amplitude with a precision of impact on eye movement generation reaching the level of contributions by individual single spikes by single neurons at the time at which SC activity is to be read out by downstream neurons (92) (Fig. 5C). At the behavioral level, this effect can also be observed when multiple target alternatives are available. In this case, the orienting response depends on the spatial congruency between the sensory event and the internal goals (104–106). Although visual input congruent with the incoming oculomotor plan can in some cases speed up sensory responses leading to the generation of express (micro)saccades to the target (107), incongruent signals can interfere with eye movement triggering delaying the movement (e.g., Refs. 19, 28, 29, 48). These results support the main involvement of the SC in specifying the orienting process (e.g., the desired vector of the gaze shift) rather than the inhibition per se, which is also consistent with the behavioral effect of direction dependency of microsaccades following visual cues (see *Parallel discovery of saccadic inhibition in microsaccades*).

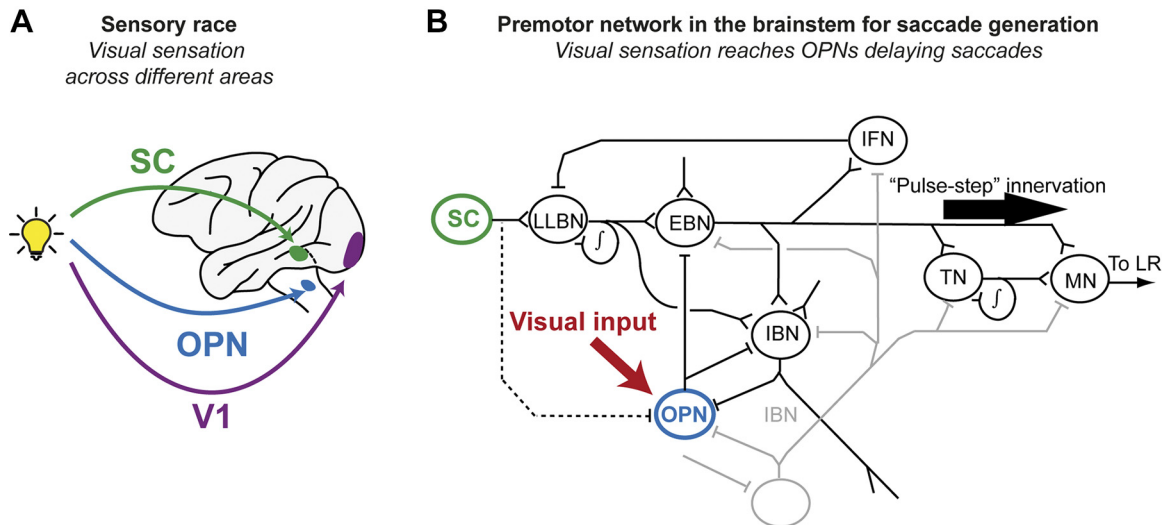
There might, thus, be a disconnect between current models of saccadic inhibition and neurophysiological evidence; the most accepted models to date are indirect and strongly rely on FEF/SC lateral inhibition explanations (23, 48, 52, 108). While local, within-area, inhibition might (partially) explain inhibition for large saccades produced by a distractor in the opposite hemifield or far away from the target (19, 20, 48), this mechanism is still incomplete to unify findings across all saccade ranges, especially for small eye movements. Indeed, all saccade amplitude ranges need to be

considered in models, because microsaccades inevitably happen even in trial-based remote-distractor paradigms. There is, therefore, a clear need to provide an alternative hypothesis to explain orienting behaviors in the presence of exogenous signals.

We suggest that such a perspective resides in acknowledging the need for an early (and thus rapid) sensory pathway into the oculomotor system that is complementary to that toward the SC, which is already well-known and accepted. Critically, such a sensory pathway must target late stages in motor control circuitry, downstream of the SC and with potentially shorter latencies than the sensory pathway to the SC. We believe that such a pathway exists and innervates inhibitory neurons in the brainstem, therefore playing a particularly critical role in preventing the system from becoming excessively reflexive due to the sensory inputs arriving in excitatory eye movement structures like the SC (Fig. 6A).

Our hypothesis is that sharing visual information-processing capabilities among different late motor control brain areas could be a critical prerequisite for avoiding undesirable motor outcomes. For example, neuropathology affecting lower brainstem nuclei (e.g., Ref. 112) can lead to compulsory orienting behavior. Hallmarks of such behavior can also be seen in both healthy humans (76) and nonhuman primates (e.g., Ref. 74) performing antisaccade tasks (72), in which eye movements show altered kinematic and directional errors. As stated above, this suggests a low-level (even brainstem) interaction between multiple planned and instructed motor programs (as well exogenous events). In fact, besides cue-induced interactions with microsaccades in the antisaccade paradigm, the subsequently generated antisaccades themselves are still susceptible to saccadic inhibition (18), which is not surprising. Cognitive control is expected to only plan





**Figure 6.** Omnipause neurons (OPNs) in the lower brainstem can regularize the race condition of Fig. 1, A and C, by being both sensitive to visual input and also potent enough to inhibit saccade generation. *A:* new visual information races from the retina, via parallel pathways, toward not only the primary visual cortex but also oculomotor control areas (e.g. SC and OPN). In our framework, the outcome of the race contributes to behavioral variability. We suggest that OPNs actively participate in such a sensory race by receiving visual input (either directly or via primary visual cortex and/or SC pathways) and by showing sensory tuning characteristics that are similar to those in the SC (109). In this way, two structures with similar visual feature tuning properties, but promoting different response behaviors (the SC promoting orienting and OPNs inhibiting saccades), can successfully achieve resetting of the active vision cycle (Fig. 1A) and coordinate behavior. *B:* classic models of saccade control invoke the role of brainstem premotor and motor nuclei in compiling the eye muscle commands. Historically, these nuclei have been described as completely motor in their function. OPNs (blue) are tonic neurons that inhibit the burst neurons (EBN) that ultimately move the eyes, with the SC (green) specifying the desired saccade vector. Whenever OPNs stop firing, a new eye movement is executed. In our new model, OPNs have early (and sensory-tuned) access to visual information that can induce visual bursts in these neurons. Such increased activation raises the threshold for triggering saccades, explaining both saccadic inhibition as well as its sensory-tuning properties. Other shown oculomotor structures in the network include: long-lead burst neurons (LLBNs), inhibitory burst neurons (IBNs), inhibitory feedback neurons (IFNs), tonic neurons (TNs), and motoneurons (MNs). SC, superior colliculus. Figure adapted with permission from Hafed (110) and Scudder (111).

the saccade; external stimuli interfering with this plan still require resetting of the active vision cycle (Fig. 1A).

Given the rapid time scales with which saccadic inhibition can emerge, we argue that it is critical that late motor control areas, only one or two synapses away from controlling the eye muscles, can additionally behave as if they are early visual areas, exhibiting early visual bursts that have latencies less than many higher-order cortical visual areas. By saying this, we suggest that such structures would be sensitive to the visual events happening in the outside world. In such a way, the oculomotor system could play a primary role in a lower level of decision-making process that requires the immediate resetting of the oculomotor plan to allow time to respond optimally to environmental changes.

### UNVEILING THE ROLE OF A NEW SENSORY-MOTOR REGION COMPETING IN THE RACE BETWEEN VISUAL SIGNALS: THE BRAINSTEM OMNIPAUSE NEURONS

As discussed above, it is well-known that the SC is strongly involved in commanding eye movements toward a visual stimulus (93, 94). More recently, it has been shown that the SC's sensory capabilities are far more complex than just simple responses to a dot stimulus as the saccade target (e.g., Refs. 109, 113–115). The SC exhibits visual sensitivity for color (116, 117), contrast (118), orientation (113), and spatial frequency (109), among others. In fact, the properties of SC visual responses, such as spike count and latency, are strongly correlated with average saccadic reaction times (10, 109, 119,

120). Therefore, the first SC visual response matters for orienting and motor timing.

This positions the SC as one of the best candidates to support orienting behaviors. However, to regularize a race condition that arises with asynchronous sensory inputs (Fig. 1, A and C), and to allow the system to (eventually) optimally orient to the stimuli, there is a need for a different structure with motor properties capable of inhibiting the orienting response triggered by an abrupt stimulus. Similar to the SC, it is critical that this structure is also responsive to sensory stimulation for all spatial locations. This last critical point is important because the properties of saccadic inhibition alluded to above demonstrate “inhibition” irrespective of location, but still with timing properties that depend on the sensory properties of the external stimulus. Finally, it would be advantageous that such a structure is very close to the motor output to rapidly act when there is a need for inhibition. We therefore propose that the perfect candidates to undertake these tasks are the omnipause neurons (OPNs) of the nucleus raphe interpositus (RIP) (121).

In the classic view of the brainstem, still accepted today, oculomotor nuclei have been described as being purely motor (122–124) (Fig. 6B). Among these nuclei, the OPNs are a small group of neurons that fire tonically during fixation and pause their activity abruptly just before and during saccades in all directions (122, 123, 125, for OPNs in rodents see Ref. 126). From microstimulation experiments in rhesus monkeys, it is well documented that sudden reactivation of OPNs can interrupt saccades in mid-flight (127). This confirms the ability of OPNs to rapidly inhibit a motor response,

fulfilling the first requirement for the sensory race hypothesis alluded to above. OPNs therefore fulfill a critical criterion for saccadic inhibition: access to the very final motor stage of triggering saccades. Note, however, that interrupting individual saccades in mid-flight (128), possibly through collicular input (e.g., Ref. 129), occurs on a different time scale from the one that we study here, in which asynchronous inputs interact with yet-to-be-executed motor plans. In this context, OPNs, provided they have pattern analysis sensory capabilities, might be much more than a simple gating mechanism for saccades, but rather a key player in finely coordinating saccadic output. This idea has also been suggested before in other contexts (130).

Even though OPNs were overwhelmingly described to be purely motor, neurophysiological research in cats showed that visual stimulation can lead to both brief interruptions of eye movements (131) as well as enhancements of OPN activity during fixation. Little evidence of similar visual responses has also been incidentally and sparsely reported in monkeys, with the observation that OPNs increased their firing rate by a few spikes after visual target presentation in eye movement tasks (10, 132–134). Interestingly, there is extensive evidence that retinal ganglion Y (alpha) cells project to several mid-brain structures involved in early visual processing and eye movement control, such as the SC and the pretectum. Some of these connections also terminate in the pons, within the dorsal raphe nucleus (e.g., Ref. 135), and it has been suggested that they can facilitate orienting and escape responses. Sensory signals can also rapidly reach OPNs via sensory areas (like the primary visual cortex) that have short visual response latencies.

Despite the above, at present, OPNs play absolutely no role in models of oculomotor control other than gating saccades. In several psychophysical and modeling studies, Buonocore et al. (19, 24, 49, 136) and Hafed and Ignashchenkova (15) leveraged the hypothesis of a quick sensory signal reaching the OPNs and demonstrated that, besides inducing saccadic inhibition, it was also possible to reliably truncate goal-directed saccades in mid-flight by presenting a stimulus within 40–50 ms before saccade onset (24, 136) (see also Ref. 20). These observations suggest that OPNs might have much richer sensory responses than previously thought, fulfilling also the second requirement for saccadic inhibition.

We then suggest that the sensory response to visual stimuli in OPNs might be much stronger and richer than previously believed. We hypothesize that a sudden increase in OPN activity following the presentation of an exogenous stimulus might be causally involved in mediating saccadic inhibition. Since saccadic inhibition is clearly modulated by the stimulus characteristics, similar to how the microsaccadic rate signature after stimulus onset shows dependence of saccadic inhibition on spatial frequency and contrast (50, 137), we suggest that OPNs express a wide range of responses that are tuned to stimulus features, much like it is now known that SC neurons also show evidence of feature tuning (109, 113). Moreover, OPNs might be visually “sensitive” for a variety of stimuli presented at different locations in the visual field, reducing the need to invoke spatial interactions in oculomotor areas such as the SC and FEF for explaining saccadic inhibition. Furthermore, we argue that endowing

OPNs with sensory capabilities, as initially observed by Evinger et al. (131), might help in maintaining flexibility over the response until the very last stages of sensory-to-motor transformation. All of these properties of OPNs are already being demonstrated now with ongoing experiments in our studies (138, 139).

## CONCLUSIONS

One of the main goals of an organism is to successfully interact with the dynamic environment in which it is immersed. To avoid continuously being captured by external events, our sensory and motor systems have to engage in a fine balancing act between being ultra-reflexive or too slow. In our opinion, inhibition must inevitably happen before orienting, at least within the oculomotor system. Depending on when an exogenous stimulus reaches our senses with respect to our own internal processing cycle, it will dictate the efficacy with which resetting of the active vision cycle takes place (Fig. 1A), and in turn the efficacy of the final orienting response.

Our hypothesis is supported by a large core of behavioral studies showing that sensory events inhibit eye movements as early as 50 ms after they occur. We propose that the primary neural circuit supporting such inhibition is located in the lower brainstem, where OPNs reside. By endowing the very final stages of oculomotor control circuits with properties of early sensory areas, it is possible to successfully balance a “sensory race” that is initiated by external events via parallel pathways from the retina and that leads different afferent areas to start spiking at different times relative to the exact same input. Whenever an excitatory circuit like the SC wins the race (i.e., receives visual signals) before an inhibitory circuit like OPNs, then an express response occurs (but it can also be a distorted response, since the external stimulus is often requiring a different behavioral outcome than the current motor plan; Fig. 5). Otherwise, resetting of the active vision cycle happens more successfully. In this regard, our prediction is that OPNs should “win” the race most often, and this is manifested in the highly robust behavioral phenomenon of saccadic inhibition.

Our framework not only explains the universal behavioral phenomenon of saccadic inhibition, but it also provides a well-grounded neural basis for explaining some of the behavioral variability observed in the timing of the output in response to exogenous stimulation. We suggest that this inhibition mechanism can act both in purely reflexive tasks as well as in more cognitively controlled ones. In our ongoing studies, we aim to investigate and detail the neurophysiological basis underpinning this early and automatic inhibition of the oculomotor system in the lower brainstem.

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## DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

## AUTHOR CONTRIBUTIONS

A.B. and Z.M.H. conceived and designed research; prepared figures; drafted manuscript; edited and revised manuscript; and approved final version of manuscript.

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