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NEUROSYSTEMS

Linking visual response properties in the superior colliculus to saccade behavior

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Abstract

Here we examined the influence of the visual response in the superior colliculus (SC) (an oculomotor control structure integrating sensory, motor and cognitive signals) on the development of the motor command that drives saccadic eye movements in monkeys. We varied stimulus luminance to alter the timing and magnitude of visual responses in the SC and examined how these changes correlated with resulting saccade behavior. Increasing target luminance resulted in multiple modulations of the visual response, including increased magnitude and decreased response onset latency. These signal modulations correlated strongly with changes in saccade latency and metrics, indicating that these signal properties carry through to the neural computations that determine when, where and how fast the eyes will move. Thus, components of the earliest part of the visual response in the SC provide important building blocks for the neural basis of the sensory–motor transformation, highlighting a critical link between the properties of the visual response and saccade behavior.

Introduction

Crucial to survival is the ability to optimally extract ongoing information about the environment through sensory channels in order to guide the appropriate behavioral responses. One of the simplest of such sensory–motor transformations to investigate is the visual guidance of saccadic eye movements (Wurtz & Goldberg, 1989). The superior colliculus (SC), a layered structure in the midbrain, is a critical sensorimotor integration node for saccade control, located at the interface between sensory input and motor output (Hall & Moschovakis, 2003). The SC receives visual input directly from the retina as well as indirectly from visual cortex (Fries, 1984; Cusick, 1988; Robinson & McClurkin, 1989; Lock *et al.*, 2003). Following the appearance of a visual stimulus, neurons in both the superficial and the intermediate SC discharge a phasic burst of action potentials (defined as a visual response) that is time-locked to stimulus appearance (Wurtz & Goldberg, 1971; Sparks, 1975; Mohler & Wurtz, 1976). Visuomotor neurons in the intermediate SC also discharge a second burst (a saccadic motor response) to drive the saccade (Mohler & Wurtz, 1976; Sparks, 1978) and these visuomotor neurons project directly to the saccade premotor circuit in the brainstem reticular formation (Rodgers *et al.*, 2006). Despite detailed understanding of the circuit, we do not yet know how the visual response in the SC is transformed into the motor command to guide behavior.

Many previous studies of this sensory to motor transformation link the neural activity in the SC to saccade behavior via examinations of saccadic motor-related activity (Sparks, 1978; Munoz & Wurtz, 1995; Hanes & Schall, 1996) or the preparatory build-up of this motor-related activity (Basso & Wurtz, 1998; Dorris & Munoz, 1998). Here we show that low-level sensory signals impact visual processing and are strongly correlated to multiple saccadic behaviors. This result is striking because these visual signals occur before and are distinctly separate from pre-motor activity and the motor response.

The quality and properties of incoming sensory signals affect the neural computations underlying the visuomotor transformation (Sparks, 1986). For short-latency express saccades (Fischer & Boch, 1983; Fischer & Weber, 1993), it appears that there is a single burst from visuomotor neurons in the SC that triggers the saccade (Edelman & Keller, 1996; Dorris *et al.*, 1997; Sparks *et al.*, 2000), which is suggestive of a direct visuomotor transformation with minimal processing. More commonly, however, during longer regular latency saccades, the visual and motor bursts in the SC are distinctly separate responses (Mohler & Wurtz, 1976). A question that arises is how do the properties of a visual stimulus change the visual response in the SC and how do these changes subsequently influence saccade behavior, if at all? Previous studies have shown that the timing and magnitude of the visual responses in the SC are modulated by contrast (Li & Basso, 2008). Furthermore, changes to the onset latencies (Bell *et al.*, 2006;

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White *et al.*, 2009) and firing rate (Dorris *et al.*, 2002; Fecteau *et al.*, 2004) of the SC visual response have also been shown to correlate with saccadic reaction time (SRT).

We hypothesize that the early visual response (i.e. the initial phasic burst of action potentials) in the SC plays a key role in the development of the motor command that will drive regular latency saccades. To test this hypothesis we manipulate target luminance as a means of modulating the timing and magnitude of the visual response in the SC and identify how changes to these signal properties link to saccade behavior. We show that despite the fact that visual and motor responses are temporally separate during regular saccades, modulations to the earliest part of the visual response carry through the visuomotor transformation to influence saccade latency and metrics.

Materials and methods

Animal preparation

All animal care and experimental procedures were in accordance with the Canadian Council on Animal Care policies on use of laboratory animals and were approved by Queen's University Animal Care Committee. Two male monkeys (*Macaca mulatta*, W: age 6 years, 8 kg; Q: 7 years, 12 kg) were used in these studies. A detailed description of the surgical techniques used to prepare animals for neuronal recording from the SC and eye movement recordings in our laboratory has been described previously (Marino *et al.*, 2008). Briefly, both animals underwent surgery under aseptic conditions for the insertion of eye coils, a stainless steel head holder, and a recording chamber that was mounted on the skull using stainless steel bone screws and dental acrylic. The recording chamber was oriented towards the SC at an angle of 38° posterior from vertical in the mid-sagittal plane. Monkeys were given at least 4 weeks to recover prior to onset of behavioral training.

Experimental tasks and behavioral stimuli

Behavioral paradigms and visual displays were under the control of two Dell 8100 computers running UNIX-based real-time data control and presentation systems (Rex 6.1) (Hays *et al.*, 1982). Monkeys were seated in a primate chair with their heads restrained for the duration of an experiment (2–4 h). They faced a display cathode ray tube monitor that provided an unobstructed view of the central visual area 60° (horizontal) × 50° (vertical). Monkeys were required to perform several visually guided saccade tasks (Fig. 1A and B). Experiments were performed in darkness with individual trials lasting ∼1–2 s depending on the variability of fixation duration and SRT. Each trial required the monkey to generate a single saccade from the central fixation point (FP) to a peripheral visual target (T). At the start of each trial, the screen turned black and after a period of 250 ms a circular grayscale FP of constant luminance (0.25° diameter spot, 3.5 cd/m^2) appeared at the center of the screen against a black background (∼0.0001 cd/m^2). Fixation of the FP was required for a variable period (500–800 ms) until either a small circular 0.25° grayscale T appeared (delay task) or the FP was extinguished (gap task). During the inter-trial interval (800–1500 ms), the display screen was diffusely illuminated to prevent dark adaptation.

The delay task (Fig. 1A) was used to dissociate visual- and saccade-related activity. In this task, the monkeys were required to continue fixation of the FP for an additional 500–800 ms after T appearance.

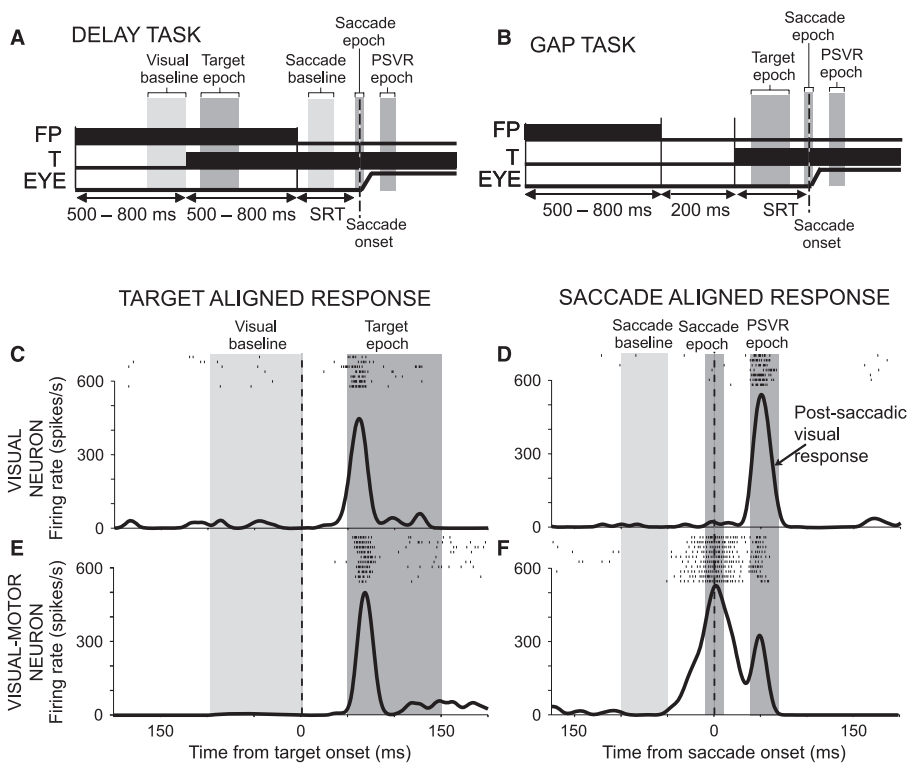

FIG. 1. (A, B) Schematic representation of temporal events in the delay (A) and gap (B) tasks for the fixation point (FP), target (T), eye position (EYE) and various analysis epochs (see text for details). Saccadic reaction time (SRT) is calculated relative to the *disappearance* of the FP in the delay task and the *appearance* of the T in the gap task. Vertical gray bars denote key analysis epochs used to classify responses and neurons. (C–F) Rasters and spike density functions of a representative V (C, D) and VM (E, F) neuron for target-aligned (C, E) and saccade-aligned (D, F) responses to the optimal target location in the delay task. A post-saccadic visual response (PSVR) can be clearly seen in both the V and VM examples.

Only after FP disappearance was the monkey allowed to initiate a saccade to the T. In the delay task, the target was presented at one location only: at the center of each neuron's visual response field. Supporting Information, Fig. S1 provides additional details regarding the characterization of visual response fields.

The gap task (Fig. 1B) served to reduce the inhibition due to active visual fixation and thereby making the oculomotor system more responsive to visual inputs (Dorris & Munoz, 1995; Paré & Munoz, 1996; Dorris *et al.*, 1997; Machado & Rafal, 2000; Krauzlis, 2003). In this task, a 200-ms period of darkness (gap) was inserted between FP disappearance and T appearance. The monkey was required to initiate a saccade to the T after its appearance. In the gap task, targets were presented with equal probability at one of two possible locations: the center of each neuron's visual response field and at a location opposite the horizontal and vertical meridians. The gap and delay tasks were presented as separate blocks of trials. Fewer trials were recorded in the delay task (typically 6–8) relative to the gap task (up to 25 trials) as the delay task was only utilized for characterizing visual and motor activity.

Targets presented into the center or opposite location (i.e. opposite horizontal and vertical meridians) of each neuron's response field ranged in eccentricity from 1.5 to 30° depending on the location of each neuron within the SC map. Target luminance was manipulated systematically using seven randomly interleaved luminance levels (0.001, 0.005, 0.044, 0.4, 3.5, 17.5 and 42.5 cd/m^2). Luminance was measured with an optometer (UDT instruments, model S471, San Diego, CA, USA) that was positioned directly against the monitor screen and centered on the stimulus (T or FP). Each correct trial was rewarded with a drop of water. A computer-controlled window ensured eye position remained within 2.5° of the FP and 5° of the T during correct trials in all tasks.

Recording techniques

Extracellular recording was performed with tungsten microelectrodes (0.5–5 MΩ impedance; Frederick Haer) inserted through guide tubes (23 gauge) that were anchored in delrin grids with 1-mm hole separations inside the recording chamber (Crist *et al.*, 1988). Electrodes were advanced with a Narishige microdrive (MO95) to the dorsal surface of the SC, distinguished by large increases in background activity following each saccade or change in visual stimuli. The electrode was then slowly lowered into the SC to record from individual visually responsive neurons.

Data collection

Neural waveforms corresponding to spikes (40-kHz sampling) and horizontal and vertical eye position (1-kHz sampling, magnetic search coil technique; Robinson, 1963) were recorded in real time with Plexon data acquisition hardware (Plexon Inc.). Accurate isolation and sorting of individual neurons was performed offline (Offline Sorter 2.5; Plexon Inc.).

Neuron classification

To characterize the activity of individual neurons across stimulus conditions, trains of action potentials averaged across identical correct trials were convolved into spike density functions using a Gaussian kernel (σ = 5 ms) for each spike (Richmond *et al.*, 1987). Spike density functions were aligned on target appearance when analysing visual responses (Fig. 1C and E) and saccade onset when analysing motor responses (Fig. 1D and F). Neurons were classified as visual-only (V, Fig. 1C and D) or visuomotor (VM, Fig. 1E and F) based on the presence or absence of saccade activity during the delay task (Fig. 1A, D and F) using the brightest target luminance. Visual and saccade activity was defined relative to a baseline. Visual baseline activity (Fig. 1A) was calculated as the average discharge from all correct trials during the 100 ms prior to T appearance. Saccade baseline activity was calculated from the average discharge on all correct trials from 100 to 50 ms prior to the onset of the saccade in the delay task (Fig. 1A). A significant visual response was defined as an increase in target-aligned activity greater than 50 spikes/s above the visual baseline during the target epoch (50–150 ms following target presentation). A significant motor response was defined as an increase in saccade aligned activity greater than 50 spikes/s above both the target baseline and the saccade baseline during the saccade epoch (± 10 ms from saccade). V neurons we describe were located within 1000 μm of the dorsal SC surface as measured from the microdrive. All VM neurons recorded were located below V neurons (McPeek & Keller, 2002) and were recorded within 2500 μm of the dorsal SC surface. A subset of the V and VM cells discharged a short burst of action potentials 50–80 ms *after* saccade onset that was distinctly separate from the motor burst (see Table 1). This subset of V and VM neurons were classified as containing a post-saccadic visual response (Fig. 1D and F; Li & Basso, 2008). A significant post-saccadic visual response was classified based on a distinct peak of activity aligned on saccade onset that was greater than 50 spikes/s above the visual baseline activity during the post-saccadic visual response epoch (50–100 ms following saccade onset).

Behavioral analyses

Data were analysed offline with custom MATLAB (MATLAB 7.4; Mathworks Inc.) software. The start and end of saccades were determined from velocity and acceleration template matching criteria, verified offline by the experimenter and corrected when necessary. Because visual response onset latency (VROL, see below) changed with target luminance, anticipatory saccades (saccades with SRTs less than the luminance-specific afferent visual delays; see below) were removed based on the calculated VROL. Anticipatory and express saccades were removed from analysis to ensure that visual and motor neural responses were temporally separated (i.e. target and saccade epochs Fig. 1A and B). This ensured that visual responses were isolated from and uncontaminated by motor activity. These were defined as all saccades with SRTs less than 50 ms after the mean luminance-specific VROL calculated in the delay task. This ensured a minimum of 50-ms temporal separation between the onset of the visual response and the onset of the saccade movement.

Error rates were calculated from the trials in which the target was presented. Early aborted trials in which the monkey failed to fixate or

TABLE 1. Neuron breakdown by monkey, task and subtype

Cell type	Monkey W	Monkey Q
Delay task		
V	26/32	30/33
VM	33/41	17/23
Gap task		
V	16/18	24/28
VM	30/42	17/22

Number of neurons containing post-saccadic visual response/total recorded neurons.

maintain fixation of the FP at the beginning of the trial prior to target appearance were eliminated from further analysis because they were not representative of active task participation. Error rates were computed from those trials that were initiated by the monkey (by actively fixating the FP at the start of a trial and then holding fixation until the target appeared). Errors included: (1) anticipation errors (saccades made after the target appearance, but before the visual response reached the SC; see Results); (2) saccade errors (saccades initiated after the target was perceived that landed outside the target window and were therefore incorrect); (3) delay task timing errors (saccades initiated to the target after target appearance but before FP disappearance); and (4) trials in which no saccade was made. The percentage error rate was compared and analysed using a z test for proportions.

Saccade endpoint accuracy was defined as the Euclidean distance in degrees between the saccade endpoint (mean eye position during the first 10 ms of fixation following the saccade) and the target location. If a small corrective saccade occurred (less than 1% of trials), the endpoint of the initial saccade, landing within the target window, was used.

Neuronal analyses

The effects of luminance on visual response properties was determined from changes in the initial phasic burst of the visual response including: VROL, the peak magnitude of the target-related discharge, the time from the VROL to the peak of the target-related discharge (representing the growth time in which the target-related discharge increased towards its peak) and the decay rate (slope) of the initial visual response. Changes to the timing and peak magnitude of the visual response are important for saccadic visuomotor transformations because these changes have been found previously to correlate with saccadic reaction time (VROL: Bell *et al.*, 2006; Peak magnitude: Dorris *et al.*, 2002; Fecteau *et al.*, 2004; Bell *et al.*, 2004). The time required to reach the peak discharge (relative to the VROL) is also important because it reflects the rate in which neural activity is increasing or accumulating within the saccade system. Finally, changes in the rate of decay or shutdown of the visual response relative to the peak are important as they give a more comprehensive assessment of the response waveform. Likewise, the effects of target luminance on the motor response were determined from changes in the peak of the motor response and the ascending slope (slope was calculated in lieu of the time to the motor peak as the precise onset of motor-related activity could not be dissociated accurately from motor preparation and/or sustained visual activity).

VROL was determined from a running non-parametric Rank Sum test of a trial-by-trial spike density function aligned to the appearance of the visual target (Poisson-like exponential growth/decay function resembling a postsynaptic potential):

$$R(t) = [1 - \exp(-t/\tau_g)]\cdot[\exp(-t/\tau_d)]$$

where $R(t)$ defines the rate as a function time, growth constant (τ_g) = 1 ms; and decay constant (τ_d) = 20 ms (Thompson *et al.*, 1996). The Poisson-like exponential growth/decay function is critical for the calculation of signal onset latencies because the kernel only temporally smoothes neural activity later in time in order to preserve accurate onset times (Thompson *et al.*, 1996). VROL was determined by the onset of stable statistical significance ($P < 0.05$) between the mean activity during the visual baseline and a moving temporal window (1-ms resolution) within the target epoch (Fig. 1A). The peak visual response was calculated independently in both the delay and the gap tasks. The magnitude and timing of the peak visual response was calculated at each target luminance from the maximum of the trial-averaged spike density function (Gaussian kernel) aligned to the appearance of the target in the target epoch (Fig. 1A). We chose a 5-ms pulse width as this value allowed for a smooth continuous function to be generated with minimal temporal smoothing of neural activity. The time to the peak was calculated as the time from T appearance to the peak response. The decay rate of the visual response was calculated from the slope of a linear regression fitted to the target aligned trial-averaged spike density function (Gaussian kernel; σ = 5 ms) from the time of the visual peak until 25 ms after the peak.

It is possible that at the lowest luminance levels there may be some increased variability of onset times when the sensory stimuli was weak. Such increased variability could potentially influence the averaged visual response properties (VROL, growth time to the visual peak, peak magnitude of the visual response and the decay rate of the initial visual response). However, this potential variability cannot be reliably quantified, because VROL cannot be reliably detected on a trial-by-trial basis at these luminance levels due to the poor signal-to-noise ratios.

The effects of target luminance on the motor response were similarly characterized by the changes in the peak and ascending slope of the saccade-related discharge (aligned to saccade onset). The peak magnitude of the saccade burst was calculated at each target luminance from the trial-averaged spike density function (Gaussian kernel; σ = 5 ms) aligned to saccade onset in the saccade epoch (Fig. 1A and B). The ascending slope of the motor-related burst was calculated from a linear regression over the saccade-aligned trial-averaged spike density function (Gaussian kernel). This slope was calculated over the last 25 ms prior to the onset of the saccade to isolate saccade-related activity. All calculations made on the neural response from each neuron were verified visually during offline analysis. All statistical comparisons were calculated with repeated-measures ANOVA with post-hoc Bonferroni-corrected pairwise comparisons unless otherwise stated.

Correlating visual response properties to saccade behavior independent of luminance condition

To show that visual response properties correlate to sacccade behavior independent of luminance task condition, we collapsed all trials together (irrespective of luminance condition). After collapsing across conditions, trials were then sorted into 10 bins according to each behavioral measurement (SRT, peak velocity, accuracy). Correlations were then performed between the averaged behavior from each bin and the averaged neural responses calculated from the corresponding trials using the method described by Hanes & Schall (1996).

Each bin could contain trials from several different luminance-level task conditions. For this analysis, growth time was calculated as the rate of rise (slope) of the growth of the visual response from the peak response until 25 ms before the peak response. This compensated for some of the trial-by-trial VROL differences that resulted when different luminance levels were averaged together in the same bin. This increased VROL variability in this analysis likewise reduced the overall correlation observed between VROL and saccade behavior relative to the previous analysis when trials were only averaged within individual task conditions (see Fig. 5).

Results

Target luminance modulated saccade behavior

We recorded saccade behavior while monitoring activity from single SC neurons in the delay and gap tasks (Fig. 1). All express and short-latency saccades were removed to ensure all visual and motor responses were temporally separate (see Methods and Table S1 for details). Consistent with our previous study (Marino & Munoz, 2009), target luminance modulated SRT, peak velocity, endpoint error and overall error rate across both the gap and delay tasks across a range of target eccentricities (3.6–27.9°) defined by each neuron's response field optimal (Fig. S1).

We performed a two-factor repeated-measures ANOVA (two tasks, seven luminance levels) to quantify the luminance-modulated effects on SRT, peak velocity and endpoint accuracy in the gap and delay tasks. Only sessions in which both the gap and the delay task were recorded were included in the ANOVA. In the omnibus ANOVA for SRT, there was a main effect of task ($F_{1,61} = 566$, $P < 0.01$), with the gap task evoking faster SRTs then the delay task (gap SRT = 202 ms, delay SRT = 293 ms). There was also a main effect of target luminance ($F_{6,366} = 139.7$, $P < 0.01$) such that increasing target luminance from 0.001 to 0.044 cd/m^2 decreased mean SRT (corrected pairwise comparisons, $P < 0.01$). There was also an interaction between task- and luminance-driven modulations in SRT ($F_{6,366} = 20.5$, $P < 0.01$) such that increased target luminance decreased SRT more strongly in the gap task (Fig. 2A).

In the omnibus ANOVA for peak saccade velocity, there was no main effect of task ($F_{1,61} = 0.24$, $P = 0.63$), but there was a main effect of luminance ($F_{6,366} = 66.2$, $P < 0.01$) such that peak velocity increased in both the gap and delay tasks from target luminance ranges from 0.001 to 0.044 cd/m^2 (corrected pairwise comparisons, $P < 0.01$). There was also an interaction between task and target luminance modulations in saccade velocity ($F_{6,366} = 6.33$, $P < 0.01$) (Fig. 2B).

In the omnibus ANOVA for the effects of target luminance on saccade endpoint error from target, there was no main effect of task ($F_{1,61} = 1.76$, $P = 0.19$), but there was a main effect of luminance ($F_{6,366} = 66.62$, $P < 0.01$) such that accuracy significantly improved as target luminance increased from 0.001 to 0.044 cd/m^2 in both tasks (corrected pairwise comparisons, $P < 0.01$) (Fig. 2C). There was also an interaction between task and target luminance modulations

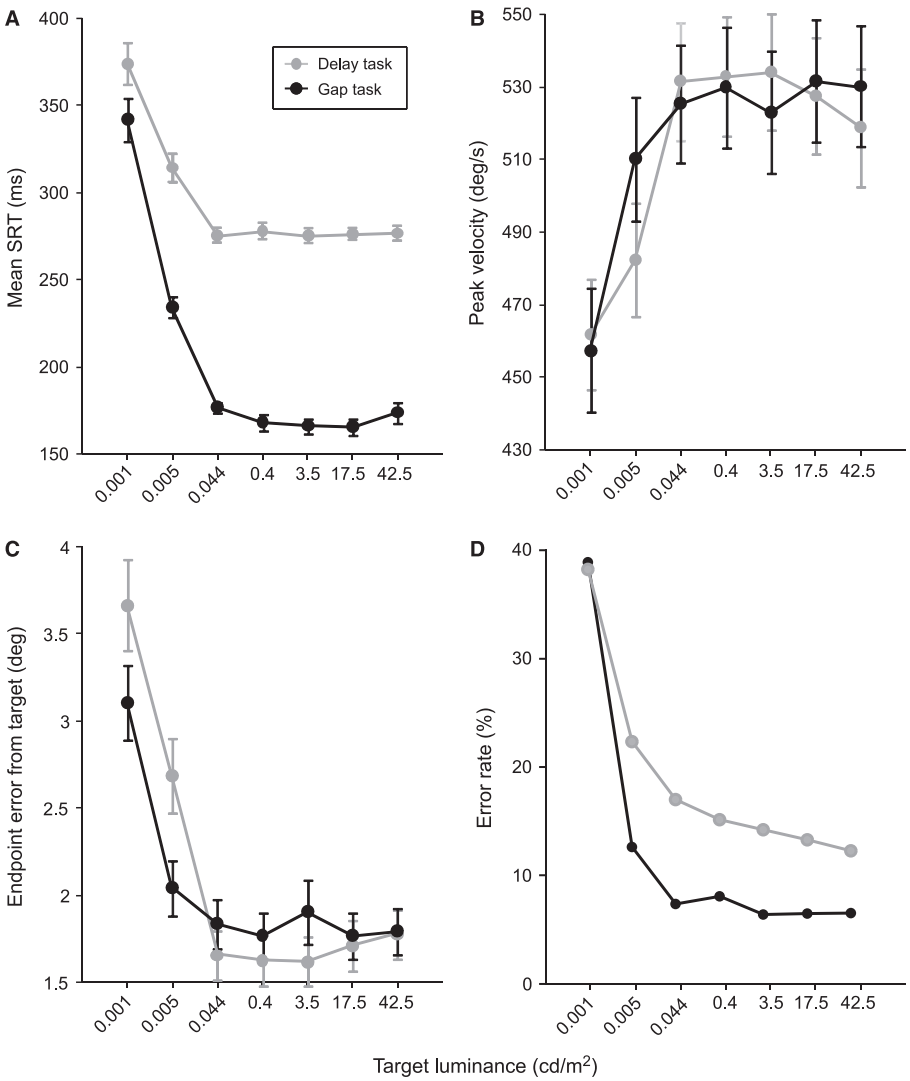

FIG. 2. Effect of target luminance on mean SRT (A), mean peak velocity (B), mean endpoint error (Euclidian distance of saccade endpoint from target in degrees) (C), and percentage error rate (D) (± SE) for the gap (black) and delay tasks (gray).

($F_{6,366}$ = 9.64, P < 0.01) such that saccades to very low luminance targets (below 0.044 cd/m^2) were more accurate in the gap task, whereas saccades to slightly higher luminance targets (between 0.044 and 42.5 cd/m^2) tended to be more accurate in the delay task (Fig. 2C).

Finally, the percentage error rate decreased significantly with increasing target luminance from 0.001 to 0.044 cd/m^2 (z test for proportions, P < 0.01) across both the gap and the delay tasks (Fig. 2D). This error rate was significantly below chance levels across all target luminance conditions, indicating that all targets were above visual detection thresholds. The error rate was lower in the gap task at all target luminance levels above 0.001 cd/m^2 (z test for proportions, P < 0.01).

Target luminance modulated SC visual activity

We recorded from 65 V and 64 VM neurons in the delay task. Of this group, 46 V and 64 VM single neurons were also recorded in the gap task (Table 1). Variations in target luminance led to systematic modulations in the visual responses of V and VM neurons. Figure 3 illustrates the population activity from all V and VM neurons recorded in the delay and gap tasks aligned on target appearance (left column) and saccade onset (right column). Increasing target luminance decreased the timing (VROL and time of peak), while increasing the magnitude of the visual response. Furthermore, increasing luminance also increased the steepness of the rise and fall of the initial phasic component of the visual response.

A different visual response was observed in some V and VM neurons that occurred ~40–60 ms *after* the onset of the saccade to the visual target (Table 1, Fig. 3, right column) that has been described previously (Li & Basso, 2008). Like the initial visual response following the appearance of the target, the timing and peak magnitude of this post-saccadic visual response scaled with target luminance. This post-saccadic visual response was only observed when the visual target was present within a neuron's response field at the time of saccade initiation.

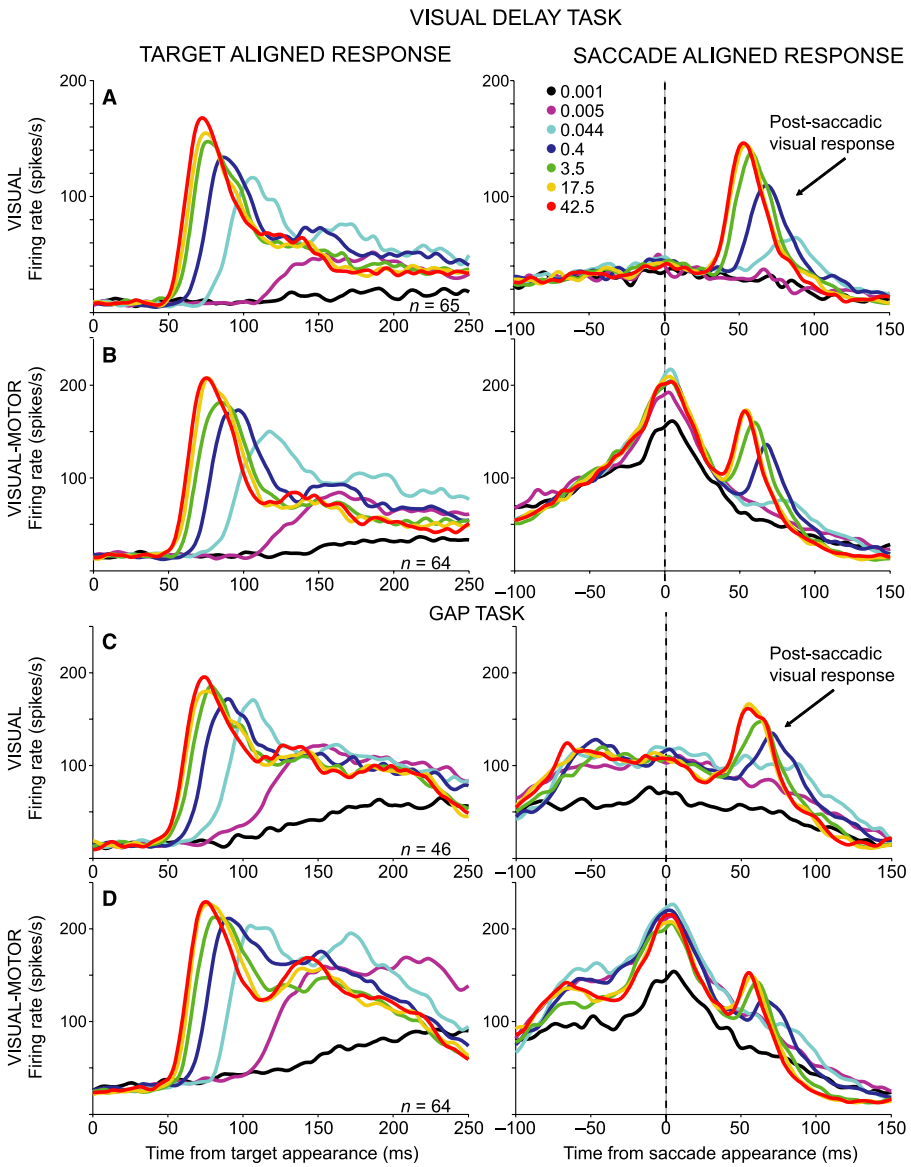

FIG. 3. Population spike density functions (Gaussian kernel σ = 5 ms) aligned on target appearance (left column) and saccade onset (right column) for all V (A, C) and VM (B, D) neurons recorded in the delay (A, B) and gap tasks (C, D) with seven randomly interleaved target luminance levels. Line colours denote target luminance (black: 0.001 cd/m^2, pink: 0.005 cd/m^2, cyan: 0.044 cd/m^2, navy blue: 0.4 cd/m^2, green: 3.5 cd/m^2, yellow: 17.5 cd/m^2, red: 42.5 cd/m^2).

Figure 4 illustrates how different components of the phasic visual response changed across the luminance manipulation. Visual response properties of the V and VM neurons were similarly modulated by luminance and no significant differences between these populations were found (see Fig. S2). Therefore, we collapsed across V and VM populations. At the dimmest target luminance, VROL was only measurable from 61% of the neurons recorded in the gap task and 40% of the neurons recorded in the delay task. As a result, repeated-measures ANOVAs for VROL and growth rate were not performed on data from the dimmest luminance unless otherwise stated. Main effects with target luminance were found in both tasks for all the visual response signal properties examined (see Table 2). The VROL and peak time decreased with increasing target luminance in the gap and delay tasks (pairwise comparisons, $P < 0.01$; Fig. 4A). The growth time of the visual response was also faster as luminance increased between 0.005 and 42.5 cd/m^2 (pairwise comparisons, $P < 0.01$; Fig. 4B).

There were also significant changes in the peak magnitude of the visual response (Fig. 4C). As target luminance increased, the peak

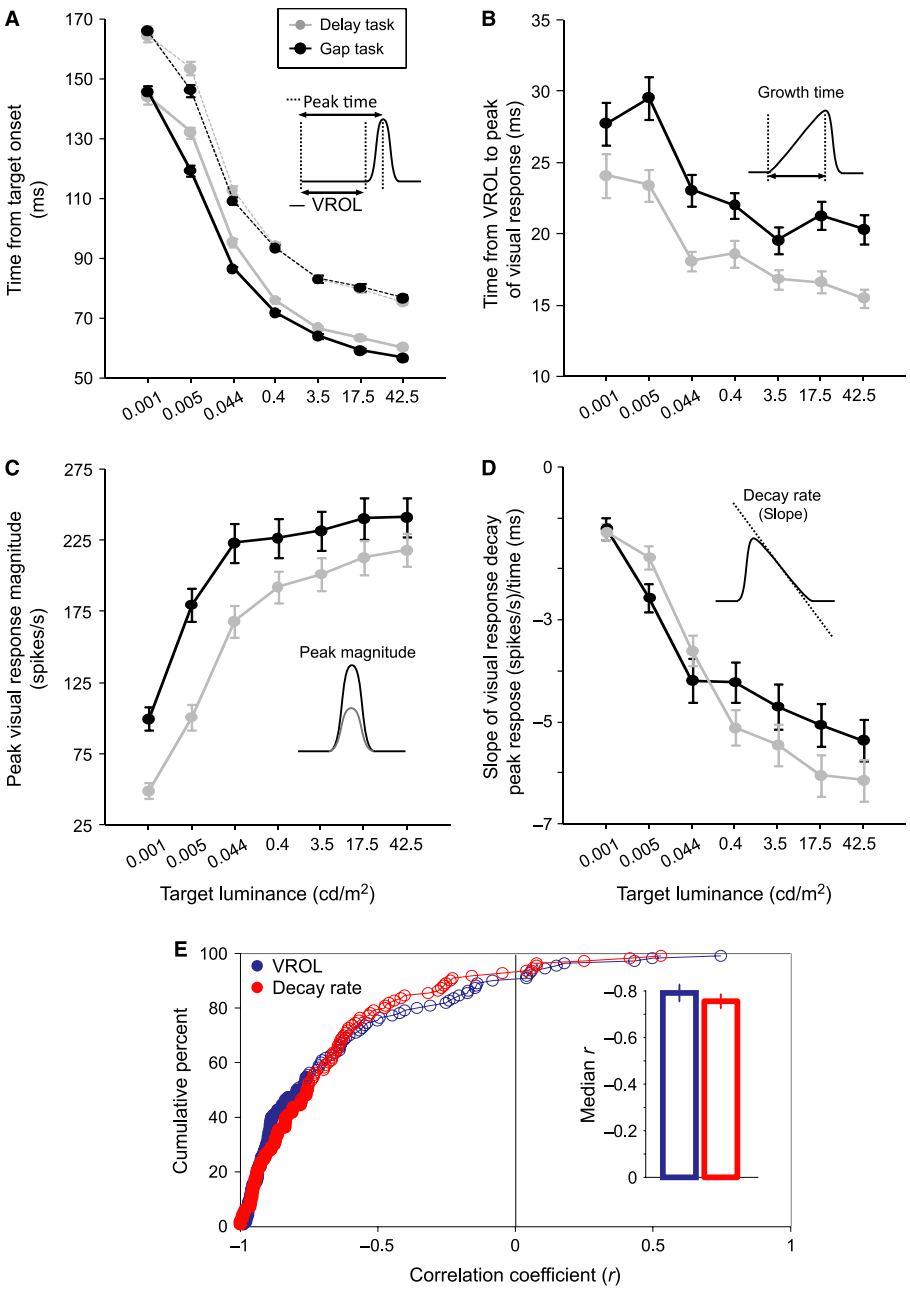

FIG. 4. Effects of target luminance on the signal properties of the visual sensory response in the delay (gray points and lines) and gap (black points and lines) tasks collapsed across all V and VM neurons. See Fig. S2 for separation of V and VM neurons. (A) Mean VROL (solid line) and peak time (dotted line) of the visual sensory response. (B) Mean growth time (time from the VROL to the peak) of the visual response. (C) Mean peak magnitude of the visual response. (D) Slope of the decay rate of the initial visual response burst. (E) Cumulative distributions of correlation coefficients and median *r* values (inset) for correlations between the peak magnitude of the visual response and both the VROL and decay rate in the gap task. Filled circles in the cumulative plots denote statistically significant correlations (within neuron). Color denotes the neural signal property that was correlated: VROL (blue), decay rate (red). Median *r* values denote how much variance was accounted for by each correlation.

TABLE 2. Statistical main effects of target luminance on visual response properties in SC

Visual response signal property	F statistic
Delay task	
VROL	$F_{5,640} = 1076$*
Peak time	$F_{6,768} = 989$*
Growth rate	$F_{5,640} = 21.1$*
Peak magnitude	$F_{6,768} = 144.3$*
Decay rate	$F_{6,768} = 78.2$*
Gap task	
VROL	$F_{5,545} = 930$*
Peak time	$F_{6,654} = 1095$*
Growth rate	$F_{5,545} = 18.2$*
Peak magnitude	$F_{6,654} = 102.9$*
Decay rate	$F_{6,654} = 37.5$*

*$P < 0.01$.

firing rate of the visual response increased up to 0.044 cd/m^2 in the gap task and up to 0.4 cd/m^2 in the delay task. In the delay task an additional increase was observed between 0.4 and 42.5 cd/m^2 (corrected pairwise comparisons, $P < 0.01$).

Finally, the decay rate of the initial burst of the visual response was also modulated by target luminance (Fig. 4D). As luminance increased, the rate of this decay became faster, leading to a steeper downward slope of the signal up to 0.044 cd/m^2 in the gap task, and up to 0.4 cd/m^2 in the delay task. A further decrease was observed between 0.4 and 42.5 cd/m^2 in the delay task (corrected pairwise comparisons, $P < 0.01$).

Relationships between visual response properties

To assess how the luminance-modulated properties of the visual response were interrelated (i.e. how the beginning and end of the visual response was related to the peak), we correlated peak magnitude with VROL and decay rate (Fig. 4E) (median r values were used to compensate for distribution skew). We found that both VROL and decay rate were significantly negatively correlated with the peak magnitude of the visual response (median r: VROL, 0.79; decay rate, 0.76). Thus, the later the visual signal arrives in the SC, the weaker its magnitude and the shallower its decay rate.

Linking visual response properties to saccade behavior

We have shown that target luminance modulated both saccade behavior (Fig. 2) and the properties of the visual response in the SC (Figs 3 and 4). To link modulations of neural activity to behavior, we correlated the properties of the neural response of V and VM cells to saccade behavior. These correlations assessed the influence of the initial phasic visual response on the sensorimotor transformation that determined the timing and metrics of the resulting eye movement. All correlations were performed with data collected in the gap task when movement initiation was a direct consequence of target appearance. Only data from the gap task were analysed because in this task the monkey reacted reflexively to the appearance of the target unlike the delay task where there was an additional 500–800 ms to detect and process the target stimulus prior to the saccade (Fig. 1A and B). The removal of all short-latency and express saccades (see Methods and Table S1 for details) ensured that all correlations between the visual response properties and saccade behavior were not contaminated by the motor response that occurs 20 ms before the saccade (Sparks, 1978; Munoz & Wurtz, 1995).

The left column of Fig. 5 illustrates the cumulative distributions of correlation coefficients between visual response properties (VROL, growth time to the visual peak, peak magnitude of the visual response and the decay rate of the initial visual response) and saccade behavior (SRT, peak saccade velocity, saccade endpoint accuracy error) for each neuron across all seven luminance levels. Filled circles denote significant correlations. Overall, all visual response properties analysed were significantly correlated with saccade behavior (Fig. 5, right panels) (one-sample t tests, all $P < 0.02$). The strongest correlations with saccade behavior were VROL and peak magnitude. These strong correlations suggest that VROL and peak magnitude of the visual response influence the latency, velocity and accuracy of the ensuing saccade. The growth and decay rate of the visual response had weaker correlations, but they were still significantly correlated with saccade behavior. It is possible, however, that these correlations were only weaker because of potential increased trial-by-trial variability in visual response onset and offset times at lower luminance levels where signal-to-noise ratios were poor (see Methods).

We performed a three-factor repeated-measures ANOVA (three saccade behaviors, four visual response properties) to quantify the differences in overall correlation strength between saccade behavior (SRT, peak velocity, endpoint error) and visual response properties (VROL, growth time, peak magnitude, decay rate). For the purpose of comparing within the ANOVA, correlations with peak saccade velocity were multiplied by −1 to ensure that the sign (positive or negative) of all mean correlations was matched. There was a main effect of saccade behavior in the correlation coefficients ($F_{2,218} = 6.24$, $P < 0.01$), with SRT being the best correlated overall and saccade accuracy being the weakest. There was also a main effect of visual response property ($F_{3,327} = 59.7$, $P < 0.01$) such that VROL correlated the strongest with saccade behavior followed by growth time, peak magnitude and then decay rate. There was no interaction between saccade behavior and visual response property ($F_{6,654} = 1.58$, $P = 0.17$; Fig. 5, right panels).

To confirm that the correlations observed between visual response properties and saccade behavior were independent of the experimentally manipulated task luminance condition, we collapsed the data across luminance conditions and reanalysed the data. This independent analysis yielded the same trend of correlations that were observed in Fig. 5 (see Fig. S3 and Supporting Information).

Overall, although some visual response signal properties correlated better to saccade behavior than others, significant correlations were found between SRT, saccade velocity and saccade accuracy and all visual signal properties measured. This suggests that the visual response not only influenced when the saccade was initiated, but it also carried through the visuomotor transformation to influence the metrics of the saccade.

Target luminance modulates the saccade motor response

To identify elements of the motor response that varied with target luminance, we analysed the peak magnitude (Fig. 6A) of the motor response at saccade onset and the rate of growth in discharge (Fig. 6B) of the motor response (i.e. slope) during a pre-saccade epoch (from 25 ms before the onset of the movement to the onset of the movement). We analysed the slope instead of absolute growth rate of the motor response because in the gap task it was not possible to identify the precise onset of the motor response following the visual response. Figure 6 shows the peak and slope of the increase in the motor response for VM neurons across the seven luminance levels tested. There was a main effect of motor peak with target luminance

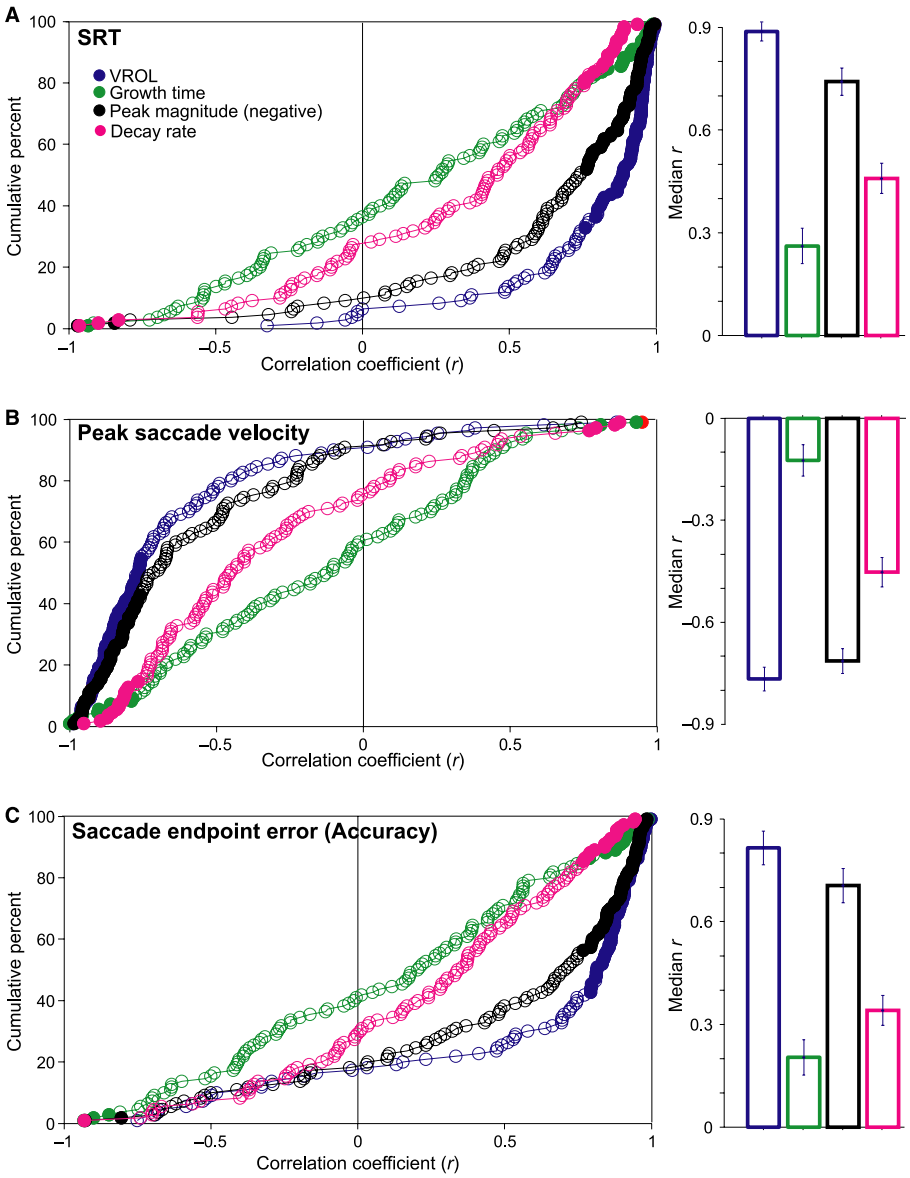

FIG. 5. Cumulative distributions of correlation coefficients (left column) and median *r* values (right column) for each behavioral and visual sensory neural variable measured within each recorded neuron in the gap task. Filled circles in the cumulative plots denote statistically significant correlations (within neuron). Color denotes the neural signal property that was correlated: VROL (blue), growth time (time from VROL to peak: green), peak magnitude (black) and decay rate (pink). All correlations with peak magnitude were multiplied by −1 to match with VROL, growth time and decay rate values. Each neural signal property was correlated independently of SRT (A), peak saccade velocity (B) and saccade endpoint error (C). Median *r* values denote how much variance was accounted for by each correlation.

($F_{6,378}$ = 15.3, $P < 0.01$), although the motor burst was only significantly modulated at the dimmest target intensity rate (corrected pairwise comparisons, $P < 0.01$; Fig. 6A). This weak modulation of motor burst magnitude at the dimmest target luminance was probably related to the reduced accuracy of the saccade endpoint at this luminance (Fig. 2C). Therefore, saccades did not always fall within the center of each neuron's motor response field, and this resulted in reduced SC motor discharge (Stanford & Sparks, 1994; Marino *et al.*, 2008). There was also a main effect of the growth rate of the motor response ($F_{6,378}$ = 3.56, $P < 0.02$). The slope of the motor burst was only significantly different between the dimmest and brightest target luminance ($P < 0.01$) (Fig. 6B). Thus, although modulations of the motor response were correlated with target luminance (Fig. 6), these correlations were weaker than those computed with the visual response (Fig. 5).

Linking motor response properties to saccade behavior

It has been previously shown that the timing of the saccadic motor burst in the SC almost perfectly correlates with saccade occurrence (Sparks, 1978). We extend this important finding by examining how luminance-related changes in the peak magnitude and slope of the motor burst correlate with the saccade behavior. Figure 7 shows the cumulative distribution of correlation coefficients between the peak magnitude and growth rate (slope) of the motor response with saccade behavior (SRT, saccade velocity and saccade accuracy). Both the peak

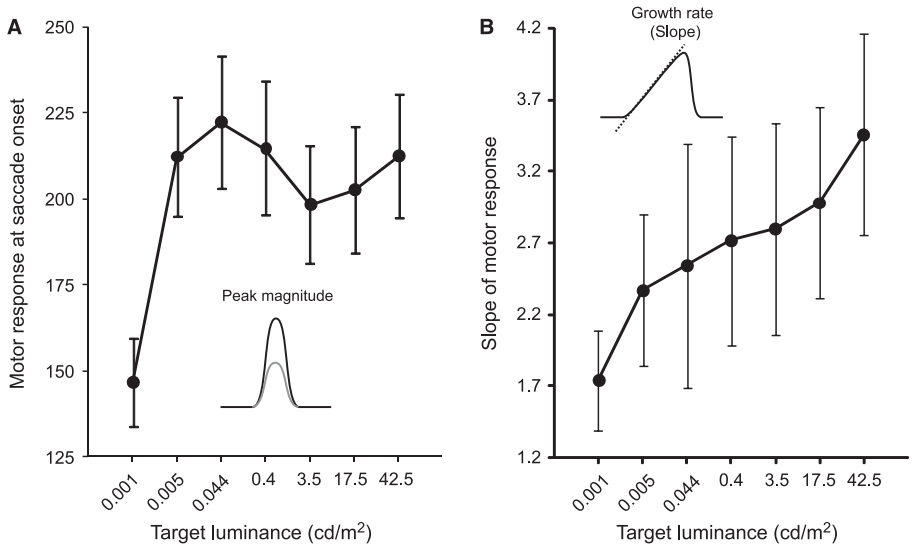

FIG. 6. Effects of target luminance on the properties of the saccade motor response in the gap task collapsed across all VM neurons. (A) Magnitude of the motor burst at saccade onset. (B) Mean growth rate (slope) of the motor response (25 ms before saccade onset to saccade onset). Error bars denote standard error.

and the growth rate of the motor response showed significant correlation with saccade behavior; however, the peak magnitude of the motor response was significantly more correlated with all saccade behaviors than its slope (growth rate) (*t* tests; SRT: $t = 3.79$, d.f. = 126, $P < 0.001$; peak velocity: $t = -2.82$, d.f. = 126, $P < 0.006$; saccade endpoint error: $t = 4.36$, d.f. = 126, $P < 0.001$; Fig 7, right column).

Discussion

Here, we have linked specific components of the visual response in the SC to saccade generation. Target luminance modulated multiple components of the neural response in the SC, which then correlated to the resultant saccade behavior. Of these modulations, the peak magnitude and onset time of the visual response correlated more strongly to saccade latency and metrics (peak velocity and accuracy) than did the growth time or decay rate. These correlations between the earliest part of the visual response and the timing and metrics of the saccade indicate that the properties of the visual response carry through into the neural computations that determine how fast the eyes move and how accurately they align the fovea with a visual stimulus. It has been previously established that the timing of the saccadic motor burst in the SC reflects the closest association between the neural responses in the SC and the performance of the saccade. This is demonstrated by a near perfect ($r = 0.99$) correlation with the timing of the motor burst and the launching of each saccade (Sparks, 1978). Here we report additional important correlations between the peak magnitude and growth rate of the motor response with variations in saccade behavior when target luminance is manipulated.

The data presented here indicate that elements of the earliest part of the visual response may play a crucial role in the subsequent calculation of the sensory–motor transformation that underlies visually guided saccades and influences behavior. This is important because these signal properties arrive in the SC at latencies that approach minimum afferent delays and precede the actual motor behavior by up to hundreds of milliseconds. This suggests that the sensory–motor transformation that takes place between the visual and motor responses in the SC is being influenced by the timing and magnitude of the earliest part of the visual response.

Relation to previous work

Previous studies have examined the effects of target luminance or contrast on visual activity within the SC. Contrast responses were initially described in the superficial SC of anesthetized cats (Bisti & Sireteanu, 1976), but have also recently been found across the SC of humans with functional magnetic resonance imaging (Schneider & Kastner, 2005) and in awake monkeys using single cell electrophysiological recording (Bell *et al.*, 2006; Li & Basso, 2008). However, the conclusions from these studies were limited because the luminance- and contrast-modulated changes of the visual and motor responses were never systematically tested or linked to behavior. Specifically, Bisti & Sireteanu (1976) reported changes in firing rate and Schneider & Kastner (2005) reported changes in functional magnetic resonance imaging blood oxygen level dependent activation in the visual response in the SC, but neither of these studies correlated these changes with saccade behavior. Likewise, both Bell *et al.* (2006) and Li & Basso, 2008 report changes in the VROL with luminance contrast; however, Bell *et al.* (2006) used only two luminance levels and did not test different luminance levels within the same neurons, while Li & Basso (2008) never correlated the observed changes in VROL or peak magnitude to behavior. Here we tested a systematic range of target luminance levels within the same neurons and determined how changes to the visual and motor response link with saccade latency and metrics.

Stages of visuomotor processing

The luminance-driven modulations of the visual response in the SC are propagated directly into the premotor circuit controlling saccade production. Specifically, the visual and motor responses from VM neurons are aligned spatially (Marino *et al.*, 2008) and these neurons project via the predorsal bundle to the saccade-generating circuit in the brainstem (Moschovakis *et al.*, 1990, 1996; Kato *et al.*, 2006;

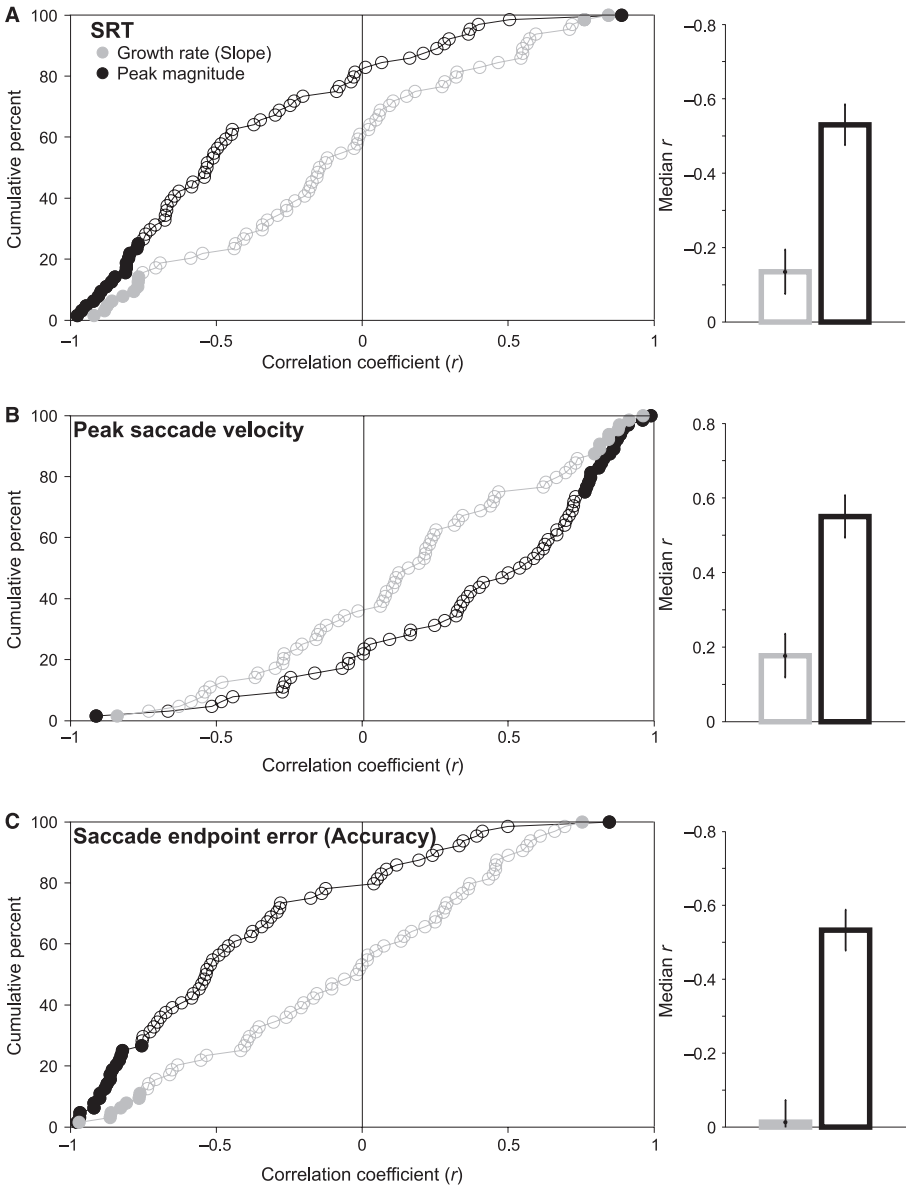

FIG. 7. Cumulative distributions of correlation coefficients (left column) and median *r* values (right column) between each behavioral and saccade motor neural variable measured (VM neurons only). Filled circles in the cumulative plots denote statistically significant correlations (within neuron). Color denotes the neural signal property that was correlated: growth rate slope (gray), and magnitude of the motor burst at saccade onset (black). Each neural signal property is correlated independently of SRT (A), peak saccade velocity (B) and saccade endpoint error (C). Median *r* values denote how much variance was accounted for by each correlation. Error bars denote standard error.

Rodgers *et al.*, 2006). Thus, luminance-based modulations of visual signals are probably used by both oculomotor (Edelman & Keller, 1996; Dorris *et al.*, 1997; Sparks *et al.*, 2000) and head premotor circuits (Corneil *et al.*, 2004, 2007, 2008) to guide visually triggered orienting.

In our study, altered luminance correlated with changes to the timing (VROL, peak time) and shape of the visual response (peak magnitude, growth time, decay rate). At brighter target luminance values, the sensory input appeared to influence visual processing more rapidly via the quicker arrival, faster growth and increased magnitude of the visual response. We hypothesize that the underlying neural mechanisms that generated these modulations in the visual response should occur early in visual processing and then propagate through stages to influence the motor response because they were observed within both V and VM neuron populations within the SC. It is presumed that V neuron subtypes are located more superficially in the SC than VM neuron subtypes (McPeek & Keller, 2002), but only saccade-related neurons in the SC have been histologically linked to specific anatomical layers where they have been identified in the intermediate gray and optic layers (Moschovakis *et al.*, 1988; Ma *et al.*, 1991). We did not find any significant difference in the timing of the VROL between V and VM populations (Fig. S2). This suggests that simple luminance channels relay visual sensory information rapidly across both the superficial and the intermediate layers of the SC. However, there was a tendency for the visual peak to increase at a higher rate in the V relative to the VM neurons (Fig. S2). Multiple interpretations could explain this temporal asynchrony. One possibility is that it could result from processing stages between the superficial

and intermediate SC. The superficial SC receives input from early stages of visual processing (i.e. the retina and primary visual cortex) (Robinson & McClurkin, 1989; Sherman, 2007). Visual activity might reach its peak more slowly in the intermediate SC because it is filtered through additional connections from the superficial SC and additional extrastriate visual cortical regions (Kunzle *et al.*, 1976; Fries, 1984; Cusick, 1988; Lock *et al.*, 2003). Alternatively, enhanced inhibition could cause the initial visual burst to terminate earlier in the superficial SC, thereby shifting the time of the peak earlier relative to intermediate SC. Finally, there was a tendency for the peak magnitude of the visual response to be larger in VM than in V neurons. If this trend is real, it indicates that the visual sensory signal could be amplified as it passes through the stages of processing prior to the VM neurons that influence resulting saccades. However, it is also possible that this decreased visual peak in V neurons relative to VM neurons could be compensated for if the population of V neurons was significantly greater than the population of VM neurons.

In addition to the SC, other important saccade-related areas like the frontal eye fields (FEF) and lateral intraparietal area (LIP) probably contribute to the calculation of the sensorimotor transformation between the visual and motor responses (Schall & Thompson, 1999; Munoz & Schall, 2003; Andersen & Cui, 2009). These areas are heavily interconnected with the SC (Pierrot-Deseilligny *et al.*, 1991; Pare & Wurtz, 2001; Munoz & Schall, 2003). Neurons in LIP and FEF have visual and saccadic responses and their activity between the visual event and the initiation of the saccade no doubt also contributes to saccade generation. It is therefore quite likely that similar correlations will emerge for these neurons if they were recorded in our experimental paradigm.

Immediate vs. delayed saccades

The delay task was used to dissociate visual- and motor-related responses temporally by imposing a delay period (500–800 ms) between the appearance of the T and the go signal to initiate a saccade (disappearance of the FP). This condition required deliberate control by the subject to prevent a reflexive saccade from being made to the T. Thus in the delay task, the more natural or automatic 'visual grasp reflex' (Hess *et al.*, 1946) must be inhibited during the delay period. As a consequence, the effects of luminance on saccade latency and metrics was reduced in the delay task relative to the gap because of the increased processing and integration time of the target stimulus. This afforded the subject 500–800 ms of additional time after the appearance of the T to detect and plan a saccade toward it.

One way to manipulate excitability in pre-motor circuits and reduce SRTs is to introduce a gap period between FP disappearance and T appearance (Saslow, 1967; Fischer & Weber, 1993; Dorris & Munoz, 1995). This reduction in SRT has been attributed to fixation disengagement prior to target appearance. Thus, in contrast to the delay task, saccade initiation in the gap task is a direct consequence of T appearance whereby a movement must be immediately and reflexively generated toward it. Furthermore in the gap task, no extra task-imposed time is available for the saccade system to more fully detect and process the target stimulus. Thus, for low luminance targets in the gap task, saccades might be launched before all possible useful visual information about the T is received and processed.

In both the gap and the delay tasks we observed increases in SRT and peak velocity as well as decreases in accuracy and error rate at the two dimmest luminance levels (Fig. 2). In addition, it was only at the dimmest luminance that we also observed a decrease in the saccadic motor response (Fig. 6). One possible explanation for these task-independent effects of luminance near detection threshold is that they are similar to those observed during memory-guided saccades. Memory-guided saccades are initiated toward a visible stimulus and require a subject to saccade to a remembered T location. Similar to our findings at the lowest luminance levels, memory-guided saccades are slower, more inaccurate (Gnadt *et al.*, 1991; White *et al.*, 1994; Edelman & Goldberg, 2001), and also demonstrate a reduced motor response in the SCi (Stanford & Sparks, 1994; Edelman & Goldberg, 2001, 2003). Thus, it is possible that visual Ts near detection threshold mimic memory guided saccades because working memory may also be recruited to aid or augment weak or noisy visual sensory input.

Evidence for visual inhibitory feedback

We observed a steepening slope in the decay of the initial visual response with increasing luminance (Figs 3, left column, and 4D). This could indicate that a neural mechanism exists whereby the strength of the initial visual response determined the magnitude of the suppression of the later part of the same response. This suppression could play a role in terminating the initial phasic component of the visual response and may provide a mechanism for controlling express saccade generation (fast saccades with SRTs that approach minimum conduction delays) that might otherwise be triggered if the visual response grows large enough to cross a neural threshold and trigger a saccade (Edelman & Keller, 1996; Dorris *et al.*, 1997).

It is unclear whether this observed suppression in the visual response could result from local inhibitory circuitry within the SC (Isa & Hall, 2009) or if it is relayed from upstream structures. One possible upstream candidate is the visual sector of thalamic reticular neurons (TRNs), which have been shown to receive excitatory inputs from the lateral geniculate nucleus (LGN) and project an inhibitory signal back to the LGN (Crabtree & Killackey, 1989; Conley & Diamond, 1990; Harting *et al.*, 1991; Uhlrich *et al.*, 2003; Fitzgibbon *et al.*, 2007). Interconnections between the SC and TRNs have been previously identified (Wilson *et al.*, 1995; Vaccaro & Mitrofanis, 1996; Kolmac & Mitrofanis, 1998; Jones, 2007), so that it is at least possible that visual activity in the SC could influence inhibitory TRN feedback and vice versa to influence the underlying visuomotor transformation. This would suggest a new role for the SC in controlling visually guided saccades. Future studies that assess the relationships between visual responses in the SC and TRN activation will be important to address these questions.

Extrinsic vs. intrinsic mechanisms

Increases in the luminance of a stimulus have been previously shown to decrease the visual response onset latency and increase the response magnitude of neuronal activity throughout several visual sensory areas including: primary visual cortex (V1), visual middle temporal area (MT), extrastriate visual cortical area V4 and SC (Albrecht & Hamilton, 1982; Sclar *et al.*, 1990; Gawne *et al.*, 1996; Gawne, 2000; Bell *et al.*, 2006; Williford & Maunsell, 2006; Lee *et al.*, 2007; Li & Basso, 2008). The widely distributed nature of these effects across multiple visual–sensory brain areas suggests an extrinsic mechanism that could be used to bias sensory signals in order to influence both visual processing and visuomotor transformations.

It has been shown that trial-by-trial variability in visual response onset times also correlates strongly with SRT in V1 (Lee *et al.*, 2010). If such individual trial variability results from purely intrinsic neural mechanisms then it is possible that the neural effects we observed could be influenced independently of the external neural inputs. This

is possible because any increased onset variability at lower luminance levels could influence the trial-averaged steepness of the decay rate and the growth time of the visual response. Thus, it is unclear whether the modulations in the visual response that we reported with changing luminance resulted entirely from extrinsic inputs or whether they were also influenced by intrinsic neuronal mechanisms.

Implications for modeling of the visual system

Although the sudden onset of visual stimuli can be uncommon during natural vision, many activities such as driving a rapidly moving motor vehicle, watching TV or playing video games often result in the sudden onset of visual information. During such viewing conditions where multiple visual stimuli are competing for foveation by the saccade system, a single saccade target must be selected from multiple visual stimuli that can be spread out spatially across the retina. The changes to the timing, magnitude and shape of visual responses that we observed in the SC with changing luminance may represent a possible neural mechanism by which the visual system helps resolve competition and could influence the order in which individual visual stimuli are selected for upcoming saccades. Similar selective enhancement or inhibition of salient features in the visual system are hypothesized to be a key feature for influencing saccades during natural viewing (Itti & Koch, 2001; Berg *et al.*, 2009). In addition, winner-take-all spatial competition models which predict both SRT and which visual targets are selected for saccades could potentially be significantly improved by incorporating these multiple visual signal properties (onset, peak, slope, decay) into their winner-take-all spatial competition models (Kopecz, 1995; Itti & Koch, 2001; Trappenberg *et al.*, 2001; Marino *et al.*, 2011). Because natural vision in primates commonly involves making visually guided saccades to targets of varying luminance, future studies of the visual or saccade system will benefit from a better understanding of how luminance impacts both sensory processing and motor behavior that these results provide.

Supporting Information

Additional supporting information may be found in the online version of this article:

Fig. S1. (A) Schematic representation of temporal events in the visual response field mapping task for the fixation point (FP), target (T) and eye position. (B) 182 target locations (black dots) presented at 24 different directions and 6–10 different eccentricities used to map the visual response fields. (C) Visual response field for the representative visually responsive neuron shown in D and E. (D,E) Target-aligned rasters and spike density functions of a representative visually responsive neuron to locations outside (D) and inside its visual response field (E).

Fig. S2. Effects of neuron V and VM subtype on visual sensory response properties recorded at the brightest luminance (42.5 cd/m^2) in the delay (gray) and gap (black) tasks. (A) VROL, (B) peak magnitude, (C) growth time, (D) decay rate. Asterisks denote statistical significance (*t* test, $P < 0.05$). All error bars denote standard error.

Fig. S3. Correlations between visual response properties and saccade behavior collapsed across luminance conditions. Cumulative distributions of correlation coefficients (left column) and median *r* values (right column) for each behavioral and visual sensory neural variable measured. Filled circles in the cumulative plots denote statistically significant correlations (within neuron). Color denotes the neural signal property that was correlated: VROL (blue), growth time (time from VROL to peak: green), peak magnitude (black) and decay rate (pink). All correlations with peak magnitude were multiplied by −1 to match with VROL, growth slope and decay rate values. Each neural signal property was correlated independently of SRT (A), peak saccade velocity (B) and saccade endpoint error (C). Median *r* values denote how much variance was accounted for by each correlation.

Table S1. Percentage saccadic reaction times removed from analysis.

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Abbreviations

FEF, frontal eye fields; FP, fixation point; LGN, lateral geniculate nucleus; LIP, lateral intraparietal area; SC, superior colliculus; SRT, saccadic reaction time; T, target; TRNs, thalamic reticular neurons; VROL, visual response onset latency; V1, primary visual cortex.

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