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RESEARCH ARTICLE

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Stimulus intensity modifies saccadic reaction time and visual response latency in the superior colliculus

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Abstract Performance in a reaction time task can be strongly influenced by the physical properties of the stimuli used (e.g., position and intensity). The reduction in reaction time observed with higher-intensity visual stimuli has been suggested to arise from reduced processing time along the visual pathway. If this hypothesis is correct, activity should be registered in neurons sooner for higher-intensity stimuli. We evaluated this hypothesis by measuring the onset of neural activity in the intermediate layers of the superior colliculus while monkeys generated saccades to high or low-intensity visual stimuli. When stimulus intensity was high, the response onset latency was significantly reduced compared to low-intensity stimuli. As a result, the minimum time for visually triggered saccades was reduced, accounting for the shorter saccadic reaction times (SRTs) observed following high-intensity stimuli. Our results establish a link between changes in neural activity related to stimulus intensity and changes to SRTs, which supports the hypothesis that shorter SRTs with higher-intensity stimuli are due to reduced processing time.

Keywords Superior colliculus · Visual · Express saccades · Intensity · Response onset latency

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Introduction

Increasing the intensity of a visual stimulus reduces the time to orient the visual axis to that stimulus (Boch et al. 1984; Darrien et al. 2001). This effect of visual intensity is also seen in other types of behavioral tasks (e.g., manual response tasks: Ulrich et al. 1998; Kammer et al. 1999; response to motion onset: Burr and Corsale 2001), which suggests that the underlying neural mechanism occurs relatively early in the visual processing pathway. It has been hypothesized that the effect of visual intensity on saccadic reaction time (SRT) may originate from accelerated processing at the retinal level (Boch et al. 1984; Barbur et al. 1998). Indeed, data obtained from mammalian retinal ganglion cells have revealed that higher-intensity stimuli evoke neural activity with shorter onset times (Kuffier 1953; Lennie 1981; Barbur et al. 1998). These observations strongly support the above hypothesis and yet the relationship between changes in neural response onset latency (ROL) and reaction time has yet to be clearly demonstrated.

While conducting a study examining the role of the intermediate layers of the superior colliculus (dSC) in crossmodal integration (Bell et al. 2005), we noticed an intriguing effect of visual stimulus intensity on specific aspects of the neural response of dSC neurons that provided valuable insight on this issue. Single unit activity was recorded from neurons in the dSC while monkeys generated saccades to high- and low-intensity visual and audiovisual stimuli. The dSC is an ideal structure for investigating the effect of visual intensity on saccadic performance because it receives numerous visual inputs and is highly involved in transforming visual information into commands to guide gaze shifts (see Munoz et al. 2000; Sparks et al. 2001 for review). Two different saccade tasks were used. The delayed-saccade task employed a delay between stimulus onset and saccade generation to dissociate the sensory- and motor-related discharges (Edelman and Keller 1996; Munoz et al. 2000; Sparks et al. 2000), allowing for assessment of the effect

of stimulus properties on the isolated sensory response. The gap-saccade task facilitated the merging of the sensory and motor discharges, increasing the probability that modifications to the sensory response would influence behavior. Our data show that increasing visual stimulus intensity reduces the onset latency of visual responses in the dSC, thereby facilitating the earlier generation of saccades. Data have been presented in abstract form (Bell et al. 2004b).

Methods

All procedures were approved by the Queen's University Animal Care Committee and were in accordance with the Canadian Council on Animal Care policy on the use of laboratory animals. Three adult male rhesus monkeys (*Macaca mulatta*), weighing between 6 and 12 kg, were used in this study. Animals were prepared for chronic experiments in a single surgical session (see Bell et al. 2005; Munoz and Istvan 1998 for details). Briefly, animals were implanted with scleral search coils to monitor eye position, a head restraint device, and a recording chamber allowing access to both SC. They were given at least 2 weeks to recover prior to behavioral training and experimentation.

Experimental procedures

The monkeys were trained to perform an audiovisual saccade paradigm with two interleaved saccade tasks. Although these data were collected for a different experimental purpose, the results presented here represent a completely separate analysis stream and do not pertain to the other study (Bell et al. 2005). Unless otherwise indicated, only the data obtained with the unimodal visual target are considered here. Each trial began with the appearance of a central, visual fixation point (FP), which the monkeys were required to fixate. After an initial fixation period of 800–1200 ms, one of the following two tasks was presented. On half of the trials, the saccade target was presented and the FP remained illuminated for an additional 400–800 ms before being extinguished ("delayed-saccade task"). The removal of the FP was the animal's cue to generate a saccade toward the target. On the other half of the trials, the FP was extinguished 200 ms prior to the presentation of the saccade target ("gap-saccade task") and the monkey generated a saccade to the target. The two different saccade tasks were presented randomly interleaved with equal probabilities.

The visual saccade target was presented either into the receptive field of the neuron (such that the saccade necessary to foveate the target was the neuron's preferred vector; see below) or to its diametrically opposite position, across the horizontal and vertical meridians.

Details regarding the behavioral and neuronal recording techniques are described elsewhere (Bell et al. 2004a). The visual stimuli were point images ($<1.0°$) generated either with a LASER (8.0 cd/m^2; Power Technologies) or an LED (0.05 cd/m^2) to yield two very different intensities. They were reflected off galvanometer-driven mirrors and back projected onto a tangent screen. Monkey Z performed the task with only high-intensity stimuli, whereas Monkeys O and R performed the task with both high and low intensities (see Table 1). For the majority of recording sessions, only one stimulus intensity was presented. Monkey O performed the task with low-intensity stimuli first, followed by high-intensity stimuli; and vice versa for Monkey R. In addition, for a number of neurons collected from monkey R, we also collected data while mapping visual receptive fields using a high-intensity visual stimulus (LASER, 8.0 cd/m^2) presented in an identical gap-saccade task format as the main experimental paradigm. The response to the visual stimulus in the main experimental paradigm was then compared to the mean response evoked when the high-intensity stimulus was positioned in the receptive field of the neuron, allowing for a within neuron comparison of responses to high- versus low-intensity stimuli in the gap-saccade task for a small subpopulation of neurons (e.g., Fig. 2a).

Data analysis

Data were analyzed offline using a Sun Ultra 60 Sparcstation running user-generated programs and a Pentium PC running MatLab software (Mathworks Inc.). Behavioral data were first run through an automated saccade detection program, which identified the beginning and end of each saccade based on velocity and acceleration template

Table 1 Behavior

	Monkey R		Monkey O		Monkey Z	
	High	Low	High	Low	High	Low
Mean SRT	107±3	147±1	145±3	186±2	177±3	NA
No. of trials	92	1073	268	596	440	NA
No. of express saccades	22	332	16	34	8	NA
No. of anticipations	5	122	9	70	25	NA
No. of anticipations (0–65 ms)	5	69	9	37	25	NA
Percent of express saccades (correct %)	26	36	6	7	2	NA
Percent of anticipations (total %)	5	11	3	12	6	NA
Percent of Anticipations (0–65 ms) (total %)	5	6	3	6	6	NA
No. of neurons						
Sensory only	0	12	1	2	6	0
Sensory-motor	4	23	9	9	16	0

Express saccades to high-intensity stimuli were defined as those with SRTs 65–95 ms (Fig. 1a). Low-intensity stimuli did not produce a bimodality and so express saccades were defined based on the neurophysiological data (Fig. 4d) as those with SRTs 100–130 ms
NA not available

matching (Waitzman et al. 1991). All marks were later verified by the experimenter and adjusted when necessary. Saccades with latencies above 400 ms or those that landed outside an acceptance window of 2–3° surrounding the target were excluded from further analysis.

Neuronal responses were analyzed by constructing spike density functions on a trial-by-trial basis based on an exponential growth/decay function (Thompson et al. 1996). The growth time constant was set to 1 ms and the decay constant was set to 20 ms. ROLs for each trial were defined as the initial point where the activation level exceeded the pretarget activity plus three standard deviations. In order to be classified as a valid response, the activity had to remain above this threshold for a minimum of 10 ms. The delayed- and gap-saccade tasks exhibit different levels of pretarget activity (Munoz et al. 2000). Therefore, pretarget activity was defined as 400–0 ms prior to stimulus onset in the delayed-saccade task and 100–0 ms prior to target onset in the gap-saccade task. Visually responsive neurons were defined as those for which the average activity in a 200 ms window following target appearance in the delayed-saccade task exceeded the pretarget activity by at least 50 spikes/s. Saccade-related neurons were defined as those with saccade-aligned activity (peak activity ±5 ms surrounding saccade onset) greater than 80 spikes/s for saccades to the neuron's preferred direction and eccentricity in the delayed-saccade task. All classifications were verified by the experimenter for consistency and accuracy. Population analysis of the neuronal data was performed using Wilcoxon rank sum tests ($P<0.05$) unless otherwise stated. All data are presented as mean ± standard error of the mean, unless otherwise indicated. For display purposes only, population spike density functions are shown as floating averages of 10 ms binwidths, plotted every 5 ms.

Results

The distribution of SRTs in the gap-saccade task is shown in Fig. 1 for the two monkeys that performed the task at both high and low intensities. A detailed breakdown of all three monkeys is provided in Table 1. Monkeys exhibited a bimodal distribution of SRTs for saccades to the high-intensity stimulus (Fig. 1a), with the first, smaller, mode ranging from about 65 to 95 ms and the second, larger, mode ranging from 95 to 200 ms. These two modes correspond to express and regular saccades, respectively (Fischer and Boch 1983; Fischer and Weber 1993; Paré and Munoz 1996). Saccades with SRTs below 65 ms were classified as anticipatory responses because they had equal probability of being initiated toward or away from the target (Fig. 1d). By contrast, no clear bimodality was evident for saccades to the low-intensity targets under the stimulus conditions used in this study (Fig. 1b). The equal ratio of correct versus incorrect saccades extended to approximately 100 ms, indicating that low-intensity stimuli evoked correct *visually triggered* saccades approximately 35 ms *later* compared to the high-intensity stimulus (Fig. 1c, d). Monkeys generated almost twice the proportion of anticipatory responses in the low-intensity stimulus sessions (214/1748, 12% vs. 55/820, 7%; Table 1). This is not surprising considering the longer range of time defining anticipatory response in the low-intensity condition.

The minimum SRT for correct visually triggered saccades was highly correlated with the ROL of visual responses in the dSC. Figure 2a shows the responses of two dSC neurons to high (black traces) versus low (gray traces) intensity stimuli in the gap-saccade task. There is a marked reduction in ROL to the high-intensity stimuli as compared to the low-intensity stimuli. This was true across the sample population. Figure 2b and c shows population spike density curves for all sensory (high: $N=7$, low: $N=14$) and sensory-motor (high: $N=29$, low: $N=32$) neurons recorded from the intermediate layer neurons in the gap and delayed-saccade tasks, respectively. ROL was significantly influenced by stimulus intensity (ANOVA: $F(2,78)=10.930$, $P<0.001$). In the gap-saccade task, high-intensity stimuli evoked responses in dSC neurons with significantly shorter ROLs (Fig. 2b, d; mean ROL for high-intensity stimuli: 59±3 ms; mean ROL for low-intensity stimuli: 86±3 ms; $P<0.001$). The effect of stimulus intensity was also seen in the delayed-saccade task (Fig. 2c, e; mean ROL for high-intensity stimuli: 63±3 ms, black line; mean ROL for low-intensity stimuli: 99±3 ms, gray-filled bars; Wilcoxon Signed Rank Test, $P<0.001$).

The gap-saccade task produced a further reduction in ROL compared to the delayed-saccade task that was significant for low-intensity stimuli ($P<0.05$) but failed to reach significance for high-intensity stimuli ($P>0.10$), which may indicate that the minimum ROLs physiologically possible had been attained. Based on the similar differences in mean ROL (59 vs. 86 ms) and the onset of correct, visually triggered saccades (~65 vs. ~100 ms) across high- versus low-intensity stimuli in the gap-saccade task, it is logical to infer that the shorter reaction times following high-intensity stimuli were due to the shorter ROL which facilitates the earlier triggering of saccades.

In order to explore the possibility that stimulus intensity may also influence processing downstream of the dSC (i.e., in the brainstem oculomotor centers), we examined the time interval between the peak of the perisaccadic burst (defined as the peak firing rate when data are aligned on saccade onset) and the onset of the saccade for all saccade-related neurons. Figure 3 shows the time interval for high- versus low-intensity stimuli in the gap (Fig. 3a) and delayed (Fig. 3b) saccade tasks. The time interval for individual neurons ranged from approximately −20–40 ms surrounding saccade onset, indicating that the saccadic burst could peak shortly after saccade onset. Nonetheless, no significant difference between the two intensities was observed for either task (Fig. 3a, b; Ps>0.20), suggesting that stimulus intensity does not directly influence the motor processing stages in this task.

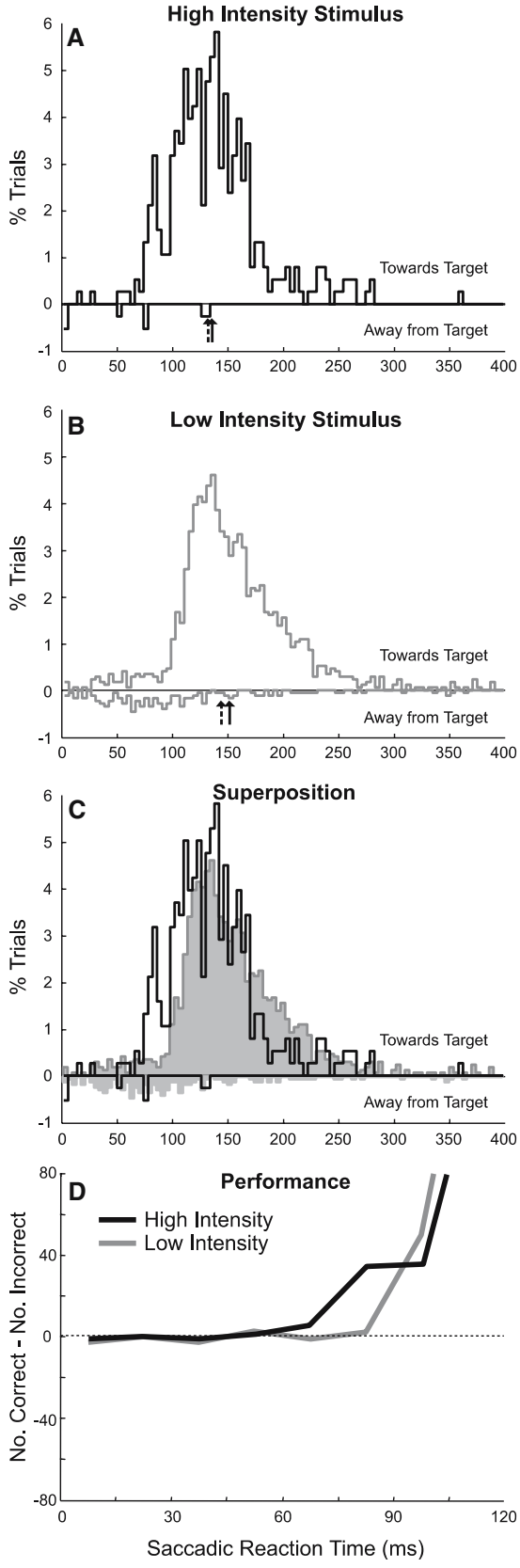

Fig. 1 The distribution of saccadic reaction times for Monkeys R and O in the gap-saccade task for high- (**a**) and low-intensity (**b**) stimuli and for high and low superimposed (**c**). Data are shown as the percent of the total number of trials (including anticipatory responses and direction errors). Negative values on the ordinate indicate saccades directed away from the target (i.e., direction errors). Histogram bin width is 4 ms. *Solid vertical arrows* indicate mean SRT of all correct trials; *dashed vertical arrows* indicate median SRT of all correct trials. **d** Performance in the gap-saccade task for all three monkeys. These traces plot the difference in number of correct responses versus the number of incorrect responses (saccade generated away from the visual target) for high- (*black* trace) and low- (*gray* trace) intensity stimuli. Data are grouped into 15 ms bins, aligned on the middle of each bin

Are low-intensity stimuli capable of eliciting express saccades?

One intriguing feature of the behavioral data is the absence of a bimodal distribution for saccades to the low-intensity stimulus (Fig. 1b), normally indicative of express and regular latency saccades (Fischer and Boch 1983; Paré and Munoz 1996). This raises the interesting question of whether low-intensity stimuli are capable of eliciting express saccades in this task. Due to the absence of a clear bimodality in the SRT histogram, we used a neurophysiological approach to address this question.

It has been proposed that express saccades are evoked when the incoming sensory signals combine with pretarget activity in the dSC to exceed saccadic threshold (Dorris et al. 1997; Munoz et al. 2000). As such, the neural profile often features a single peak when aligned on target onset, whereas regular saccades feature multiple peaks (Edelman and Keller 1996; Dorris et al. 1997; Sparks et al. 2000). This is illustrated for high-intensity stimuli for a single neuron and across the population of sensory-motor neurons (Fig. 4a, b).

We searched for evidence of single versus multiple peaks among the low-intensity stimuli data. Figure 4c shows an example of an individual sensory-motor neuron. In this example, trials with SRTs < 130 ms tended to show a single burst of activity of greater magnitude, whereas those with SRTs > 130 ms tended to show multiple peaks of activity. Figure 4d shows population spike density functions for all sensory-motor neurons across a range of SRTs in the low-intensity stimulus conditions. Although not as clear as in the high-intensity condition, these data show evidence of a single, larger peak at shorter SRTs and smaller, delayed peaks for trials with longer SRTs. Based on this qualitative evidence, it appears that express saccades *might be* elicited following low-intensity stimuli, but occur later in time relative to those evoked by higher-intensity stimuli. This is consistent with a behavioral study conducted in monkeys that reported express saccades within this approximate range using stimuli of similar intensity (Boch et al. 1984).

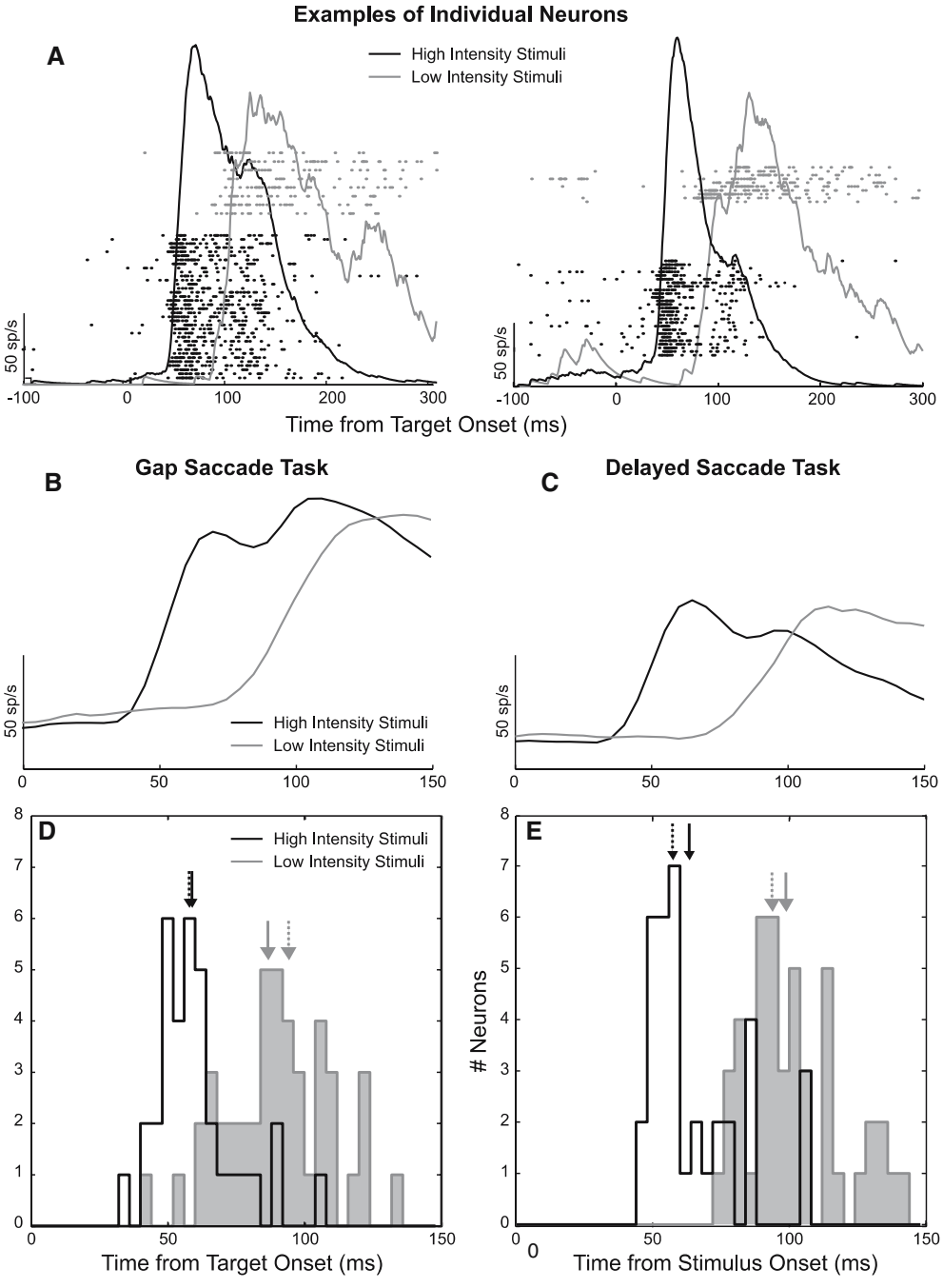

Fig. 2 Comparison of response onset latency for high- and low-intensity visual intensities in the delayed- and gap-saccade tasks. **a** Examples of the responses of two different dSC neurons to high (*black* traces) versus low (*gray* traces) intensity stimuli in the gap-saccade task. Each *row* of rasters indicates an individual stimulus presentation. These two neurons show a marked difference between the response onset latencies to the two stimuli, which was highly consistent within the small subpopulation of neurons exposed to both stimuli ($n=7$). **b, c** Population spike density curves for all sensory and sensory-motor neurons in the gap (**b**) and delayed saccade tasks (**c**). **d, e** Histograms of ROLs for sensory and sensory motor neurons in the gap (**d**) and delayed saccade tasks (**e**). Bin width is 4 ms. *Solid vertical black* and *gray arrows* indicate mean response onset latency for high- and low-intensity stimuli, respectively. *Dashed arrows* indicate median response onset latency

Discussion

The goal of this study was to explore the neural mechanisms underlying the relationship between stimulus intensity and SRT. Neural responses in the dSC were evoked earlier with the high-intensity stimulus (Fig. 2), facilitating the triggering of earlier saccades that led to shorter SRTs compared to the low-intensity stimulus (Fig. 1). These data provide valuable evidence in support of previous hypotheses suggesting links between decreased response onset latencies with reductions in SRT (Boch et al. 1984; Weber et al. 1991; McPeek and Schiller 1994). This further implies that increasing stimulus intensity should reduce RT in other nonoculomotor tasks as well, which is indeed the case (e.g., Pins and Bonnet 1996; Ulrich et al. 1998; Burr and Corsale 2001; Schiefer et al. 2001).

Consistent with previous behavioral studies (Boch et al. 1984; Weber et al. 1991), high-intensity stimuli evoked express saccades from 65 to 95 ms. The ability of low-intensity stimuli to evoke express saccades was less clear. Traditionally, express saccades have been defined by the presence of a bimodal distribution in the SRT histogram (e.g., Fig. 1a; see Fischer and Weber 1993). This method, which relies purely on behavior, is limited to those

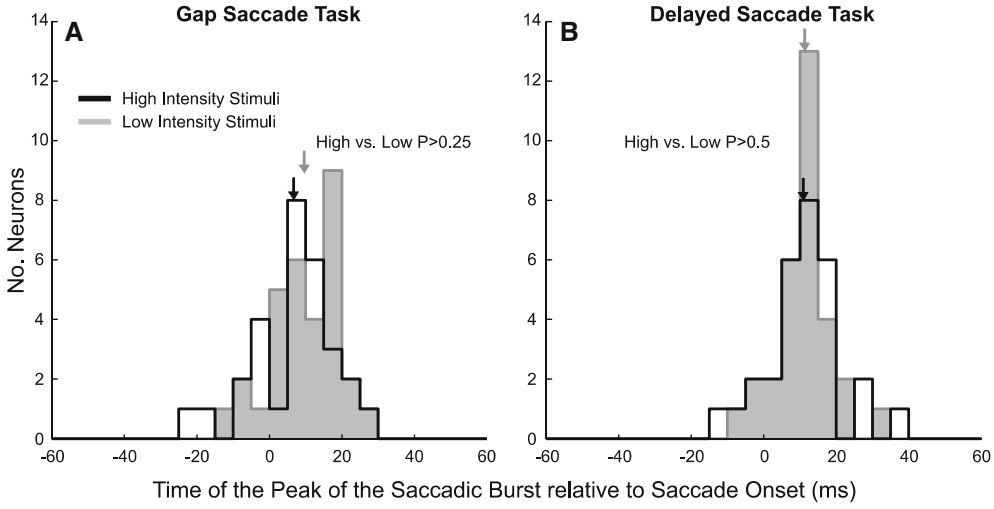

Fig. 3 Analysis of the time interval between the peak of the perisaccadic burst and the onset of the saccade. Histogram of the time of the peak of the burst relative to saccade onset for the gap (**a**) and delayed (**b**) saccade tasks. Histogram bin width is 5 ms. *Vertical arrows* indicate the mean interval for the high- and low-intensity stimuli

instances where bimodality is immediately evident, which is sometimes not the case (e.g., Reuter-Lorenz et al. 1991; Kingstone and Klein 1993). In the current study, we used neurophysiological correlates of express saccades to address whether express saccades can be triggered by low-intensity stimuli and estimate where the division between the two modes should be placed (Fig. 4). Saccades generated to the low-intensity stimuli with shorter SRTs, featured many of the hallmarks of express saccades (Edelman and Keller 1996; Dorris et al. 1997; Sparks et al. 2000), such as increased pretarget activity compared to regular latency saccades and a single peak of activity among sensory-motor neurons (Fig. 4), which is what would be expected with the overlap of both sensory- and motor-related discharges. Thus, while bimodality in the SRT distribution is perhaps the easiest way to distinguish between express and regular latency saccades, the methodology used here could be useful when such bimodality in the SRTs is not immediately evident.

The effect of stimulus intensity on reaction time is not limited to the visual modality (e.g., audition, Corneil et al. 2002). It seems unlikely, however, that behavioral changes following manipulation of auditory or tactile targets are evoked via the same mechanism described here for the visual modality. Unlike the visual system, which must first perform a relatively lengthy photochemical transduction

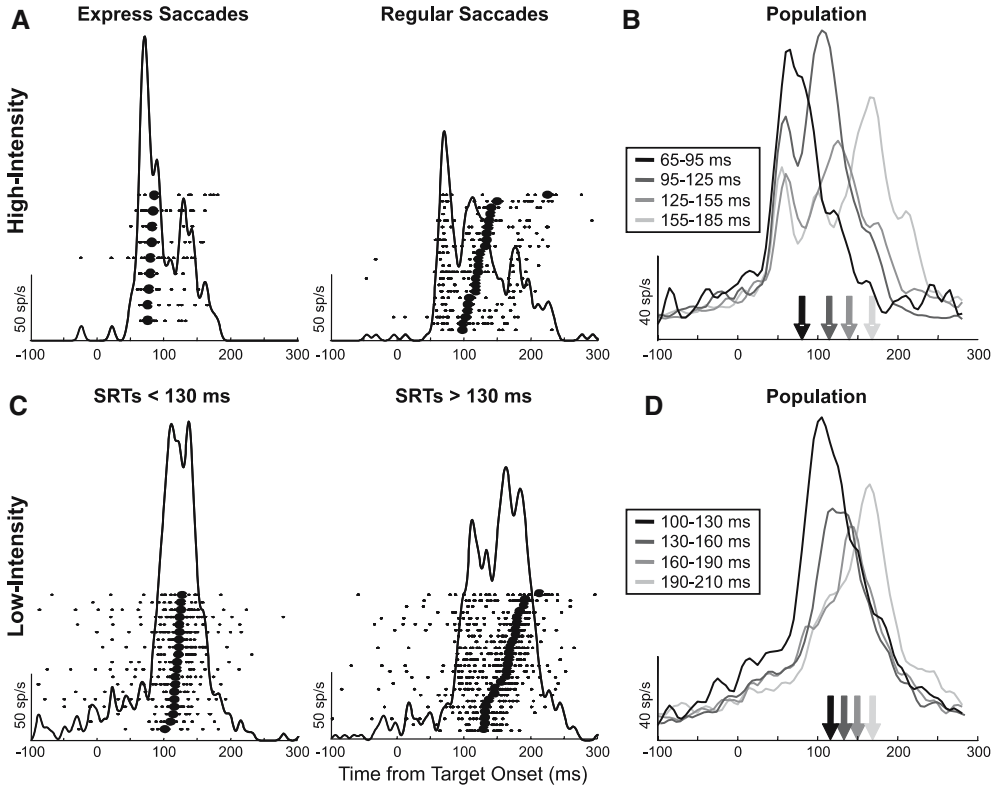

Fig. 4 Spike density functions for sensory-motor neurons recorded in the SC of monkeys performing the gap-saccade task. **a**, **c** Individual sensory-motor neurons recorded during presentation of high- (**a**) and low-intensity (**c**) stimuli. In the case of saccades to high-intensity stimuli (**a**), trials have been divided into express (SRTs: 65–95 ms; *left panel*) and regular latency saccades (SRTs > 95 ms; *right panel*). In the case of saccades to low-intensity stimuli (**c**), trials have been divided into those with SRTs < 130 ms (*left panel*) and those with SRTs > 130 ms (*right panel*). **b**, **d** Population functions for all sensory-motor neurons, sorted according to SRT. In order to improve the resolution of these data, these population curves were generated using a spike density functions based on a normal (Gaussian) probability distribution ($\sigma = 4$ ms)

in the retina, both audition and somatosensation employ mechanoreceptors to translate physical energy into a neural signals. As a result, neural responses to these modalities already have extremely short ROLs (e.g., ~40 and ~20 ms in the SC for auditory and tactile stimuli, respectively; Wallace et al. 1996). Therefore, coding changes in aural or tactile intensity as changes in ROL is perhaps not as effective at facilitating changes in SRT as it is for the visual modality. Previous studies have shown that increasing aural or tactile intensity increases the magnitude of the evoked neural activity (e.g., auditory responses in the dSC; Perrault et al. 2003). It is possible, therefore, that variations in sensory response magnitude in a task such as the one used in the current study could influence SRT by providing a greater contribution to the motor output of the dSC. However, there have yet to be systematic studies examining the effect of aural or tactile intensity on the relationship between neural activity and behavior.

Conclusions

Increasing visual stimulus intensity reduces the ROL for activity in the intermediate layers of the SC. As a result, activity related to the saccade response can begin sooner thereby triggering a saccade with shorter latency, leading to the behavioral effect of visual stimulus intensity on SRT.

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