Mutual inhibition and capacity sharing during parallel preparation of serial eye movements

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Many common activities, like reading, scanning scenes, or searching for an inconspicuous item in a cluttered environment, entail serial movements of the eyes that shift the gaze from one object to another. Previous studies have shown that the primate brain is capable of programming sequential saccadic eye movements in parallel. Given that the onset of saccades directed to a target are unpredictable in individual trials, what prevents a saccade during parallel programming from being executed in the direction of the second target before execution of another saccade in the direction of the first target remains unclear. Using a computational model, here we demonstrate that sequential saccades inhibit each other and share the brain's limited processing resources (capacity) so that the planning of a saccade in the direction of the first target always finishes first. In this framework, the latency of a saccade increases linearly with the fraction of capacity allocated to the other saccade in the sequence, and exponentially with the duration of capacity sharing. Our study establishes a link between the dual-task paradigm and the ramp-to-threshold model of response time to identify a physiologically viable mechanism that preserves the serial order of saccades without compromising the speed of performance.

Introduction

Karl Lashley (1951) in his seminal work on the formulation of the serial order in behavior postulated a simultaneous neural representation of all serial elements of an action before the action began. Several models, including a class of neural networks that assume a simultaneous representation of competitive elements of a sequence, have been put forth to account for serial order in different types of behavior like typing, speech, etc. (Botvinick & Plaut, 2004; Cooper & Shallice, 2006; Glasspool & Houghton, 2005; Grossberg, 1978; Jordan, 1997; Rhodes, Bullock, Verwey, Averbeck, & Page, 2004; Sandamirskaya & Schöner, 2010). Neurons in the prefrontal cortex and the frontal eye field indeed exhibit concurrent representation of forthcoming sequential manual movements (Averbeck, Chafee, Crowe, & Georgopoulos, 2002; Barone & Joseph, 1989; Funahashi, Inoue, & Kubota, 1997; Mushiake, Saito, Sakamoto, Itoyama, & Tanji, 2006) and saccadic eye movements (Fujii & Graybiel, 2003; Tian, Schlag, & Schlag-Rey, 2000), respectively. The eyes move in an ordered sequence often during regular activities like reading or scanning natural images (Land, 2009; Morrison, 1984; Rayner, 1998). Nevertheless, no reported attempt has been made to model the serial order of saccades.

Previous studies that posited simultaneous planning of competing (Camalier et al., 2007; Findlay & Walker, 1999; Ludwig, Farrell, Ellis, & Gilchrist, 2009) or consecutive saccades (Dominey & Arbib, 1992; Ludwig, Mildinhall, & Gilchrist, 2007; Van Loon, Hooge, & Van den Berg, 2002) mainly focused on their spatial orientations or latency distributions.

We have examined how the brain preserves the order of successive saccades despite stochasticity in the rate of concurrent planning by modeling behavior of human subjects when they shift the gaze to the locations of two targets in the order of appearance of the targets. Previous studies have reported that the duration of fixation between consecutive saccades may decrease below the average latency of saccade made in isolation to a single target, indicating that the preparation of the second saccade prior to the end of first saccade is feasible (Becker & Jürgens, 1979). Further, the intersaccadic interval (ISI) decreases down to a limit as the second target appears progressively earlier than the onset of saccade to the first target, which suggests that consecutive saccades directed to two visible targets are processed in parallel. The latency of both saccades in a sequence progressively increase as the delay between appearances of the targets decreases from 200 to 50 ms (Ray, Schall, & Murthy, 2004). A similar pattern of increase in the response latency occurs in the dual-task

paradigm as well (Kahneman, 1973; McLeod, 1977; Navon & Gopher, 1979; Pashler, 1994), when two independent tasks are performed in a rapid succession (Tombu & Jolicœur, 2002). The capacity-sharing theory of dual-task interference postulates that the smaller the fraction of capacity allocated to a task, the longer a subject takes to produce the corresponding response (Navon & Miller, 2002; Tombu & Jolicœur, 2003). The response to the first task may even be generated after the response to the second task, if the first task is extremely difficult (Leonhard, Fernández, Ulrich, & Miller, 2011), suggesting that the distribution of limited resources (capacity) of information processing between tasks is critical to preserve the order of responses.

We have designed a model to test our hypothesis that the planning stages of successive saccades share capacity when they overlap in time to ensure that the first saccade is always executed in the direction of the first target and the subsequent saccade in the direction of the second target. In our model, two interactive signals concurrently rise to a threshold at stochastic rates to generate successive saccades. The strength of interaction between processes planning saccades depends on the proportion of capacity allocated to each saccade. Whether successive saccades share capacity has never been tested and the neural mechanism of sharing capacity between tasks is unknown as well. Nonetheless, studies on monkeys that relate the buildup activity of cortical and subcortical neurons to saccade initiation (Dorris, Paré, & Munoz, 1997; Hanes & Schall, 1996; Ipata, Gee, Goldberg, & Bisley, 2006; Tanaka, 2007) and parallel planning of successive saccades (McPeek & Keller, 2002; Murthy et al., 2007; Phillips & Segraves, 2010) partially support the physiological feasibility of this model. Simulation of our model suggests that the sharing of capacity and mutual inhibition between a pair of processes planning saccades preserve the order of saccade execution and account for many latency phenomena observed during sequential saccadic eye movements.

Methods

Experiment

The task, stimuli, and recording techniques used in the experiment have been described in detail elsewhere (Ray et al., 2004). In summary, fourteen healthy human subjects participated in a modified double-step task (Figure 1a). All subjects gave their informed consent in accordance with the Institutional Human Ethics Committee of the National Brain Research Centre, India. In 60% of total trials, called *no-step* trials, a green target appeared after a random fixation period between 300 and 800 ms at one among eight possible locations, which were 45° apart from each other at an eccentricity of 12° visual angle from the central fixation spot. In the remaining trials, called *step* trials, a second target of red color appeared at a randomly chosen location non-adjacent to the green target on the imaginary circular array. These targets were identical in size, shape, and luminance and remained on the screen until the end of the trial. The delay between the appearances of the targets (commonly called the stimulus onset asynchrony or SOA) was randomly chosen from 50, 100, 150, and 200 ms. Subjects were instructed to shift gaze to the location of the green target in no-step trials and generate successive saccades rapidly to the location of the green and red targets in the order they appeared in step trials. No-step and step trials were randomly interleaved. To test the stability of our model, we examined two additional subjects who were not explicitly instructed to generate saccades "as quickly as possible," unlike other fourteen subjects. Eye movements were recorded using an infrared eye tracker (ISCAN, MA, USA) at 200-Hz sampling rate. TEMPO/VIDEOSYNC (Reflective Computing, MO, USA) software generated stimulus and collected and stored the sampled eye positions in every trial. The beginning and end of each saccade were demarcated offline when the eye velocity crossed a 30°/s threshold. The latency of a saccade was calculated from the delay between the appearance of a target and the onset of the saccade directed to the target. Trials that produced blink-perturbed saccades were not considered for subsequent analyses.

Capacity sharing by sequential saccades

Tombu and Jolicœur (2003) previously established a relationship between response time and SOA in the dual-task paradigm based on the assumption that the rate of processing a response is linearly proportional to the fraction of capacity allocated to the corresponding task. Following their basic assumptions, we derived equations for the latency of saccades directed to the first (RT1′) and second (RT2′) targets (see Appendix A):

$$RT1' = \left(\frac{SP-1}{SP}\right) \times SOA + \left(\frac{SP-1}{SP}\right) \times A2 + \frac{A1+B1}{SP},$$

$$\text{(1)}$$

$$RT2' = A1 + B1 + B2 - SOA. \tag{2}$$
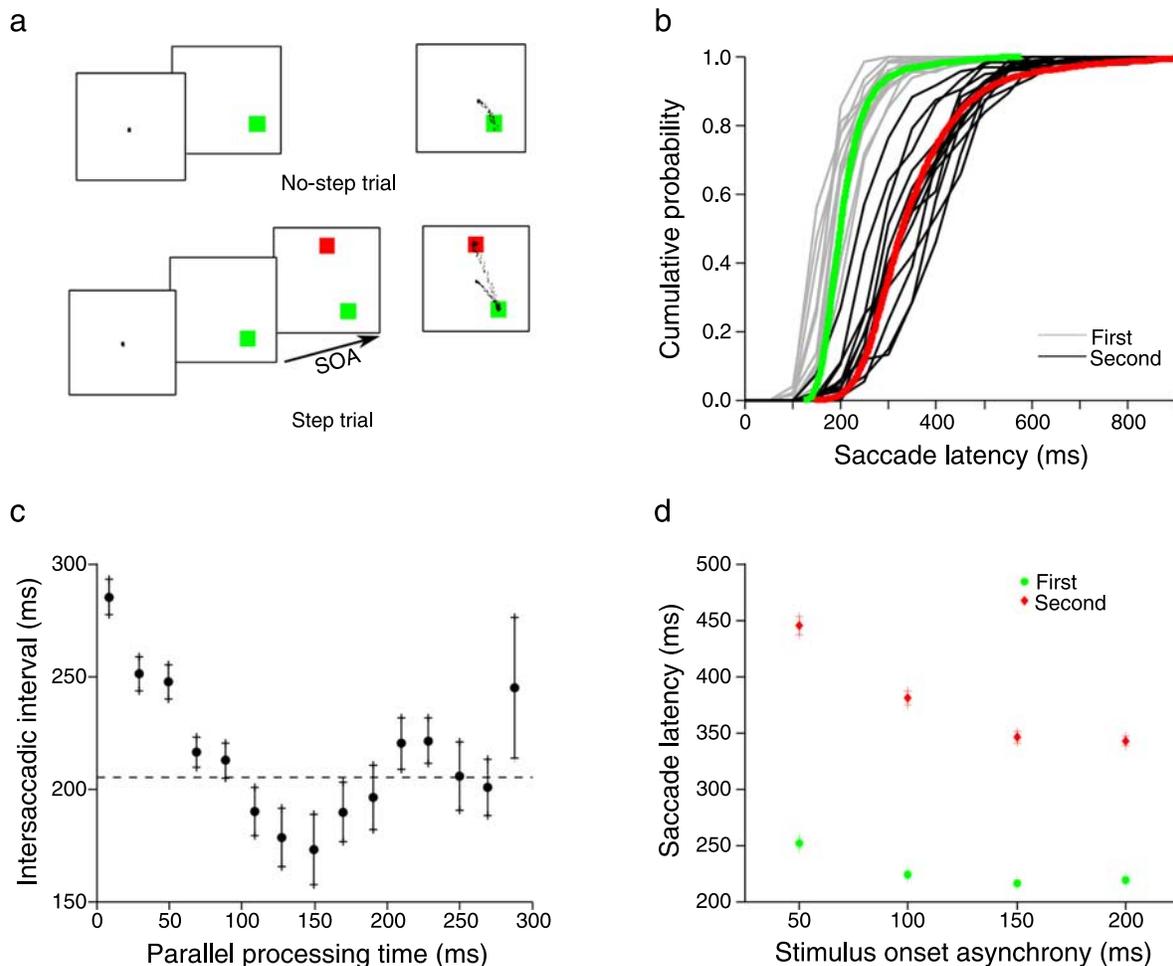
Figure 1. (a) Schematic of the temporal sequence of stimuli and behavior in the task. In *no-step* trials, subjects made saccades to a singleton green target. In *step* trials, a second red target appeared after SOA. Subjects directed their gaze from the fixation spot to the first target and, subsequently, to the second target. (b) Latency distributions of saccades directed to the first (gray) and second (black) targets for individual subjects (thin lines) and across the population of subjects (thick lines; first: green, second: red). (c) Plot of mean intersaccadic interval (ISI) against parallel processing time (PPT) across subjects. Horizontal broken line shows the mean saccade latency in no-step trials. (d) Average latencies of saccades directed to the first (circle) and second (diamond) targets are contrasted at different SOAs. Error bars in (c) and (d) show the standard error of the mean ISIs and saccade latencies, respectively.

In Equations 1 and 2, A1 (A2) and B1 (B2) refer to the duration of the visual processing stage and planning stage of a saccade directed to the first (second) target, respectively, when saccades are generated in isolation or do not share capacity (Figure 2a). The delay between onsets of the targets is SOA. The sharing proportion (0 < SP < 1) is the fraction of capacity allocated for planning a saccade in the direction of the first target. The remaining capacity (1 − SP) is assumed to be allocated for planning another saccade in the direction of the second target.

We modified Equations 1 and 2 such that the sharing proportion (SP) from saccade latency can be derived without explicit knowledge of the duration of planning stages (B1, B2). Suppose that the duration of capacity sharing between planning stages of saccades is D (e.g.,

D_1/D_2 in Figure 2b). In Equation 1, substituting SOA by $(RT1' − A2 − D)$ and $(A1 + B1)$ by RT1, we get

$$RT1' = \left(\frac{SP-1}{SP}\right) \times (RT1' - A2 - D) + \left(\frac{SP-1}{SP}\right)$$
$$\times A2 + \frac{RT1}{SP},$$
$$RT1' = \left(\frac{SP-1}{SP}\right) \times (RT1' - D) + \frac{RT1}{SP},$$
$$\frac{RT1'}{SP} = \left(\frac{1-SP}{SP}\right) \times D + \frac{RT1}{SP},$$
$$RT1' = (1 - SP) \times D + RT1. \tag{3}$$
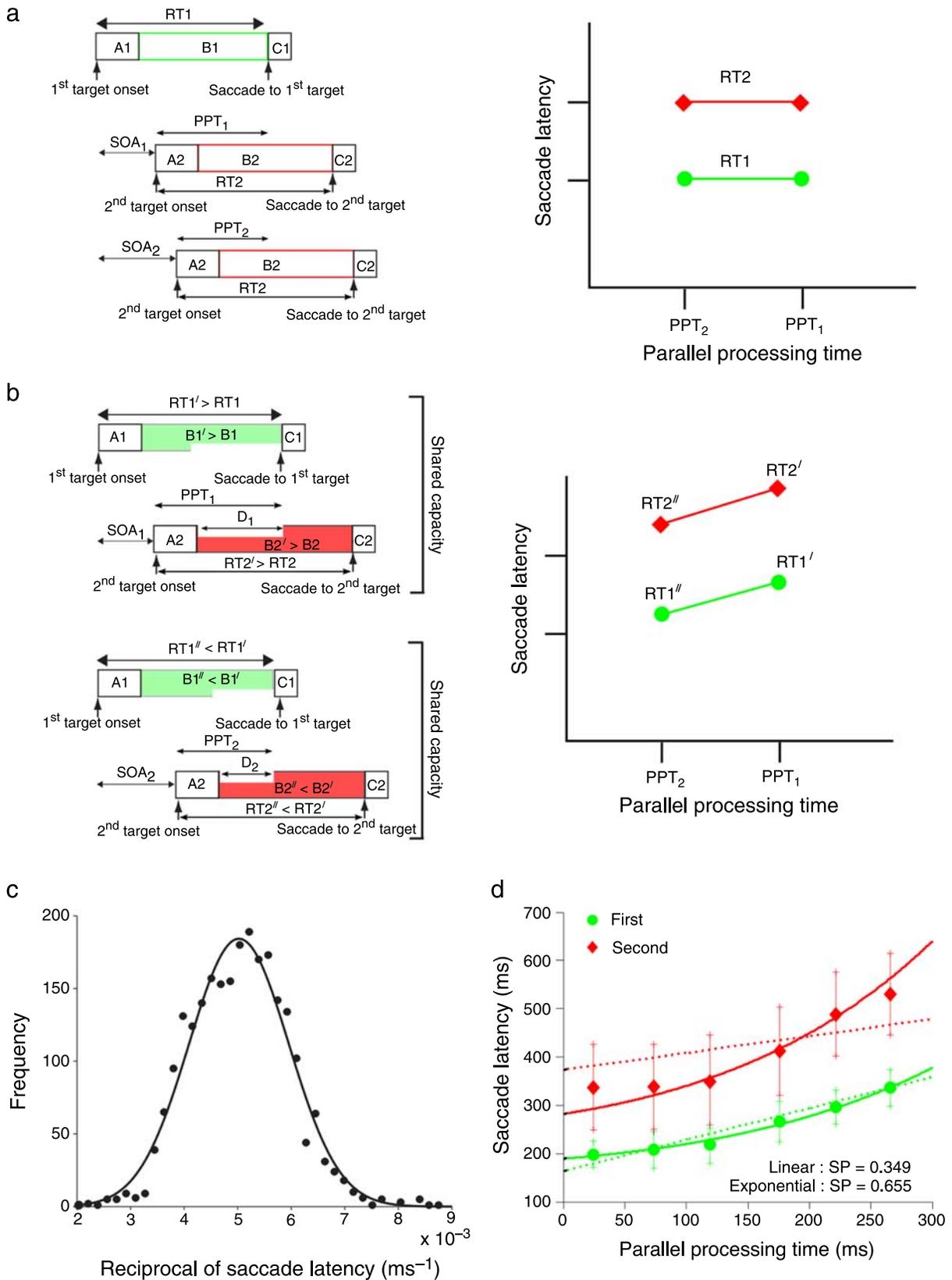
Similarly in Equation 2, substituting SOA by $RT1' - A2 - D$ and $(A2 + B2)$ by RT2, we get

$$RT2' = RT1 + B2 - RT1' + A2 + D,$$

$$RT2' = (RT1 - RT1') + (A2 + B2) + D,$$

$$RT2' = -(1 - SP) \times D + RT2 + D,$$

$$RT2' = SP \times D + RT2. \qquad (4)$$

Note that according to Equations 3 and 4, the latency of a saccade increases linearly with the fraction of capacity allocated to the other saccade in a sequence and the duration ($D > 0$) of capacity sharing between saccades.

Interactive ramp-to-threshold model of sequential saccades

Model design

The empirical data show that the latency of saccades directed to the first and second targets increased

Figure 2. (a) A model of parallel processing of sequential saccades in which capacity is unlimited (left). The duration of sensory, planning, and execution stages of a saccade directed to the first (second) target is A1 (A2), B1 (B2), and C1 (C2), respectively. The latencies of saccades directed to the first (RT1) and second (RT2) targets remain constant when the parallel processing time decreases from PPT_1 to PPT_2 as the delay between onsets of targets increases from SOA_1 to SOA_2 (right). (b) A capacity-sharing model of sequential saccades in which planning stages share capacity when they overlap in time (left). The duration of the planning stage of a saccade directed to the first target increases from B1 to B1′ (top, green) and from B1 to B1″ (bottom, red), and that of a saccade directed to the second target increases from B2 to B2′ (top, green) and from B2 to B2″ (bottom, red), when the delay between onsets of the targets increases from SOA_1 to SOA_2. Because the duration (D) of capacity sharing decreases as the stimulus onset asynchrony (SOA) increases, this model predicts that the latency of saccades directed to the first and second targets will also decrease with decreasing parallel processing time (left). (c) A Gaussian fit of the histogram of the reciprocal of saccade latency in correct no-step trials to estimate the average ($\pm SD$) processing rate of saccades. (d) The mean latency of saccades directed to the first (circle) and second (diamond) targets in correct step trials increased with the mean parallel processing time (PPT). The sharing proportion (SP = 0.655) was estimated from an exponential (solid green) fit of the latency of saccades directed to the first target. In contrast, a linear (dotted green) relationship between saccade latency and PPT as predicted by the classical capacity-sharing theory estimated SP = 0.349. Exponential (solid red) and linear (dotted red) fits of the latency of saccades directed to the second target are contrasted. Error bars show the standard deviations of the mean saccade latencies.

exponentially with the duration of overlap between processing stages of consecutive saccades in contradiction to the prediction by the linear capacity-sharing model (Figure 2d). The data obeyed the following equations:

$$RT1' = \left\{ e^{r \times (PPT - A2)} \right\} \times (1 - SP) \times (PPT - A2) + RT1, \quad (5)$$

and

$$RT2' = \left\{ e^{r \times (PPT - A2)} \right\} \times SP \times (PPT - A2) + RT2, \quad (6)$$

where PPT (*parallel processing time*) is the time of onset of saccade to the first target relative to the time of appearance of the second target (e.g., PPT_1/PPT_2 in Figure 2b), and r (>0) is a constant. Substituting (PPT − A2) by D in the above equations, we get

$$RT1' = \left(e^{r \times D} \right) \times (1 - SP) \times D + RT1, \qquad (7)$$

$$RT2' = \left(e^{r \times D} \right) \times SP \times D + RT2. \qquad (8)$$

Equations 7 and 8 differ from Equations 3 and 4 by an additional exponential ($e^{r \times D}$) term on the right-hand side of each equation.

We modeled sequential saccades to understand the mechanism underlying exponential increase of latency with the duration of capacity sharing. We extended the central idea of the ramp-to-threshold models of saccadic reaction time. This class of models assumes that a decision signal rises to a threshold at a stochastic rate to generate a saccade (Brown & Heathcote, 2008; Carpenter, 1981; Laming, 1968; Luce, 1986; Nakahara, Nakamura, & Hikosaka, 2006; Purcell et al., 2010; Smith, 1995). In our model, two decision signals simultaneously rise at stochastic rates to elicit saccades in respective directions upon arriving at a threshold. From a physiological perspective, a ramp-to-threshold signal may reflect an accrual of information over time by means of recurrent self-excitation of a neural computing unit. Models that incorporate a recurrent self-excitation mechanism sample and accumulate evidence in favor of alternative choices and provide a common framework to understand motor planning and decision making. The resulting accumulated information reliably mimics the time-varying neural activity of non-human primates performing decision-making tasks (Lo & Wang, 2006; Mazurek, Roitman, Ditterich, & Shadlen, 2003; reviewed in Gold & Shadlen, 2007).

In the absence of an interaction, sequential saccade planning can be modeled by a pair of decision signals, each rising from the baseline (= 0) to a threshold (= 1) to initiate a saccade in the direction of the corresponding target. When these signals overlap in time, their ratio may provide a means to calculate how likely the first saccade is

going to be directed to the first target. Suppose that u_1 and u_2 are the signals corresponding to saccades in the direction of the first and second targets, respectively (Figure 3, left). The rate of increase of u_1 and u_2 remain fixed in a trial but vary across trials. In light of previous

findings that while the likelihood ratio merely provides an instantaneous evidence in support of a hypothesis between alternatives (Carpenter & Williams, 1995), the log-likelihood ratio serves as a natural currency for making a decision (Bogacz, Usher, Zhang, & McClelland, 2007;
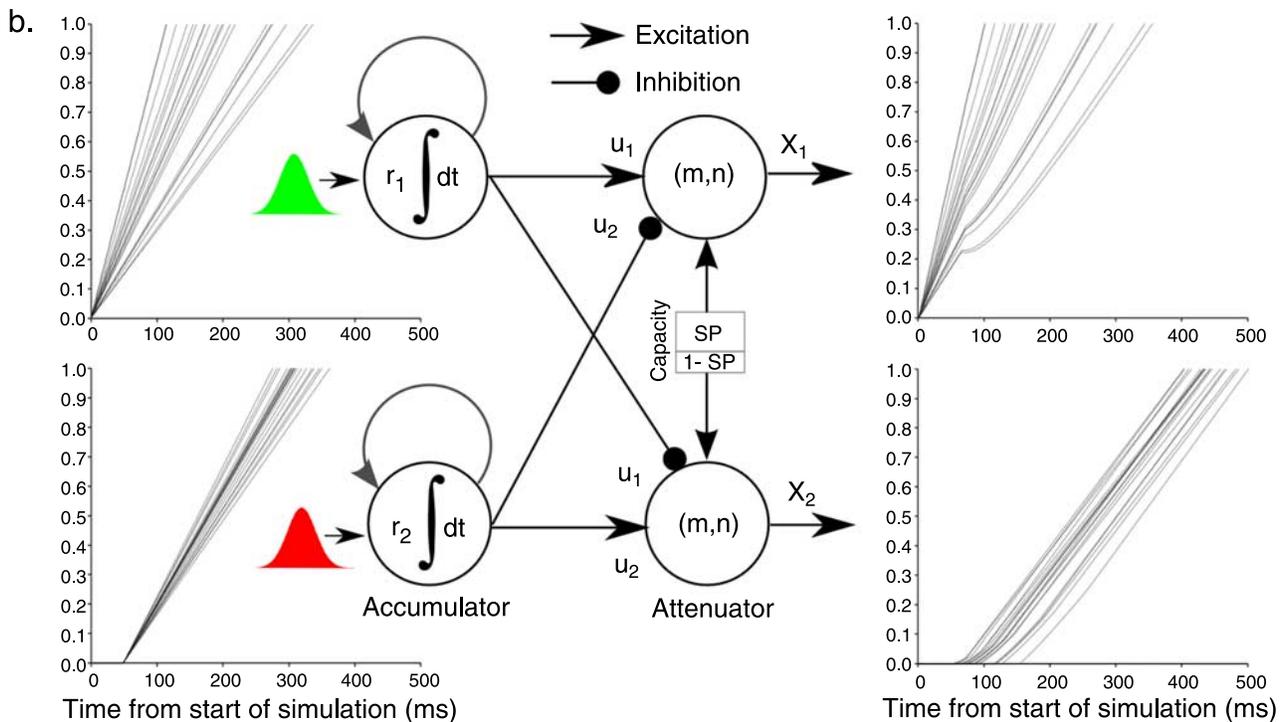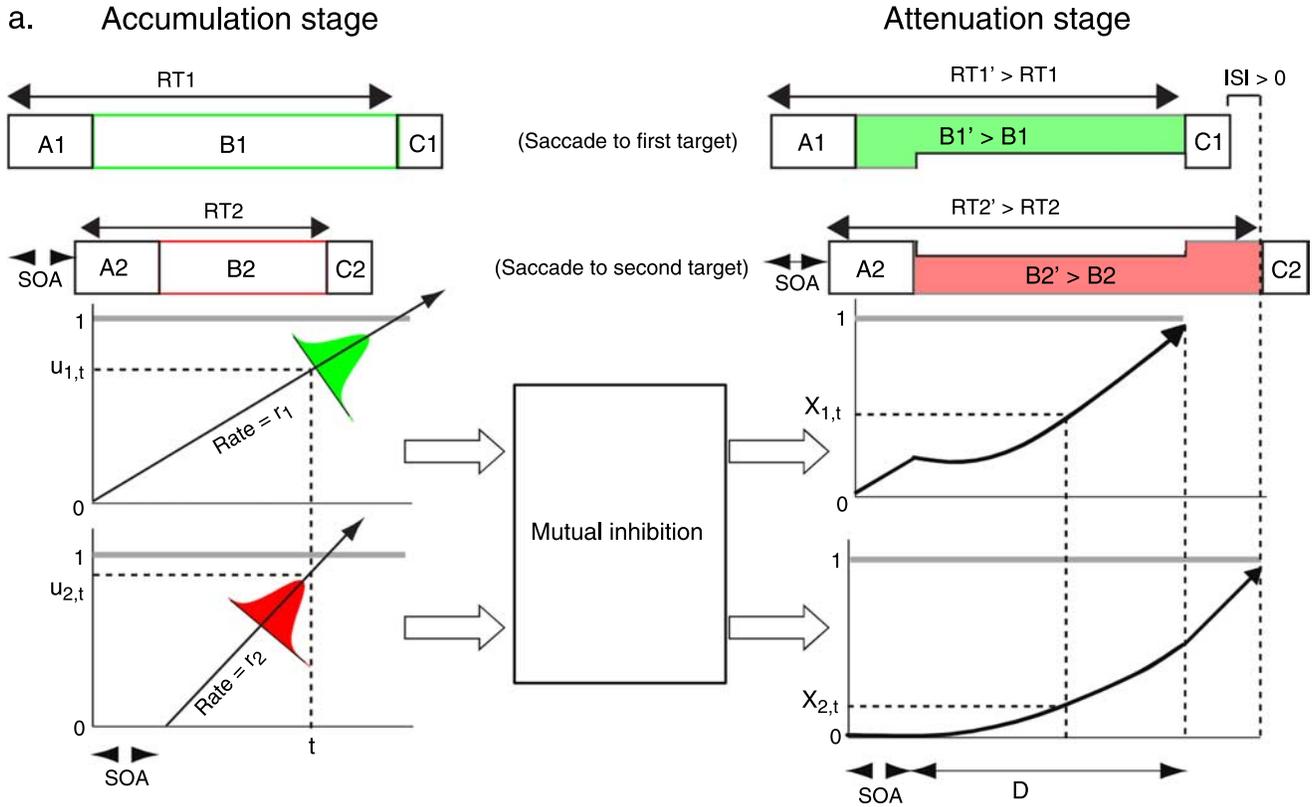
Wald & Wolfowitz, 1948), we defined a *Confidence Index* (CI) as $\log\left(\frac{u_1}{u_2}\right)$ to quantify the level of confidence with which one can predict the direction of the first saccade. When u_1, $u_2 > 0$, the first saccade is more likely to be directed to the first target if $\frac{u_1}{u_2} > 1$ or CI > 0. On the other hand, $\frac{u_1}{u_2} < 1$ or CI < 0 implies that the saccade aimed at the second target is more likely to be executed first. The direction of the first saccade is completely ambiguous when CI $= 0$ or, stated differently, when decision signals for saccades are identical (i.e., $u_1 = u_2$).

In addition, we defined the *Degree of Concurrency* (DoC) as the duration of overlap between the planning stages of consecutive saccades in a step trial divided by the average duration of saccade planning in no-step trials, i.e., DoC $= \frac{D}{B1}$. The maximum value DoC can achieve is 1 when $D = B1$, and DoC ≤ 0 implies serial processing of saccades. Replacing D by $\left\{\frac{B1-(SOA+A2-A1)}{SP}\right\}$ (see Appendix A), we get DoC $= \left[\left\{\frac{B1-(SOA+A2-A1)}{SP}\right\} \div B1\right]$. Because A1, A2, B1, and SP are assumed constant, the degree of concurrent preparation of saccades increases as SOA decreases. In contrast, the possibility of the first saccade being elicited in the direction of the first target decreases as SOA decreases, due to the stochastic rate of saccade planning. This implies DoC $\propto \frac{1}{CI}$. Replacing DoC by $\frac{D}{B1}$ and CI by $\log\left(\frac{u_1}{u_2}\right)$, we get $\frac{D}{B1} = -n \times \log\left(\frac{u_1}{u_2}\right)$, where n (>0) is a proportionality constant. We assumed that r_1 is the rate of saccade planning to a singleton target in a no-step trial. Because no-step and step trials were randomly interleaved, subjects were expected to start planning a saccade to the first target at the same rate (r_1) in the step

Figure 3. (a) Schematic of the capacity-sharing model (above) and an equivalent interactive ramp-to-threshold model (below) of parallel preparation of sequential saccades directed to two targets that appeared in succession after SOA. A*n*, B*n*, and C*n* refer to the duration of visual processing, planning, and execution stages, respectively, of the *n*th saccade in the sequence; $n = 1, 2$. In the absence of capacity sharing, the latency of the *n*th saccade RT*n* = A*n* + B*n*. When planning stages of saccades share capacity, B*n* increases to B*n*′, giving rise to saccade latency RT*n*′ = A*n* + B*n*′. In the equivalent ramp-to-threshold model of saccade, B*n* = $1/r_n$, where r_n is the growth rate of activity of a process planning a saccade in the direction of *n*th target. In our model, r_n remains fixed in a trial but varies normally across the trials. In the attenuation stage, processes that plan saccades inhibit each other during the overlap period (D) to maintain the order of consecutive saccades (ISI > 0). (b) Sample simulation of 20 step trials in Simulink. In the model, two integrators independently accumulate information at constant rates r_1 and r_2 sampled from normal distributions. Each of the two attenuators receives output signals u_1 and u_2 from the accumulators and inhibit them to X_1 and X_2, respectively, which trigger sequential saccades upon arriving at a threshold activity ($= 1$).

trial unaware that a second target would be forthcoming. Replacing the duration (B1) of saccade planning in the no-step trial by $\left(\frac{1}{r_1}\right)$, we get

$$r_1 \times D = -n \times \log\left(\frac{u_1}{u_2}\right) \text{ or } e^{r_1 \times D} = \left(\frac{u_2}{u_1}\right)^n. \quad (9)$$

We measured the coefficient r ($= 0.0033$ ms^{-1}) in Equations 7 and 8 from the fit (Figure 2d). Because $\frac{1}{r}$ ($= 300$ ms) is of the order of a visually guided saccade latency in ordinary conditions, we speculate that r is related to the rate (r_1) of planning the saccade in isolation (no-step trial). Hence e$^{r \times D} \propto$ e$^{r_1 \times D}$. Substituting e$^{r \times D}$ in Equations 7 and 8 by ($m \times$ e$^{r_1 \times D}$) or $\left\{m \times \left(\frac{u_2}{u_1}\right)^n\right\}$, where m (>0) is a proportionality constant, we get

$$RT1' = m \times \left(\frac{u_2}{u_1}\right)^n \times (1 - SP) \times D + RT1, \quad (10)$$

$$RT2' = m \times \left(\frac{u_2}{u_1}\right)^n \times SP \times D + RT2. \quad (11)$$

Equations 10 and 11 establish a link between the capacity-sharing model and the linear ramp-to-threshold model of sequential saccade latency. Net increases in the latency of saccades directed to the first and second targets due to capacity sharing for duration D are

$$\Delta RT1' = RT1' - RT1 = m \times \left(\frac{u_2}{u_1}\right)^n \times (1 - SP) \times D, \quad (12)$$

and

$$\Delta RT2' = RT2' - RT2 = m \times \left(\frac{u_2}{u_1}\right)^n \times SP \times D. \quad (13)$$

Therefore, with every unit increase in D, the respective saccade latency increases by

$$\Delta RT1' = m \times \left(\frac{u_2}{u_1}\right)^n \times (1 - SP), \quad (14)$$

and

$$\Delta RT2' = m \times \left(\frac{u_2}{u_1}\right)^n \times SP. \quad (15)$$

Given that the proportion of allocated capacity (i.e., SP) is fixed in a trial, Equations 14 and 15 suggest that the ratio of magnitudes of corresponding decision signals is

critical to ensure $(\text{SOA} + \text{RT2}') > \text{RT1}'$, which is a must to maintain the order of saccades in a sequence. Therefore, attenuation of one signal should take the present state of the other signal into account to prevent ordering error. This may be perceived as a mutual inhibition between consecutive saccades. We hypothesized that the magnitude of a pair of signals simultaneously planning saccades in the direction of the first (u_1) and second (u_2) targets is reduced at every time step by the amount given on the right-hand side of Equations 14 and 15, respectively. When the rate of accumulation of information to generate a saccade to the second target is considerably higher than that to the first target, exertion of an inhibitory control may be a means to prevent the signal rising to generate the second saccade from reaching threshold before the first saccade is generated. The purpose of such inhibitory control is to dampen the ascent of the signal thereby elongating the planning stage of the saccade to the second target. This will cause the duration (D) of capacity sharing to increase, which in turn will increase the latency of the saccade to the first target (RT1′). Such an increase in RT1′ will demand further slowdown of planning the saccade to the second target, and the interaction between planning stages will continue until the signal rising to generate a saccade to the first target reaches the threshold. To test the idea that both mutual inhibition and capacity sharing are critical to preserve the order of execution of consecutive saccades, we designed a model using Matlab Simulink software with an embedded S-Function written in C language, which consists of two processing stages, an accumulation stage and an attenuation stage (Figure 3).

Model description

We made the following key assumptions to construct the model: (1) the appearance of a target activates an accumulator after 70 ms, which is about the average delay of visual signals in the FEF (Buschman & Miller, 2007; Pouget, Emeric, Stuphorn, Reis, & Schall, 2005; Schmolesky et al., 1998); (2) the duration of visual processing of each target is the same; (3) in every trial, activity in the accumulators at time t are denoted by $u_{1,t}\{= (r_1 \times t)\}$, and $u_{2,t}\{= r_2 \times (t - \text{SOA})\}$, where r_1 and r_2 are independently sampled from two sets of normally distributed numbers with means that correspond to the mean rate of planning saccades to the first and second targets; (4) the variance of the rate of planning saccades in the direction of two targets are the same. (5) Each of the two attenuators at the next stage receives signals $u_{1,t}$ and $u_{2,t}$ from both accumulators at time $t + 1$, where these input signals inhibit each other and generate output signals $X_{1,t+1}$ and $X_{2,t+1}$, respectively. In the absence of any interaction, the output from an attenuator merely reflects the activity of its corresponding accumulator. (7) The strength of inhibition exerted by each decision signal on another depends on the proportion of capacity they

share. (8) Because the firing rate of a neuron cannot be negative, our model prevents X_1 and X_2 from falling below zero. (9) The interaction continues until either X_1 or X_2 reaches the threshold normalized to 1 to elicit a saccade in the direction of the corresponding target. During simulation of the model, $X_{1,t}$ and $X_{2,t}$ were computed as

$X_{1,0} = X_{2,0} = 0$ and

$$
\left.
\begin{aligned}
X_{1,t+1} &= u_{1,t} && \text{if } u_{2,t} = 0 \text{ or } X_{2,t} = 1 \\
&= u_{1,t} - m \times (1 - \text{SP}) \times \left(\frac{u_{2,t}}{u_{1,t}}\right)^n && \text{if } u_{1,t}, u_{2,t} > 0 \text{ and } X_{1,t}, X_{2,t} < 1 \\
&= 0 && \text{if } X_{1,t+1} < 0
\end{aligned}
\right\},
$$
(16)

$$
\left.
\begin{aligned}
X_{2,t+1} &= u_{2,t} - m \times \text{SP} \times \left(\frac{u_{2,t}}{u_{1,t}}\right)^n && \text{if } u_{1,t}, u_{2,t} > 0 \text{ and } X_{1,t}, X_{2,t} < 1 \\
&= u_{2,t} && \text{if } X_{1,t} = 1 \\
&= 0 && \text{if } X_{2,t+1} < 0
\end{aligned}
\right\}.
$$
(17)

Finding parameters of the model

We collated data from all correct trials across the population of fourteen subjects to find the value of two free parameters (m and n) of the model that are independent of any idiosyncratic behavior of individual subjects. No-step trials that yielded identical ($\pm 10^{-4}$ ms^{-1}) reciprocals of saccade latencies were grouped together to estimate the mean (μ) and standard deviation (σ) of rate of saccade processing (visual processing and motor planning) from a fit by a Gaussian function of the form $f(x) = \frac{1}{\sqrt{2\pi\sigma^2}} e^{-\left(\frac{x-\mu}{\sqrt{2}\sigma}\right)^2}$, where μ (= 0.005) and σ (= 0.00095) were the coefficients of the fit (Figure 2c). In this analysis, we did not include trials that produced saccades of latency shorter than 100 ms. The goodness of fit in terms of R^2 (= 0.97), which was close to the maximum (= 1.0), suggested that the Linear Approach to Threshold with Ergodic Rate (LATER) model that assumes a Gaussian distribution of the rate of saccade processing can emulate saccade latencies in no-step trials (Reddi & Carpenter, 2000). A two-sample Kolmogorov–Smirnov test showed that the distribution of the reciprocal of saccade latency in 2404 correct no-step trials was indifferent ($P = 0.178$) from that of equal number of random samples from a normal distribution with mean ($\pm SD$) of (0.005 $\pm$ 0.00095) ms^{-1}.

Two free parameters of the model, m (= 17.62) and n (= 4) were optimized by minimizing the difference between expected saccade latencies and simulated saccade latencies in 1000 step trials at SOA = 50 ms, using "Least Square Method" in "Parameter Estimation" tool of Simulink (The Mathworks) software. We derived the

expected latencies of saccades directed to the first and second targets using SP (= 0.655), r (= 0.0033 ms^{-1}), RT1 (= 208 ms), and RT2 (= 318 ms), as obtained from exponential fits (Figure 2d), and D $\left(= \frac{1}{r_1} - \text{SOA}\right)$ in Equations 7 and 8, respectively. Since the duration of visual processing of both targets were assumed the same (i.e., A1 = A2), the duration (D) of overlap between planning stages of consecutive saccades was equal to the difference between the duration of planning a saccade to the first target and SOA. The rate (r_1) of planning a saccade to the first target varied from trial to trial and was calculated, first, by subtracting 70 ms from the expected duration of saccade processing, then calculating the reciprocal of the difference. The reciprocal of a value sampled from a normal distribution with mean (= 0.005 ms^{-1}) and standard deviation (= 0.00095 ms^{-1}) provided the expected duration of saccade processing. The same value of r_1 was used in the model as the rate of increase of u_1. The rate (r_2) of increase of u_2 in each simulated step trial was sampled from a distribution of mean (= $\frac{1}{\text{RT2}-\text{A2}}$ = $\frac{1}{318-70}$ ms^{-1}) = 0.004 ms^{-1} and standard deviation = 0.00095 ms^{-1}.

Simulation of the model

A total of 1000 step trials for each of the four SOAs (50, 100, 150, and 200 ms) were simulated in a discrete time step of 1 ms for 1 s. Values of parameters used in the model are summarized in Table 1. Simulation of a step trial began at $t = 0$ when an integrator started accumulating information. Another integrator started accumulating information after SOA. A pair of attenuators individually received output from both integrators and attenuated them. When the output signals of attenuators reached the threshold, the simulator registered the onset of saccades in the corresponding directions. The interval between the time when an integrator started accumulation and the corresponding attenuated signal reached the threshold was considered as the duration of the planning stage of a saccade. We calculated saccade latency by adding a visual processing delay of 70 ms to the duration of the planning stage and an additional 50 ms as the duration of saccade execution to calculate the time of saccade end.

Results

Summary of experimental data

Figures 1b–1d summarize the saccadic behavior of human subjects in a modified double-step task (see Ray et al., 2004 for details). Despite idiosyncrasies in the latency distributions of sequential saccades [mean ($\pm SD$) skewness, first: 1.4 ± 0.68; second: 0.83 ± 0.87] as shown in Figure 1b (thin lines), two trends were observed across the population of subjects. First, the duration of fixation between consecutive saccades or intersaccadic interval (ISI) decreased with increasing parallel processing time (PPT) or the duration of overlap between processing stages of saccades [mean ($\pm SD$) linear regression slope = -0.29 (± 0.23), $P < 0.05$]. Second, the latency of consecutive saccades increased as the stimulus onset asynchrony (SOA) decreased [mean ($\pm SD$) linear regression slope, first: -0.15 ± 0.21; second: -0.59 ± 0.36; $P < 0.05$]. In the aggregated data across subjects, a decrease in ISI down to a limit at longer PPTs (Figure 1c) and an increase in saccade latency with decreasing SOA (Figure 1d) indicated a capacity-limited mechanism of saccade programming.

Capacity-sharing account of behavior

How the distribution of capacity may affect latencies of consecutive saccades have been contrasted schematically in two hypothetical cases: In one model, consecutive saccades do not share capacity during parallel processing (Figure 2a), and in the other, consecutive saccades share capacity when their planning stages overlap in time

Parameter	Value
μ_1 (mean 1st saccade processing rate)	0.005 ms^{-1}
σ (standard deviation of 1st saccade processing rate)	0.00095 ms^{-1}
r_1 (rate of 1st saccade planning)	$r_1 = \frac{1}{\frac{1}{R}-70}$, R was sampled from a normal distribution with mean (= μ_1) and standard deviation (= σ)
μ_2 (mean rate of 2nd saccade planning)	0.004 ms^{-1}
r_2 (rate of 2nd saccade planning)	Sampled from a normal distribution with mean (= μ_2) and standard deviation (= σ)
SP (sharing proportion)	0.655
m	17.62
n	4

Table 1. List of parameters used in the model for simulation.

(Figure 2b). Suppose that the latency of a saccade directed to the first target is RT1 (= A1 + B1) and that to the second target is RT2 (= A2 + B2). The symbols A1 and A2 refer to the duration of visual processing of the first and second targets, respectively; B1 and B2 refer to the duration of planning saccades in the direction of the first and second targets, respectively. When the second target appears after SOA_2 ($>SOA_1$), processing stages of saccades overlap for a relatively shorter period of time ($PPT_2 < PPT_1$). Because the ongoing preparation of a saccade does not prevent preparation of the subsequent saccade when each saccade in a sequence uses independent pool of resources (Figure 2a, left), latencies of consecutive saccades should remain invariant across all parallel processing times (PPTs) as shown in Figure 2a (right). In the capacity-limited condition, planning stages of consecutive saccades share capacity for an interval of time denoted by D_1, when the second target appears after SOA_1 (Figure 2b, top). As a result, B1 increases to B1′, and B2 increases to B2′. Accordingly, RT1 increases to RT1′ (= A1 + B1′) and RT2 increases to RT2′ (= A2 + B2′). When the second target appears after SOA_2 ($>SOA_1$), the processing stages of saccades overlap for a relatively shorter period of time ($PPT_2 < PPT_1$), and capacity is shared for a shorter duration ($D_2 < D_1$; Figure 2b, bottom). As a result, RT1 increases to RT1″ (= A1 + B1″) and RT2 increases to RT2″ (= A2 + B2″), where RT1 < RT1″ < RT1′ and RT2 < RT2″ < RT2′. The capacity-limited model predicts that the latency of saccades directed to the first and second targets will increase with PPT (Figure 2b, right).

The experimental data show that the reciprocal of the first saccade latency in both no-step and step trials are less outlier-prone from a normal distribution, in comparison to the reciprocal of the second saccade latency [Figures 1b and 2c; kurtosis: first (no step) = 4.0, first (step) = 3.2, second (step) = 9.5], similar to what Van Loon et al. (2002) previously observed. The first (green) and second (red) saccade latencies both increased with PPT (Figure 2d), as predicted by the capacity-sharing model. To test whether a capacity-sharing model can account for distributions of saccade latencies in step trials, we measured PPT from the time of onset of saccade directed to the first target relative to the time of appearance of the second target. Step trials in which the processing stages of saccades did not overlap (i.e., PPT < 0) were not considered for subsequent analyses, and the rest were divided into groups so that trials in each group yielded identical (±25 ms) PPT. Figure 2d shows the plot of the mean latency of saccades directed to the first (RT1′) and second (RT2′) targets against the mean PPT for each group of trials. The reciprocal of the standard deviation of the corresponding mean saccade latency weighted every data point on the plots. To fit the data, we replaced D in Equations 3 and 4 by (PPT − A2), where we considered an average visual delay of 70 ms in the frontal eye field as the duration of visual processing (A2) of the second target. From the linear fit ($R^2 = 0.94$; green dotted line) of the data by

RT1′ = (1 − SP) × (PPT − A2) + RT1, we obtained RT1 = 209.1 ms and SP = 0.349. To account for the non-linearity in the plot, we also fitted RT1′ by RT1′ = $\{e^{r \times (PPT − A2)}\}$ × (1 − SP) × (PPT − A2) + RT1, where $r > 0$. From the exponential fit ($R^2 = 0.99$; green solid line), we obtained RT1 = 208.5 ms, SP = 0.655, and r = 0.0033 ms^{-1}. Note that in correct no-step trials, the mean (±SD) saccade latency across the population of subjects was 205 (±40) ms. Subsequently, we fitted the plot of RT2′ versus PPT by RT2′ = SP × (PPT − A2) + RT2, using SP (= 0.349) as obtained from the linear fit of RT1′, and by RT2′ = $\{e^{r \times (PPT − A2)}\}$ × SP × (PPT − A2) + RT2, using SP (= 0.655) and r (= 0.0033 ms^{-1}) as obtained from the exponential fit of RT1′. We obtained RT2 = 398 ms and 318.4 ms from the linear (red dotted line) and exponential (red solid line) fits, respectively. The exponential function fitted the data better than the linear function (R^2: linear = 0.52, exponential = 0.93) and closely approximated the average (±SD) latency (328 ± 95 ms) of saccades to the second target that appeared maximum 70 ms prior to the onset of the first saccades (i.e., $0 < PPT \leq 70$).

Mutual inhibition model of sequential saccades

We speculated that the non-linear increase in latencies of consecutive saccades with the duration of capacity sharing was due to interactions between neural processes that planned the saccades. We examined whether a pair of independent and non-interacting integrators, each of which accumulating evidence at a constant rate, can simulate sequential saccades in the right order. Successive appearances of the targets triggered these integrators to generate two decision signals that linearly increased from a baseline (= 0) to initiate sequential saccades upon arriving at a fixed threshold (= 1). In a simulated step trial, the growth rate of each of these signals was sampled from a normal distribution with mean (±SD) of 0.005 (±0.00095) ms^{-1}. Simulation of 2000 step trials at the stimulus onset asynchrony (SOA) of 50 and 200 ms each showed that saccades targeted to the second target were executed first in 17.35% and 9% trials, respectively.

Based on the observation that, on the one hand, the classical capacity-sharing model is inadequate to account for the latency distributions of consecutive saccades, while, on the other hand, an independent ramp-to-threshold model is inadequate to account for the order of consecutive saccades, we designed a model in which a pair of processes that progressively raised activity to a threshold to generate consecutive saccades shared capacity and inhibited each other (Figure 3). In this model, a pair of integrators generated decision signals that increased from a baseline (= 0). Each of these integrators independently accumulated information to generate the decision signal at a rate that varied from trial to trial but remained constant in a

trial. Signals from both integrators were fed into each of the two attenuators where they inhibited each other with strength that depended on the proportion of capacity they shared and the ratio of information accumulated by individual integrators up to that point of time. The time-varying outputs from attenuators initiated consecutive saccades in the direction of corresponding targets, upon arriving at a fixed threshold (= 1).

Validation of the model

Simulated and observed data are compared in Figures 4a–4c to show that capacity sharing and mutual inhibition between saccades are critical for (1) the preservation of the serial order of saccades, (2) a progressive increase in the latency of consecutive saccades with decreasing stimulus onset asynchrony (SOA), and (3) a gradual decrease in the intersaccadic interval (ISI) down to a limit with increasing parallel processing time (PPT). Distributions of the simulated and observed saccade latencies are compared in Figure 4a. The mean (±SD) simulated latency (first: 213 ± 58 ms; second: 373 ± 112 ms) was not different from the corresponding mean (±SD) observed latency (first: 225 ± 65 ms; second: 371 ± 112 ms). On average (±SD), a total of 336 (±64) correct step trials contributed data to calculate the mean saccade latency at each SOA. The mean (±SD) observed latency of the first and second saccades significantly ($P < 0.001$) increased from 219 (±63) ms to 252 (±71) ms and from 342 (±90) ms to 445 (±121) ms, respectively, as SOA decreased from 200 to 50 ms. Similarly, the mean (±SD) simulated latency of the first and second saccades significantly ($P < 0.001$) increased from 207 (±41) ms to 232 (±82) ms and from 343 (±76) ms to 438 (±147) ms, respectively, as SOA decreased from 200 to 50 ms (Figure 4b). Step trials with identical (±10 ms) PPTs were grouped together to plot the mean ISI against the mean PPT for each bin spanning 20 ms (Figure 4c). Each data point was weighted by the reciprocal of the standard deviation of the corresponding mean ISI for the fit by a function of the form $f (\text{PPT}) = a \times e^{-b \times \text{PPT}} + c$. Slopes of the exponential fits (R^2: simulation = 0.92, experiment = 0.75) reached above −0.1 at PPT of 134 ms (observed) and 157 ms (simulated) and leveled off $\{\text{ISI}_{\min} = \lim_{\text{PPT} \to \infty} f (\text{PPT}) = c\}$ at ISI of 196 ms (observed) and 190 ms (simulated). Simulated saccades directed to the second target started at least 67 ms and on average (±SD) 237 (±93) ms after the end of saccades directed to the first target, indicating that the order of sequential saccades was always maintained.

Stability of the model

In principle, a stable capacity-sharing model that accounts for the progressive postponement of consecutive
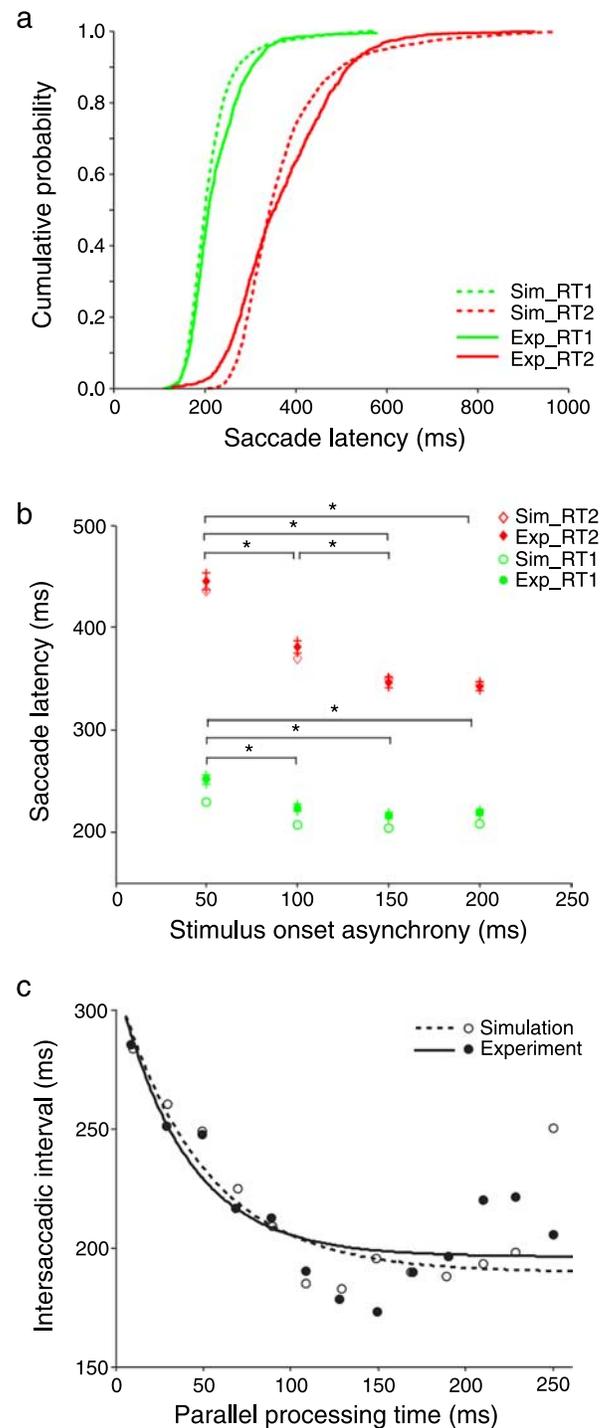
Figure 4. (a) Distributions of simulated (dotted) and observed (solid) latencies of saccades directed to the first (green) and second (red) targets. (b) Average simulated (unfilled) and observed (filled) latencies of saccades directed to the first (circle) and second (diamond) targets are contrasted at different SOAs. Error bars show the standard errors of the mean saccade latencies. Significant ($P < 0.001$) differences between two sets of saccade latencies at different SOAs are marked by asterisks. (c) Exponential fit of intersaccadic interval (ISI) plotted against parallel processing time (PPT) for observed (solid) and simulated (broken) data, respectively.

responses due to the shortening of the delay between the appearances of stimuli should also be able to fit the behavior in a situation when the distribution of capacity is highly biased in favor of a response. We recorded eye movements of two additional subjects in the same task, but they were not encouraged to generate the subsequent saccades "as fast as possible," unlike other subjects examined previously. We anticipated that possibly more capacity would be allocated to saccades directed to the first target in the absence of an urgency of generating subsequent saccades to the second target. The left panel of Figures 5a and 5b each shows the plots of the mean ISI against the mean PPT (top) in ten bins each spanning 15 ms on the abscissa and the mean latency of the first and second saccades against SOA (bottom) for subjects KR and KV, respectively. Previously, we showed that the decrease of ISI with increasing PPT between 0 and 150 ms is a hallmark of parallel processing of consecutive saccades (Ray et al., 2004). Our data indicate that while subject KR exhibited parallel processing of sequential saccades, in case of subject KV, preparation of saccades to the second target was apparently postponed until the onset of saccades to the first target [regression slope: -0.55 (KR), 0.08 (KV); correlation coefficient: -0.24 (KR), 0.06 (KV); $P < 0.001$ (KR), $P = 0.21$ (KV)]. Saccades directed to the second target were progressively postponed as SOA decreased [regression slope: -0.63 (KR), -0.80 (KV); correlation coefficient: -0.97 (KR), -0.99 (KV); $P < 0.05$]. In contrast, none of the subjects postponed saccades directed to the first target when SOA decreased.

In order to test the stability of our model with the optimized free parameters m (= 17.62) and n (= 4), we compared the simulated data with the data recorded from individual subjects. We measured the sharing proportion (SP), the mean rate of processing saccades in the direction of the first target (μ_1), the mean rate of planning saccades in the direction of the second target (μ_2), and the variance (σ) in the mean rate of saccade processing for each subject following the techniques described in the Finding parameters of the model section. Values of these parameters are shown in Table 2. Both subjects allocated about 18% (i.e., $1-SP$) of total capacity for planning saccades to the second target in comparison to 34.5% in previous cases when other subjects were encouraged to speed up saccades to the second target. Figures 5a and 5b (right panels) show the plots of the mean simulated ISI against the mean simulated PPT (top) in ten bins, each spanning 15 ms on the abscissa, and the mean RT of the simulated first and second saccades against SOA (bottom) for subjects KR and KV, respectively. Our model successfully simulated the behavior of subject KR who exhibited some degree of parallel processing of sequential saccades and subject KV as well who apparently processed consecutive saccades in series [regression slope: -0.27 (KR), -0.09 (KV); correlation coefficient: -0.09 (KR), -0.03 (KV); $P < 0.05$ (KR), $P = 0.49$ (KV)]. Saccades directed to the second target were progressively postponed

as SOA decreased [regression slope: -0.60 (KR), -0.82 (KV); correlation coefficient: -0.99; $P < 0.05$]. In contrast, the simulated latency, like the observed latency, of saccades directed to the first target did not show correlation with SOA.

Predictions of the model

Figure 6a shows how the distribution of capacity influences simulated saccade latency. In a complete bottleneck condition (i.e., SP = 1), planning a saccade to the second target is postponed until the onset of the preceding saccade to the first target, which causes a sharp rise in the mean second saccade latency at the shortest SOA, but the mean first saccade latency remains almost invariant across SOAs. As SP decreases (i.e., second saccade uses larger portion of capacity), the first saccade latency increases and the second saccade latency decreases at the shortest SOA. Simulation of our model also shows that the serial order of saccades is not always preserved without mutual inhibition ($m = 0$), while other parameters of the model are kept fixed (Figure 6b). Note that the order of saccade execution is switched in less than 50% of total simulated trials even when two targets appear simultaneously (SOA = 0 ms), because the average rate of planning saccades to the second target was naturally slower than that to the first target (Table 1). The attenuated signals X_1 and X_2 in a simulated step trial and that averaged across the trials are shown in Figures 6c and 6d, respectively. The order of saccades in a sequence may be switched if a saccade to the second target is planned independently ($m = 0$) at a relatively faster speed. Our model suggests that the order of consecutive saccades is maintained by mutual inhibition ($m > 0$) between decision signals rising to a threshold to generate saccades, irrespective of the speed of their rise (Figure 6c). Figure 6d shows the output of each attenuator averaged across 1000 trials and aligned on the beginning of the simulated first saccades, which suggests that the rate of presaccadic increase in the activity of neurons related to saccade planning is slower when the planning stage of a saccade temporally overlaps with that of another saccade, in comparison to when a saccade is planned in isolation. A steady rise of the decision signal corresponding to the first saccade slowly halts after the appearance of the second target until ~100 ms prior to the saccade onset due to an inhibitory interaction.

Discussion

The primary objective of this study was to identify a mechanism that allows simultaneous preparation of sequential saccades as an alternative to one in which
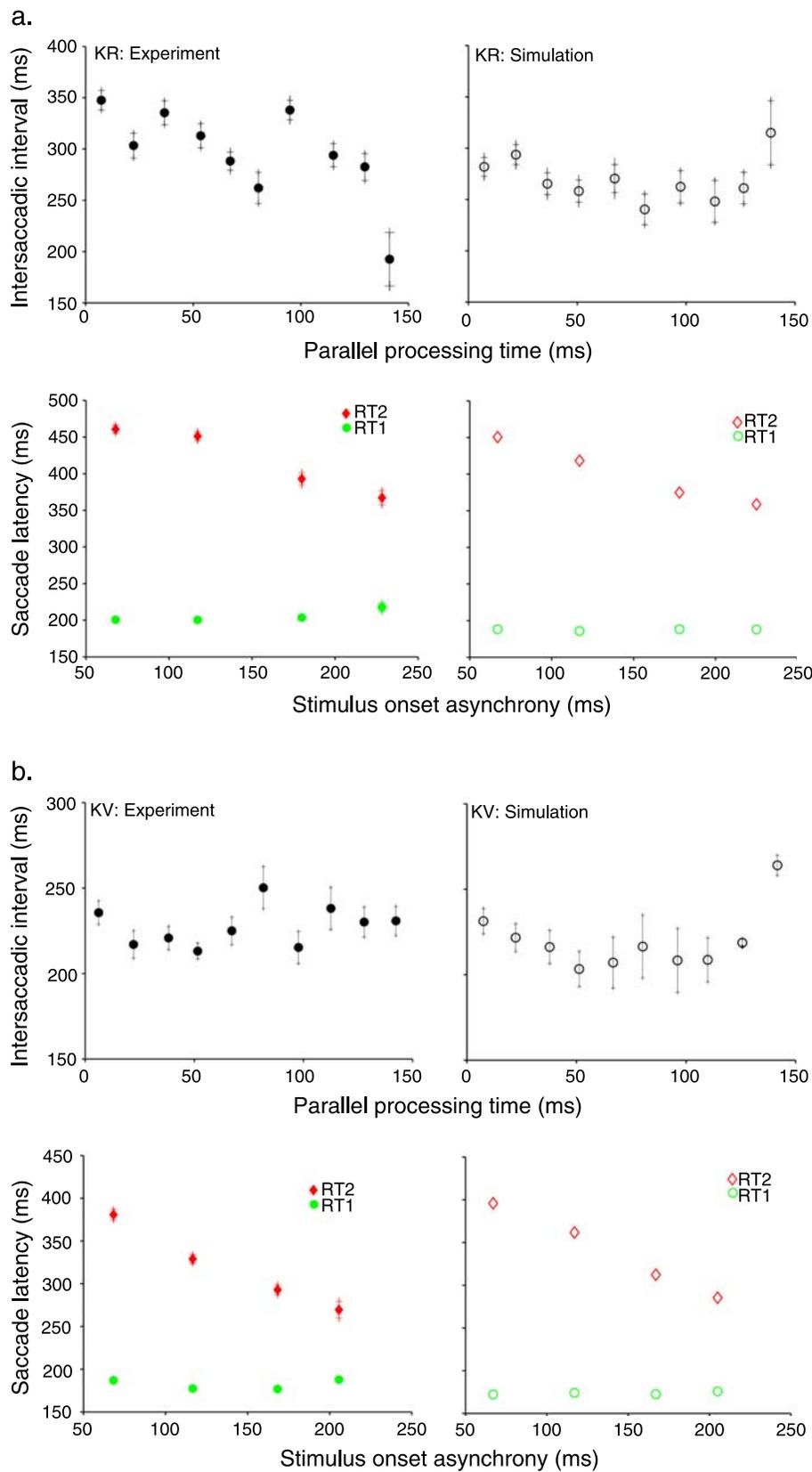
Figure 5. (a) Mean ISI is plotted against the mean PPT for ten bins each spanning 15 ms on the abscissa (top), and the mean latency of the first (circle) and second (diamond) saccades are plotted against SOA (bottom), measured from the data recorded from subject KR (left panel) and simulated on the computer (right panel). (b) The same plots derived from the data recorded from subject KV (left panel) and simulated on the computer (right panel). Error bars show the standard errors of the mean saccade latencies and the mean intersaccadic intervals.

		Parameter		
Subject	SP	μ_1 (ms^{-1})	μ_2 (ms^{-1})	σ (ms^{-1})
KR	0.8155	0.0056	0.0030	0.0012
KV	0.8179	0.0061	0.0044	0.0013

Table 2. Sharing proportion (SP), average rate of processing of saccades directed to the first target (μ_1), average rate of planning of saccades directed to the second target (μ_2), and variance (σ) in the mean rate of saccade processing for two subjects. These parameters in addition to the subject-independent fixed parameters of the model ($m = 17.62$, $n = 4$) were used to simulate behavior of subjects KR and KV.

individual saccades are planned strictly in series (Goldberg & Bruce, 1990; Robinson, 1973) or the entire sequence is organized as a preprogrammed package to preserve the serial order of saccades (Ditterich, Eggert, & Straube, 1998; Lévy-Schoen & Blanc-Garin, 1974; Zingale & Kowler, 1987). The intersaccadic fixation duration decreases with increasing delay between onsets of the target for the second saccade and the first saccade and may even fall below the average saccade latency, suggesting parallel processing of consecutive saccades (Becker & Jürgens, 1979; McPeek & Keller, 2002; Murthy et al., 2007; Murthy, Ray, Shorter, Schall, & Thompson, 2009).
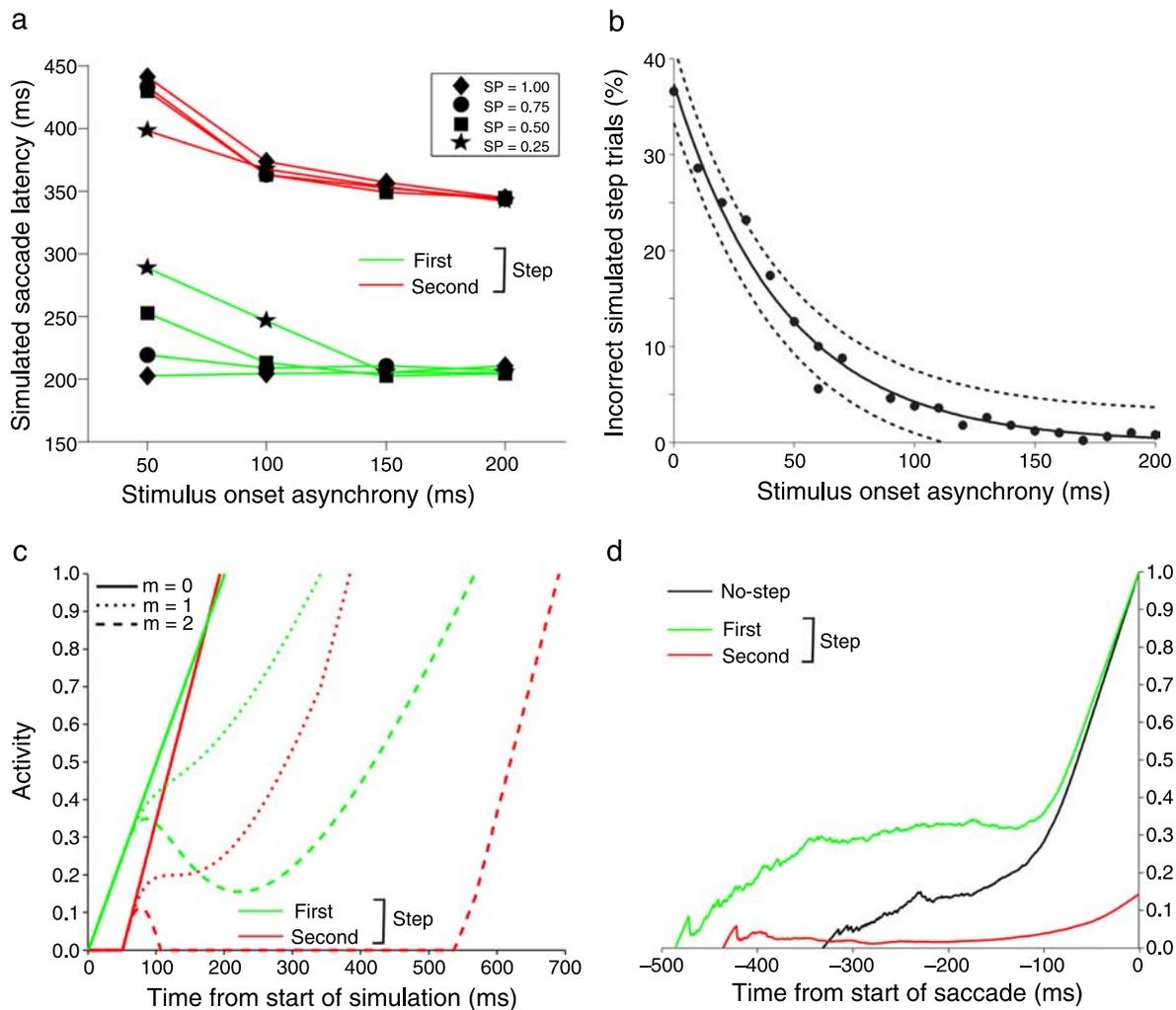
Figure 6. (a) Mean latency of simulated saccades directed to the first (green) and second (red) targets are shown as a function of stimulus onset asynchrony (SOA) with different proportion of allocated capacity (diamond: SP = 1, circle: SP = 0.75, square: SP = 0.50, polygon: SP = 0.25). The effect of capacity sharing is most prominent at the shortest SOA. (b) In the absence of mutual inhibition between saccades, the percentage of simulated step trials in which the targets were followed in the opposite order of their appearances decreased exponentially (solid, $R^2 = 0.98$) as displayed with 95% prediction bound (dotted). (c) An example of the preservation of serial order of saccades by means of mutual inhibition. In the absence of mutual inhibition (solid: $m = 0$), a saccade in the direction of the second target (red) may be elicited before a saccade in the direction of the first target (green), if planned at a faster rate. Mutual inhibition between saccades (e.g., dotted: $m = 1$, dashed: $m = 2$) preserves the order of their directions. (d) Activity in an attenuator averaged across step trials corresponding to saccades generated in the direction of the first (green) and second (red) targets that appeared 50 ms apart and that averaged across no-step trials (black).

On the contrary, an inverse relationship between the stimulus onset asynchrony (SOA) and saccade latency suggests a potential processing bottleneck in the brain (Lünenburger, Lindner, & Hoffmann, 2003; Ray et al., 2004). In this study, to reconcile these two apparently antagonistic phenomena together, we designed a model in which two interactive decision signals rise to a threshold to initiate successive saccades. Our model reliably accounts for an increase in saccade latency with decreasing SOA and a decrease in ISI down to a limit with increasing PPT. Most importantly, simulation of the model suggests that consecutive saccades share capacity and inhibit each other when processed in parallel to maintain their order without using feedback information of the progress in saccade planning, presumably to expedite saccade execution. Our model can accommodate cognitive control (e.g., allocation of attention) over sequential saccades as well by preferentially allocating capacity to one or the other saccade in the sequence, in other words, by modulating the value of the critical parameter SP in Equations 16 and 17.

Although the influence of the appearance of the second target on the first saccade latency cannot be ruled out due to the remote distractor effect (Ludwig, Gilchrist, & McSorley, 2005; Walker, Deubel, Schneider, & Findlay, 1997), it is unlikely that the increase in the second saccade latency was due to the presence of the first target, because a remote distractor has no effect on the saccade latency when it appears ~80 ms or earlier than the onset of the saccade target (Bompas & Sumner, 2009). Furthermore, because the frequencies of the shorter and longer SOAs were equal, it is also unlikely that SOA played any role in favoring the parallel mode over the serial mode of saccade processing (Miller, Ulrich, & Rolke, 2009).

Limitations of the model

In our model, for simplicity and compatibility with literature on the capacity-sharing model of dual-task interference, we assumed that the visual processing, saccade planning, and saccade execution occur in series (Pashler, 1994; Tombu & Jolicœur, 2003). Whether saccadic reaction time is the sum of duration of distinct subprocesses (Donders, 1868/1969; Sternberg, 1969) or information flows continuously resulting in an overlap between visual processing and motor planning stages (Eriksen & Schultz, 1979; Meyer, Osman, Irwin, & Yantis, 1988) is controversial. However, electrophysiological studies on behaving animals show that the saccade planning in the frontal eye field (FEF) begins after completion of visual processing of the target (Thompson, Hanes, Bichot, & Schall, 1996; Woodman, Kang, Thompson, & Schall, 2008) and lend support to the concept of compartmentalization of sensory and motor processing stages. Our data showed that the saccade latency increases exponentially with the parallel processing time (PPT) that

includes both the duration of visual processing and saccade planning; therefore, the nature of transfer of information between these stages is not critical to describe the phenomenon. Our assumption that the saccade planning starts strictly 70 ms after the target onset in every trial may not be correct, but the quality of fit of the data by the model suggests that this delay adequately serves the purpose across many trials.

Another important point to be noted here is that although the average latency of simulated saccades matched the average of experimental saccade latencies, their distributions were slightly different as shown in Figure 4a. An Ansari–Bradley dispersion test showed that the latency distribution of simulated and observed saccades directed to the first target were identical in two cases, when SOA was 150 ms and 200 ms, but the latency distribution of simulated and observed saccades directed to the second target were different for all SOAs. The reason for this anomaly was the assumption that uncertainties in the speed of saccade planning to both targets were the same, which seems to be incorrect. Unfortunately, because there was no means to measure the variability in the latency of saccades made in isolation to the second target in the correct step trials, this shortcoming of our model was unavoidable. However, the validity of our model does not hinge on its ability to reproduce the latency distributions rather on the ability to successfully simulate the relationship between the average saccade latency and SOA and between ISI and PPT (Figures 4b and 4c). An invariance of the first saccade latency with SOA for subjects who were not explicitly instructed to generate saccades rapidly to the second target suggests a processing bottleneck, which in theory corresponds to the situation SP = 1 in Equation 7. On the contrary, our model suggests that the subjects did not allocate full capacity (SP ≈ 0.82) for planning saccades in the direction of the first target. This anomaly can be resolved if we consider that faster processing of saccades to the first target ($\mu_{1,KR} = 0.0056$ and $\mu_{1,KV} = 0.0061$ compared to $\mu_{1,Population} = 0.005$) effectively reduced the duration of capacity sharing. Thus, the magnitude of postponement of first saccades decreased as well, resulting in a putative processing bottleneck.

Capacity limitations in the context of eye movements

In the literature, the term "capacity" has been described in many ways: as a structural resource for simultaneously holding information of multiple objects in memory (Fukuda, Awh, & Vogel, 2010), as a form of attention enabling stimulus identification (Marois, Chun, & Gore, 2000), and as a strategic resource for response selection (Pashler, 1999) and executive control (Sigman & Dehaene, 2006). Several electrophysiological and imaging experiments have been carried out on humans and

non-human primates to identify neural substrates of capacity limitations in the working memory and executive control systems (e.g., Buschman, Siegel, Roy, & Miller, 2011; D'Esposito et al., 1995; Luck, 1998; Palva, Monto, Kulashekhar, & Palva, 2010). Nonetheless, whether capacity limitation represents a structural bottleneck or a strategic inadequacy is still vague (Marois & Ivanoff, 2005), and "the precise nature of this limited-capacity stage of processing remains unspecified" (Tombu & Jolicœur, 2005).

Results from previous attempts to identify a bottleneck in planning eye movements are controversial. Some studies that combined eye and hand movements toward the same target did not notice a delay in either movement (Mather & Fisk, 1985; Mather & Putchat, 1983; Megaw & Armstrong, 1973), but others found interference between tasks, one of which involved an eye movement (Malmstrom, Reed, & Randle, 1983; Pashler, Carrier, & Hoffman, 1993; Stuyven, Van der Goten, Vandierendonck, Claeys, & Crevits, 2000; Remington, Wu, & Pashler, 2011). Although our task does not recruit two independent tasks, but in the context of parallel processing of sequential saccades, the observed slowing of the second response with decreasing stimulus onset asynchrony (SOA), which is known as the "psychological refractory period" (PRP) effect (Telford, 1931) in the dual-task paradigm, suggests that the saccade planning is capacity limited.

Interactive ramp-to-threshold models of response time

Our study establishes a link between the dual-task paradigm (Welford, 1952) and the double-step paradigm (Wheeless, Boynton, & Cohen, 1966) using concepts derived from the capacity-sharing model and the LATER model of response time. Previous attempts to relate a ramp-to-threshold model of reaction time to dual-task interference suggested that an interaction between neural populations accumulating information is required to account for a response time correlation during coordinated eye–hand movements (Dean, Martí, Tsui, Rinzel, & Pesaran, 2011) and a central response selection bottleneck that enforces postponement of the planning of the second response until the onset of the first response (Sigman & Dehaene, 2005; Zylberberg, Slezak, Roelfsema, Dehaene, & Sigman, 2010). In the domain of non-linear accumulator models, the concept of an inhibitory interaction between competing decision signals has been implemented successfully to fit the pattern of the neural activity underlying the behavior in tasks that mainly probed choice, performance, or accuracy (e.g., Arai, Keller, & Edelman, 1994; Bogacz et al., 2007; Boucher, Palmeri, Logan, & Schall, 2007; Cutsuridis, Smyrnis, Evdokimidis, & Perantonis, 2007; Gold & Shadlen, 2003; Ratcliff, Hasegawa, Hasegawa, Smith, & Segraves, 2007; Trappenberg, Dorris, Munoz, &

Klein, 2001; Usher & McClelland, 2001; Wong & Wang, 2006). On the other hand, an interaction between a pair of LATER units has been used to fit the distribution of response time (Leach & Carpenter, 2001; Story & Carpenter, 2009) and the duration of fixation between a pair of evoked or spontaneous saccades (Roos, Calandrini, & Carpenter, 2008). Our preference for the linear accumulator(s) was solely intended to reduce the number of parameters of the model. We do not claim that our model is the only model that can explain the serial order of saccades; however, we do believe that the introduction of the idea of capacity sharing and mutual inhibition between ramping decision signals is an exciting prospect and the proposed framework is a plausible one.

Physiological feasibility of the model

A common belief that the retinal information about the difference between the current position of the gaze and the location of the target is the only source to generate an eye movement (Robinson, 1973) fails to explain how a saccade can be directed to a target that disappears before the onset of the preceding saccade directed to another target (Hallett & Lightstone, 1976), suggesting that the retinal information of the location of the second target combined with the extraretinal signal of present eye position is critical for the production of rapid sequential saccades (Bock, Goltz, Belanger, & Steinbach, 1995; Collins, 2010; Doré-Mazars, Vergilino-Perez, Collins, Bohacova, & Beauvillain, 2006; Honda, 1997; Joiner, FitzGibbon, & Wurtz, 2010; Munuera, Morel, Duhamel, & Deneve, 2009), which has been confirmed physiologically (Mays & Sparks, 1980). Nevertheless, research is ongoing to understand how these signals are combined (Sommer & Wurtz, 2002; Vaziri, Diedrichsen, & Shadmehr, 2006) or the type of extraretinal signals—eye position or eye displacement—are used (Goossens & Opstal, 1997). Furthermore, the ability to plan consecutive saccades in parallel requires an obligatory control to prevent a saccade from being directed to an intermediate location between respective targets (Chou, Sommer, & Schiller, 1999; Findlay & Gilchrist, 1997; Ottes, Van Gisbergen, & Eggermont, 1984) or being curved (McPeek, 2006). More recent works show that the oculomotor system prepares motor commands by converting goals into corresponding movement vectors (Quaia, Joiner, FitzGibbon, Optican, & Smith, 2010) to bypass an update in the "goal space" due to the gaze shift (Duhamel, Colby, & Goldberg, 1992), suggesting that the ramping signals in the accumulators of our model represent motor plans and not the goals for saccades.

Movement neurons in the frontal eye field (FEF) of the primate brain seemingly increase firing frequency linearly before saccade execution at a rate inversely related to the saccade latency (Hanes & Schall, 1996), but a delayed

presaccadic augmentation in the firing frequency of the FEF visuomovement neurons occurs exclusively when the saccade is inevitable (Ray, Pouget, & Schall, 2009). The biphasic pattern of simulated activity in attenuators (Figure 6b) resembles visuomovement activity in the FEF that has been modeled in different other contexts by a lateral inhibition between competing decision signals rising to a threshold (Hamker, 2005; Stanford, Shankar, Massoglia, Costello, & Salinas, 2009). Alternatively, mutual inhibition may also be mediated by the basal ganglia that disinhibit superior colliculus (SC) through the direct and indirect pathways (Hikosaka, Takikawa, & Kawagoe, 2000). Visuomovement neurons in the SC have been seen to contribute to processing consecutive saccades in parallel (McPeek & Keller, 2002). The presence of multiple pathways might be a particularly attractive architecture to implement such a capacity limitation through inhibitory control.

Appendix A

We assumed that A1 (A2), B1 (B2), and C1 (C2) refer to the duration of the visual processing stage, planning stage, and execution stage of a saccade directed to the first (second) target, respectively, when saccades are generated in isolation or do not share capacity (Figure 2a). The duration of the planning stage of a saccade directed to the first target increases from B1 to B1′, and that of a saccade directed to the second target increases from B2 to B2′, if consecutive saccades share capacity (Figure 2b). When the second target appears after SOA (i.e., SOA_1/SOA_2 in Figure 2b, top), planning stages of consecutive saccades overlap (SOA + A2) after the onset of the first target and planning of both saccades continue simultaneously until the onset of a saccade. Suppose that the fraction of capacity allocated for planning a saccade in the direction of the first target, denoted by the *sharing proportion*, is SP (0 < SP < 1). The remaining capacity (1 − SP) is assumed to be allocated for planning another saccade in the direction of the second target. A saccade in the direction of the first target is planned with full capacity (SP = 1) for a period of (SOA + A2 − A1) until planning of a saccade to the second target begins. During this period, the oculomotor system finishes {(SOA + A2 − A1) × SP} or (SOA + A2 − A1) part of planning of a saccade directed to the first target. The remaining part of the planning, which is {B1 − (SOA + A2 − A1)}, takes time equal to $\left\{\frac{B1-(SOA+A2-A1)}{SP}\right\}$ to finish with a fraction of capacity (0 < SP < 1). The net amount of time these saccades share capacity is denoted by $D = \left\{\frac{B1-(SOA+A2-A1)}{SP}\right\}$. Therefore, the delay (RT1′) between the onset of the first target and a saccade in that direction is the sum of the

duration of the visual processing stage and planning stage with full and partial capacities as shown in order in the following equation:

$$RT1' = [A1 + (SOA + A2 - A1) + D],$$

$$RT1' = \left[A1 + (SOA + A2 - A1) + \left\{\frac{B1 - (SOA + A2 - A1)}{SP}\right\}\right],$$

$$RT1' = \left(\frac{SP-1}{SP}\right) \times SOA + \left(\frac{SP-1}{SP}\right) \times A2 + \frac{A1+B1}{SP}. \tag{A1}$$

During capacity sharing, $[D \times (1 - SP)]$ or $\left[\frac{B1-(SOA+A2-A1)}{SP} \times (1 - SP)\right]$ part of planning of the saccade in the direction of the second target is accomplished, leaving $B2 - \left\{\frac{B1-(SOA+A2-A1)}{SP} \times (1 - SP)\right\}$ part to be planned with full capacity (SP = 1), which takes time equal to $\left\{B2 - \frac{B1-(SOA+A2-A1)}{SP} \times (1 - SP)\right\}$. Therefore, the delay (RT2′) between the onset of the second target and a saccade in that direction is the sum of the duration of the visual processing stage and planning stage with partial and full capacities as shown in order in the following equation.

$$RT2' = \left[A2 + D + \left\{B2 - \frac{B1 - (SOA + A2 - A1)}{SP} \times (1 - SP)\right\}\right],$$

$$RT2' = \left[A2 + \left\{\frac{B1 - (SOA + A2 - A1)}{SP}\right\} \right.$$
$$\left. + \left\{B2 - \frac{B1 - (SOA + A2 - A1)}{SP} \times (1 - SP)\right\}\right]. \tag{A2}$$

After simplification, we get

$$RT2' = A1 + B1 + B2 - SOA. \tag{A3}$$

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