

ICAT: a computational model for the adaptive control of fixation durations

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Abstract Eye movements depend on cognitive processes related to visual information processing. Much has been learned about the spatial selection of fixation locations, while the principles governing the temporal control (fixation durations) are less clear. Here, we review current theories for the control of fixation durations in tasks like visual search, scanning, scene perception, and reading and propose a new model for the control of fixation durations. We distinguish two local principles from one global principle of control. First, an autonomous saccade timer initiates saccades after random time intervals (local-I). Second, foveal inhibition permits immediate prolongation of fixation durations by ongoing processing (local-II). Third, saccade timing is adaptive, so that the mean timer value depends on task requirements and fixation history (Global). We demonstrate by numerical simulations that our model qualitatively reproduces patterns of mean fixation durations and fixation duration distributions observed in typical experiments. When combined with assumptions of saccade target selection and oculomotor control, the model accounts for both temporal and spatial aspects of eye movement control in two versions of a visual search task. We conclude that the model provides a promising framework for the control of fixation durations in saccadic tasks.

Keywords Computational modeling · Eye movements · Adaptive control · Fixation duration

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Introduction

Visual perception is a highly dynamic process during which saccadic eye movements continually scan the environment (Findlay and Gilchrist, 2003). Because of the anatomy of the retina and resulting acuity limitations outside of the foveal region, eye movements are essential for human vision. The generation of saccadic eye movements is based on two largely independent pathways for spatial (*where*) and temporal (*when*) control (Findlay and Walker, 1999). Due to this independence, a research strategy focusing on spatial aspects of saccade generation turned out to be successful, so that, over the last decade, considerable progress was made on the question of how the next saccade target is selected. Most important, this research generated detailed computational models that account for the selection of fixation locations during reading (e.g., Engbert and Krügel, 2010; Engbert, Nuthmann, Richter, and Kliegl, 2005; Reichle, Pollatsek, Fisher, & Rayner, 1998), scene perception (e.g., Itti and Koch, 2001; Kienzle, Franz, Schölkopf, and Wichmann, 2009; Torralba, Oliva, Castelhano, and Henderson, 2006), and visual search (e.g., Najemnik and Geisler, 2005; Rutishauser and Koch, 2007; Zelinsky, 2008).

Different from the spatial control of eye movements, temporal control processes, which determine the duration of a fixation, were almost exclusively addressed in eye movement models in reading (e.g., Engbert et al., 2005; Reichle et al., 1998; for an overview see Reichle, Rayner, and Pollatsek, 2003). To fill this gap, Rayner (2009) suggested that experimental paradigms developed to analyze fixation durations in reading should be more widely adapted to other domains of eye movement research. This research strategy was recently applied to a series of experiments that utilized the stimulus onset delay paradigm (Rayner and Pollatsek, 1981) to analyze the control of fixation durations in scene viewing (Henderson and Pierce, 2008; Henderson and Smith, 2009). On the basis of these results, Nuthmann, Smith, Engbert and Henderson,

(2010) proposed a computational model for the control of eye movements in scene viewing (CRISP), which implemented some of the principles developed in the SWIFT model for saccade generation in reading (Engbert et al., 2005).

In this article, we generalize the computational approach introduced by Engbert et al. (2005) and Nuthmann et al. (2010) and address the control of fixation durations from a broader perspective. The starting hypothesis is that fixation durations are controlled on two different levels: (1) Local control, depending on processing difficulty of the item in foveal vision, affects the current fixation, and (2) global control related to overall task requirements affects fixations over a sustained period, and not just single fixations. Current models provide detailed explanations of local control. For example, in reading, a low-frequency word consumes more processing time and produces higher fixation durations (Reichle et al., 2003). However, global control is either neglected or only assumed implicitly, even though it constitutes a key mechanism in a number of theories—for example, for the adjustment of fixation durations across tasks (Hooge and Erkelens, 1998). According to our hypothesis of global and local control principles, average fixation durations depend on our previous experience with specific visuomotor tasks and on anticipated processing difficulty. Using mathematical modeling and computer simulations, we investigate the interplay between global and local control. Our approach is in agreement with experimental results on fixation durations derived from different paradigms, and we demonstrate that a limited set of control principles can account for the temporal control of eye movements across tasks.

Global control of fixation durations

Fixation durations range from less than a hundred milliseconds to more than a second. Some of this variability is related to global adjustments that affect fixations over sustained periods of time. At least three major sources can be distinguished that cause global adjustments. First, variability of fixation durations is related to global properties of the stimulus material. For example, overall visual clutter in a scene has an impact on fixation durations (Henderson, Chanceaux, and Smith, 2009). Interestingly, local clutter around the point of fixation does not alter fixation durations. In addition, mean fixation durations increase with decreasing luminance of pictures (Loftus, 1985) and with decreasing contrast during viewing of line drawings (Loftus, 1985), scene perception (Loftus, Kaufman, Nishimoto, and Ruthruff, 1992), visual search (Näsänen, Ojanpää, and Kojo, 2001), and reading (Bowers and Reid, 1997). Low- and high-pass filtering of an image changes average fixation durations (Groner, Groner, and von Mühlenen, 2008; Mannan, Ruddock, and Wooding, 1995), and stimulus difficulty modulates fixation durations, which increase with increasing target–distractor similarity (Hooge and Erkelens, 1998; Jacobs, 1986; Vlaskamp, Over, and

Hooge, 2005) and increasing complexity of stimulus elements (Gould and Dill, 1969).

Second, task-specific processing (Rayner et al., 2007) affects average fixation durations that last about 225–250 ms during reading, 275–325 ms during scene perception, and 180–275 ms during visual search (for a review, see Rayner, 2009). These modulations do not depend solely on stimulus properties and remain when the same stimulus material is viewed under differing instructions. During scene perception, fixations are longer when a scene is memorized, in comparison with searching the same scene (Henderson, Weeks, and Hollingworth, 1999; but see Castelano, Mack, and Henderson, 2009). During reading, average gaze durations decrease when a text is skimmed (Just and Carpenter, 1987) or the same text is read repeatedly (Hyönä and Niemi, 1990) and lengthen during elaborate processing (Bohn and Kliegl, 2007) and proof-reading (Wotschack, 2009). Similarly, fixation durations increase with memory load during visual search (Gould, 1973).

Third, large interindividual differences are observed in mean fixation durations. To some degree, these differences can be linked to practice. Reading fixations shorten from first to sixth grade (McConkie et al., 1991), and old and young readers use different strategies while reading a text (Rayner, Yang, Castelano, and Liversedge, 2011). Interindividual differences persist across tasks, revealing a tendency to a preferred average fixation duration in participants (Castelano and Henderson, 2008; Rayner et al., 2007). According to our hypothesis, enduring modulations of fixation durations are the result of an adjustment of global control strategies, while local control strategies may modify the duration spent on individual fixations.

Theories of local control of fixation durations

Local fixation duration modulations have been investigated extensively. Resulting theories can be classified into three categories, which assume that there is control by a cognitive trigger (direct control), indirect control, or mixed control (Rayner, 1977; Rayner and McConkie, 1976; Rayner and Pollatsek, 1981). *Cognitive trigger* theories assume that the decision to move the eyes is based solely on processing of visual input during a fixation. Saccades might be triggered by the decision as to whether a target is present in the fixated area during visual search (Rayner, 1995), identification of an object in scene perception (Henderson, 1992), or estimation of a word's familiarity in reading (Reichle et al., 1998). For scanning tasks, Reichle, Pollatsek, and Rayner (2013) suggested that the trigger to initiate a new saccade program is synchronized with the trigger that shifts attention to the next object.¹

¹ More complex variants are conceivable where saccades are triggered by processing beyond the foveal region or by multiple competing processing streams. In this article, we restrict cognitive trigger theories to their simplest form, where saccade programs are initiated by processing of input from the foveated area.

Thus, cognitive trigger theories postulate a tight link between processing and saccade initiation. Due to the triggering mechanism, fixation durations are instantaneously adjusted to processing demands at fixated locations, and the interpretation is that fixation durations reflect processing during a fixation. Obviously, cognitive triggers are an attractive model for generating hypotheses about the relation between oculomotor control and ongoing cognition.

As an alternative, *indirect control* theories assume that the control of fixation durations cannot be limited to processing of the fixated region. The motivation for this claim is that average fixation durations range from 200 to 300 ms, a time window that might be too short for the control of fixation durations by a cognitive trigger. First, because of saccadic suppression, visual input is reduced during the first 35 ms of a fixation (Volkman, Schick, and Riggs, 1968). Second, due to the eye–brain lag, visual input needs at least 50 ms to be transmitted from the retina to the cortex (Foxe and Simpson, 2002; Lamme and Roelfsema, 2000; Poghosyan and Ioannides, 2007). Third, programming of saccades takes 150–175 ms (Becker and Jürgens, 1979; Rayner, 1998; Rayner et al., 1983; note that Schall and Thompson, 1999, give slightly smaller estimates of 100–150 ms). While processing may continue in parallel with the programming of a saccade, the decision of a cognitive trigger has to be based on the information available beforehand. From these physiological restrictions, a very short time interval is available for cognition to trigger saccade programming, and even if cognitive trigger theories are an adequate approximation of saccade generation, decisions to move the eyes would be based on partial knowledge about the fixated region.

Contrary to control by cognitive triggers, indirect control suggests that there is no link between processing and saccade initiation. Instead, saccades are triggered by an autonomous process after a random time interval (Hooze and Erkelens, 1998; Kolers, 1976); that is, each time interval is sampled from a predefined distribution. The mean duration of this autonomous process is adjusted to match task demands but is not influenced by processing of the fixated area. Thus, indirect control theories assume that the average fixation duration depends on adjustments by global control. Such a theory is supported by the well-established empirical observation that additional processing time is predominantly acquired by refixations, and not by prolongation of the current fixation.

Finally, *mixed control* theories allow influences of the current fixation, but here, cognitive processing is not the single triggering mechanism for saccades. For reading, Henderson and Ferreira (1990) proposed that saccades are initiated when attention moves to the next word or after a deadline independent of current processing is reached (cf., Engbert and Kliegl, 2001). Thus, fixation durations might be controlled by a combination of cognitive trigger mechanisms and indirect control mechanisms. A different class of mixed control

theories assume that fixation durations are terminated primarily by indirect control, where processing may prolong the current fixation by inhibiting the execution of the next saccade (Engbert et al., 2005; Feng, 2009; Yang and McConkie, 2001).

Fixations durations across tasks

In the upcoming section, we review the literature on fixation durations during visual search, reading, and scene perception and discuss how these results relate to theories of the control of fixation durations.

Fixation durations during visual search

Hooze and Erkelens (1996, 1998, 1999) investigated the control of fixation durations in a series of visual search tasks. In a first experiment (Hooze and Erkelens, 1996), mean fixation durations reflected average task demands, but durations of single fixations were not adjusted to properties of the fixated stimulus. Instead, saccades were initiated before foveal analysis of the fixated stimulus was completed. This caused a large proportion of saccades to move the eyes away from the target symbol, even though participants were instructed to keep the eyes on the target after an initial fixation. In a second experiment, participants' eyes were guided by foveal information (Hooze and Erkelens, 1998). During this direction-coded search, participants were explicitly instructed to always move the eyes according to the direction given by the fixated stimulus, but a large proportion of the fixations were too short to select the correct saccade target. Hooze and Erkelens (1998) concluded that saccades are initiated after random time intervals independently of ongoing foveal processing. In such a random-timing model, a mean timer interval is set by a global control mechanism to a value that matches mean processing demands of the foveal (Hooze and Erkelens, 1999) and/or parafoveal (Vlaskamp and Hooze, 2006) analysis, while single fixation durations are not adjusted to ongoing foveal processing.

Greene and Rayner (2001) examined eye movements in another direction-coded search task. Different from Hooze and Erkelens's (1996, 1998) displays, the arrangement of stimulus elements by Greene and Rayner was denser and permitted parafoveal preview of neighboring symbols. As a consequence, Greene and Rayner observed a higher proportion of correctly directed saccades and argued for a tight link between processing and eye movement control in dense, as compared with sparse, displays. However, Greene and Rayner observed a fraction of saccades that were not directed toward the target as predicted by indirect control theories (Hooze and Erkelens, 1998).

Results from both experiments can be explained by indirect control, since saccade selectivity depends on the relation between fixation duration and processing. If processing of a

stimulus finishes before a saccade is executed, saccades will be directed according to the informative stimulus. However, if a saccade is executed before processing is finished, saccades will be unrelated to the direction prescribed by the stimulus. Since both processing difficulty and specification of the random timer depend on multiple factors, such as stimulus, preview, and visibility, saccadic performance may vary from unselective to almost perfectly selective across tasks (see Trukenbrod and Engbert, 2007, for a direction-coded search task with even higher saccade selectivity). In line with this interpretation, saccades in visual search have been shown to be more selective after long fixation durations than after short fixations (Hooge and Erkelens, 1999).

Contrary to indirect control, some studies have observed immediate fixation duration modulations during visual search. Fixation durations increase with complexity of stimulus elements (Salthouse and Ellis, 1980) and increasing target–distractor similarity of fixated objects (Becker and Williams, 2011; Trukenbrod and Engbert, 2007; Williams and Pollatsek, 2007). Hence, rejection of distractors has been suggested as the event triggering saccades in visual search (Becker and Williams, 2011; Rayner, 1995).

Recently, Hooge, Vlaskamp, and Over, (2007) examined the influence of previously fixated items on later fixations. The most intriguing result was an asymmetry in the control of fixation durations, representing a bridge between the seemingly contradictory results. When the preceding fixation was placed on an easy item, fixation durations increased immediately on difficult symbols. In contrast, if the preceding fixation was placed on a difficult stimulus, durations of the next fixation were unaffected by the difficulty of the fixated symbol. Hooge et al. relaxed the assumption of pure indirect control and proposed a mixed control strategy in visual search. According to this strategy, fixation durations are affected asymmetrically by local and global control mechanisms: (1) Immediate prolongations of fixation durations can be seen when processing demands increase (local control) but (2) decrease only gradually (with a temporal delay) when processing demands decrease (global control).

Asymmetric control seems to play a pivotal role in the control of fixation durations during visual search. Next, we explore the control of fixation durations in other tasks and discuss how these observations relate to the notion of asymmetric control.

Fixation durations during reading

Control of fixation durations has been investigated extensively during text reading, and several word properties have been shown to affect fixation durations (for reviews of eye movements in reading, see Rayner, 1998, 2009). Printed word frequency (Just and Carpenter, 1980) and predictability (Ehrlich and Rayner, 1981) are two of the most important

factors influencing eye movement behavior at various levels. Both first-fixation durations and gaze durations are elevated on low-frequency (Inhoff and Rayner, 1986; Kennison and Clifton, 1995; Kliegl, Grabner, Rolfs, and Engbert, 2004; Rayner and Duffy, 1986) and unpredictable (Inhoff, 1984; Rayner and Well, 1996; Zola, 1984) words. Since lexical processing of a word is affected by frequency and predictability, Reichle et al. (1998) suggested lexical access as the cognitive event triggering eye movements during reading.

Several lines of evidence support the notion of a general and fast lexical influence on fixation durations in reading. First, Reingold, Yang and Rayner (2010) inspected the first of multiple fixations on a word. Even fixation durations before refixations were modulated by frequency (see Rayner, Sereno, and Raney, 1996). Second, inspection of first-fixation durations revealed that frequency (Reingold, Reichle, Glaholt, and Sheridan 2012; Staub, White, Drieghe, Hollway, and Rayner, 2010) and predictability (Staub, 2011) have an effect on the shift of fitted ex-Gaussian distributions. In addition, frequency altered the skewness of distributions, leading to larger frequency effects for long fixation durations. Thus, the entire range of fixation durations reflects lexical processing. Third, in the *disappearing text paradigm*, where the fixated word disappears or is masked about 60 ms after the onset of a fixation, frequency manipulations modulate fixation durations as in normal reading (Liversedge et al., 2004; Rayner, Liversedge and White, 2006; Rayner, Liversedge, White, and Vergilino-Perez, 2003; Rayner et al., 2011). Hence, the visual input from the first 60 ms is sufficient to encode all information needed for lexical processing.

Cognitive trigger theories postulate a causal link between linguistic processing and subsequent fixation durations. Thus, saccades should be triggered only after successful lexical processing of a word. Contrary to this, preventing preview of a word prior to its fixation postpones or weakens word frequency effects, which reappear in later fixation duration measures (Inhoff and Rayner, 1986; Sereno and Rayner, 2000). Without parafoveal preview of words, the proportion of fixations unaffected by frequency rises dramatically from about 9% with preview to over 60% without preview (Reingold et al., 2012). Thus, while most fixation durations are influenced by lexical processing under normal reading conditions, the influence of lexical processing diminishes drastically when preview is impeded. Furthermore, Bouma and de Voogd (1974) demonstrated that reading proceeds without interference when a text is presented at a rate not determined by the participant. Thus, word frequency seems to modulate fixation durations, but it is not a particular linguistic processing event related to a word's frequency that initiates new saccade programs (Deubel, O'Regan, and Radach, 2000). Nevertheless, it is beyond question that lexical processing has an immediate influence on fixation times during normal reading (Reingold et al., 2012).

In line with this interpretation, Yang and McConkie (2001) identified several principles for the control of fixation durations during reading. Participants read paragraphs in a gaze-contingent experiment. On each page, the text was replaced for the duration of one fixation by an alternative stimulus with varying similarity to the original text. In all conditions, fixation durations remained highly variable. More important, features of the new stimulus differentially affected saccades after short, medium, and long fixation durations. Saccades after short fixations (<150 ms) remained unchanged in all stimulus conditions. Saccades after medium fixation durations (>175 and <250 ms) were inhibited when the alternative stimulus disturbed word shape or word boundary information. Only saccades that occurred after long fixation durations (>275 ms) were inhibited by disruptions of cognitive processing. Similar to the asymmetric control of fixation durations in visual search (Hooge et al., 2007), saccades seemed to be initiated after random time intervals and were prolonged by ongoing processing.

Interestingly, an asymmetric fixation duration pattern has also been reported by Kennison and Clifton (1995) in a reading task. While participants read sentences, preview of a target word was prevented. Fixation durations on the target word were analyzed depending on the frequency of the preceding word. After a high-frequency word was fixated, fixation durations were affected by word frequency of the target word. Fixations were longer on low-frequency words than on high-frequency words (285 vs. 318 ms). In contrast, fixation durations after a fixation on a low-frequency word did not differ between high-frequency and low-frequency words (311 vs. 307 ms). Thus, increasing processing demands caused an immediate prolongation of fixation durations, while decreasing processing demands showed no immediate effect. However, until now, most research has focused on fixation durations on single words. Systematic manipulations of two or more words are needed to test the assumption of asymmetric control in reading.

Fixation durations during scene perception

Control of fixation durations has been rather neglected in scene perception research (Henderson, 2003; Henderson and Hollingworth, 1998; for reviews, see Henderson and Hollingworth, 1999; Henderson and Ferreira, 2004). Henderson and Hollingworth, (1992) proposed a model based on sequential attention shifts to account for eye movements in scene perception. If processing of the foveated object has not proceeded sufficiently before a deadline is reached, a refixation is initiated. Otherwise, as soon as the fixated object is identified, attention moves to the next object and triggers a saccade. In line with this assumption, changing a region during a saccade toward it prolongs subsequent fixation times even when viewers remain unaware of the change (Henderson and Hollingworth, 2003; see Hayhoe, Bensinger, and Ballard,

1998, for a similar result in a visual working memory task). Similarly, foveal masks presented at the beginning of a fixation prolong the duration proportional to the duration of the mask, while a parafoveal mask presented at the same time has a much weaker effect (van Diepen and d'Ydewalle, 2003). Thus, foveal processing seems to be crucial for the control of fixation durations. Furthermore, semantically implausible or inconsistent objects are fixated longer (Friedman and Liebelt, 1981; Henderson et al., 1999; Loftus and Mackworth, 1978). The effect typically shows up in gaze durations as refixating implausible objects more often but is fragile on the level of first-fixation durations (Henderson and Hollingworth, 1998). As an exception, De Graef et al., (1990) reported differences, which appeared primarily in later stages of scene exploration (i.e., after more than eight fixations).

Asymmetries in the control of fixation durations have not yet been reported for scene perception. However, the control principles discussed for reading (Yang and McConkie, 2001) and visual search (Hooge et al., 2007) provide a coherent way to describe effects on gaze durations, as well as individual fixation durations. Modulations of gaze durations are primarily a result of refixations in scene perception (Henderson and Ferreira, 2004) and only rarely are reflected in individual fixation durations (Henderson and Pierce, 2008). An indirect control mechanism automatically triggers new saccades (similar to a saccadic deadline; see Henderson, 1992). The eyes move to another object if the foveated object has been processed sufficiently or gain additional processing time through a refixation if more processing is needed. Furthermore, if processing progresses swiftly, fixations may be immediately prolonged by processing of the foveated region. Depending on the exact time course of processing, this may result in early modulations of first fixations by syntactic inconsistencies or in late modulations of later fixations by semantic inconsistencies (Võ and Henderson, 2009).

Fixation durations in the stimulus-onset delay paradigm

A paradigm with gaze-contingent stimulus presentation (Rayner, 1975) has been used to investigate eye movements during reading (Ishida and Ikeda, 1989; Morrison, 1984; Rayner and Pollatsek, 1981), visual search (Vaughan, 1982; Vaughan and Graefe, 1977), and scene perception (Henderson and Pierce, 2008; Henderson and Smith, 2009; Shioiri, 1993). At the end of a saccade, the stimulus is replaced by a mask (either a blank screen or an alternative stimulus), and the original stimulus returns after a variable delay. This stimulus-onset delay (SOD) ranged from zero to several hundred milliseconds in experiments. Even though multiple factors varied across experiments (e.g., task, size, and structure of the mask), all experiments revealed a fraction of fixation durations that increased proportionally with the SOD. More important, when the analysis was restricted to fixations longer

than the SOD, mean fixation durations increased almost perfectly by the amount of the SOD (with a slope of ~ 1 for the relation between SOD and average fixation duration). Thus, a significant proportion of fixation durations are controlled by foveal processing.

The same experiments, however, showed the existence of a second proportion of fixations that were not controlled by processing. As a consequence, multiple modes in fixation duration distributions have been reported in the SOD paradigm (Henderson and Pierce, 2008; Morrison, 1984; Shioiri, 1993). It has been suggested that short fixation durations are preprogrammed during preceding fixations and that the number of preprogrammed fixations increases with increasing SOD. For example, Morrison reported 15%–30% anticipatory saccades when the stimulus was delayed for 200 ms but 50%–75% for delays of 350 ms. Since preprogrammed saccades are specified during the preceding fixation, the proportion of preprogrammed saccades should not increase with increasing SOD (at least for fixation durations beyond 200 ms).

Even though results from the SOD paradigm have been widely interpreted as evidence for cognitive trigger theories, the existence of a second population of short fixation durations has generally been attributed to a mixed control mechanism (e.g., Henderson and Pierce, 2008; Henderson and Smith, 2009; Rayner and Pollatsek, 1981). In line with this, modeling results of the SOD paradigm are more compatible with the concept of mixed control. Nuthmann et al. (2010) developed the CRISP model on the basis of a random timer with foveal inhibition.² While the random timer represents indirect control, foveal inhibition allows modulations of the indirect control mechanism by processing. This mixed-control model, which is in line with the control principles proposed by Hooze et al. (2007), generated two types of saccades (a “scene-independent” and a “scene-dependent” type) and recovered the two populations of fixations with characteristic mean values and variances.

Modeling motivation and goals

A single mechanism for the control of fixation durations across tasks

In the previous sections, we demonstrated that fixation durations across tasks might be controlled by a very similar set of rules. Computational implementations of mixed control strategies provide a viable concept for the local control of fixation durations in reading (SWIFT; Engbert, Longtin, and Kliegl,

2002; Engbert et al., 2005) and scene viewing (CRISP; Nuthmann et al., 2010). In both domains, models of mixed control with a random timer, where fixation durations are sampled from a predefined distribution and inhibited by processing, reproduce complex patterns of mean values and variances of fixation durations. The same mechanisms offer a promising candidate for the asymmetric control of fixation durations observed during visual search (Hooze et al., 2007).

Global control of fixation durations While it is generally assumed that indirect control mechanisms and corresponding modulations need to adapt to actual processing needs, these adjustments have been neither experimentally nor computationally investigated. Here, we explore the interaction of local and global control processes to understand the influence of adaptive processes on fixation duration control.

Variability of fixation durations Until now, models for the temporal control of eye movements have focused primarily on average fixation duration patterns. Nonetheless, fixation durations are highly variable and may, under some circumstances, lead to multimodal distributions (cf. simulations of Yang and McConkie, 2001, in [Supplementary Material](#)). A model for the control of fixation durations needs to account for both mean fixation durations and their distributions.

Temporal constraints Finally, indirect control modulated by processing provides a solution to the neurophysiological constraints inherent to the control of fixation durations. Because of the eye–brain lag, it takes at least 50 ms for visual input to be transmitted from the retina to areas of the visual cortex (Lamme and Roelfsema, 2000). Another 150–175 ms are required after the decision to initiate a saccade to actually execute it (Rayner, 1998). Since average fixation durations are on the order of 200–300 ms in tasks like reading, visual search, or scene perception, only a little time is left for processing of the visual input.

Indirect control combined with inhibition allows initiating saccades independently of a specific processing event. In such a framework, cognitive processing and saccade programming occur simultaneously and mostly independently of each other. In contrast, since saccades can be initiated only after successful processing of the foveated region, the eyes remain at an already processed location for about 150 ms in cognitive trigger theories. Since indirect control theories do not require a saccade target to initiate a new saccade program, processing after the initiation of a new saccade program may be used for eye guidance. In line with this, it has been shown that the target of the next saccade can be modified up to 50 ms before execution of a saccade (Becker and Jürgens, 1979). Thus, additional processing time can easily be obtained by refixating the target area.

² Saccade cancellation was used as an additional process to simulate the pattern of mean values and variances. The role of saccade programming and cancellation in this model will be discussed below.

Core principles of the model

In the next sections, we propose and analyze a framework based on inhibitory control with adaptive timer (ICAT). The fundamental assumption of the ICAT model is that a fine-tuned mixed control mechanism (i.e., a balance of random timing and inhibition) is present in all visuomotor tasks. Such a fine-tuning is essential for the functioning of local control processes, because inhibition can serve the purpose of processing needs only if the random timer is set to a task-sensitive mean value. Three core principles of fixation duration control interact in the ICAT model. The local principles were previously implemented in SWIFT and CRISP but, here, interact with an adaptive global control process.

Local-I: autonomous saccade initiation

Intervals between two subsequent decisions to start saccade programs are generated by a random timer with fixed mean value and standard deviation. This principle provides the main source for the inherent stochasticity.

Local-II: foveal inhibition

Demanding foveal analysis inhibits the random timer in order to delay the next saccade that terminates the current fixation. Foveal inhibition generates immediate effects of ongoing processing on fixation durations but is asymmetric, since it only prolongs the current fixation.

Global: timer adaptation

The mean value of the random timer and the strength of inhibition are modified by processing difficulty experienced during the preceding fixations. For constant processing demands, average fixation durations will be well adapted to processing needs. For varying processing difficulty, the mean value of the saccade timer and the strength of foveal inhibition are updated according to the specific fixation history.

What is the functional relevance of these three principles? While the local-II and global principles are needed to tune the scanning rate of the saccadic system to the cognitive processing difficulty, the local-I principle might be interpreted as a mechanism that randomizes behavior (Carpenter, 1999). Such an interpretation is based on the idea that during the evolution of visuomotor systems, stereotyped responses to upcoming stimuli needed to be avoided, since predictable behavior might have been disadvantageous in predator–prey relations.

Does the random timer concept generalize to other aspects of human motor control? In voluntary interval timing, the random-timer concept was introduced by Wing and Kristofferson (1973; see also Vorberg and Wing, 1996). Interestingly, the addition of a stochastic drift process to the timer

provided a significant improvement of the Wing–Kristofferson model (Collier and Ogden, 2004). From the perspective developed here, such a stochastic drift can be interpreted as a free-running adaptive mechanism that generates random modifications to the timer.

ICAT: a model of saccade initiation intervals

The presentation of our model is divided into two parts. First, we develop a mathematically rigorous implementation of the ICAT principles and illustrate the qualitative behavior with numerical simulations. This section is largely independent of (1) oculomotor aspects of saccade generation and (2) spatial aspects of saccade target selection. Second, we evaluate ICAT in two versions of a visual search task. Simulations in this section are based on a full model of eye movement control considering both temporal and spatial aspects of eye movements (for details about the computational implementation, see the [Supplementary Material](#)). The full model consists of the principles outlined in ICAT, combined with assumptions from an existing model of eye movement control during reading (SWIFT; Engbert et al., 2005). Our simulations demonstrate that ICAT generates a realistic control of fixation durations when spatial control of eye movements is taken into account.

Local-I: a random-walk model of saccade generation

Saccade initiation intervals³ are implemented as a stochastic process, since random-walk models have been used successfully to account for variability of reaction times in behavioral decision processes (Ratcliff, 1978; **for a review on two-choice decisions, see** Smith and Ratcliff, 2004) and eye fixation durations (e.g., Engbert and Kliegl, 2001; Nuthmann et al., 2010). We assume that a random-walk process (Gardiner 1990; van Kampen, 1981) with state $n(t)$ at time t , starting from $n(0)=0$ at time $t=0$, triggers a new saccade program after reaching a threshold N_t at the *first-passage time* t_{fp} with $t_{fp} = \min\{t | n(t) = N_t\}$. In a first step, we implement a one-step process with constant transition probability w over time for a transition from state n to state $n+1$ and vanishing probability for all other transitions. In this case, the random walk is a discrete-state, continuous-time *Markov process*, with exponentially distributed waiting times τ .

$$\rho(\tau) = w \exp^{-w\tau} . \quad (1)$$

³ In the first section, we prefer to use the term *saccade initiation intervals* instead of fixation durations, since the control principles of the ICAT model generate durations between two successive saccade initiations without addressing the programming and execution of saccades. In general, fixation durations depend on saccade initiation intervals. The relation, however, is not deterministic and will be illustrated in detail in the next section.

Exponentially distributed waiting times were sampled by computing a log-transform of a uniformly distributed random number ξ ; that is,

$$\tau = -\frac{1}{w} \log(1-\xi) \quad (2)$$

where ξ is drawn from independent and identically distributed pseudorandom numbers with constant probability over the interval $0 \leq \xi < 1$.

The rise-rate to the threshold N_t is determined by the transition probability \hat{w}_j ,

$$\hat{w}_j = \frac{N_t}{T_j} \quad (3)$$

where N_t denotes the number of states of the random walk of the autonomous timer and T_j is the mean duration of the saccade initiation interval j . Note that the mean timer interval T_j remains constant during the entire interval j and adaptive changes of T_j are relatively slow, as compared with the fast elementary transitions of the random walk. This separation of time scales provides the basis for the computer implementation of local and global control processes discussed below.

Realizations of the random-walk process are illustrated in Fig. 1a. The black line highlights a single run. Each random walk consists of multiple elementary steps with durations randomly drawn from an exponential distribution. In most cases, the random walk remains for a short duration in a given state. However, some long durations can be observed. A random walk ends when a predefined threshold, the maximum of the discrete state variable $n=N$, is reached. As a result, stochastic fluctuations at the level of elementary transitions produce the variance of fixation durations.

Distributions of saccade initiation intervals (or first-passage times of the random walk) for three different thresholds N_t are shown in Fig. 1b. Each distribution consists of 100,000 realizations of the random walk. For all parameter combinations, saccade initiation intervals vary, with a pronounced peak at an intermediate duration. Properties of the distribution change with the number of states N_t . For small N_t s, saccade initiation intervals are more variable and skewed, while large N_t s result in less variable and approximately normal-distributed saccade initiation intervals.

Local-II: foveal inhibition

According to our second principle, saccade initiation intervals can be prolonged by foveal processing. Foveal processing is represented as an activation $a_f(t)$ that changes over time t (Fig. 2a). Before processing, information content of the foveal

region is unknown; after processing, the foveal region has been identified. In both cases, foveal activations $a_f(t)$ will have a value of zero. During processing of the foveal region, activations $a_f(t)$ evolve in two stages. In a first stage, activations rise to a maximum value A_j , which corresponds to the processing difficulty of the foveated region. In a second stage, activations decline until the foveal region is processed. Because cognitive processing can continuously interact with the random walk of the saccade timer, the transition probability is time dependent,

$$w_j(t) = \hat{w}_j h_j[a_f(t)] \quad (4)$$

where $h_j[a_f(t)]$ is the foveal inhibition based on the time-dependent activation $a_f(t)$ in the foveal region during saccade timer interval j .⁴ Throughout the article, we use a very general form of the inhibition function $h_j[\cdot]$; that is,

$$h_j[a_f(t)] = \exp\left\{-\alpha\left(\frac{a_f(t)}{A_j}\right)^\beta\right\} \quad (5)$$

where the two free parameters, $\alpha > 0$ and $\beta > 0$, determine the shape of the inhibition function and the expected processing difficulty A_j of timer interval j scales the strength of inhibition. Values of the inhibition function $h_j[\cdot]$ range from 0 to 1, so that foveal inhibition by Equation 4 decreases the transition probability (as compared with the baseline \hat{w}), resulting in prolonged intervals of the saccade timer.

Putting together Equations 4 and 5, we obtain the time dependence of the random walk's transition probability:

$$w_j(t) = \frac{N_t}{T_j} \exp\left\{-\alpha\left(\frac{a_f(t)}{A_j}\right)^\beta\right\} \quad (6)$$

Modulations of the transition probability by foveal inhibition are illustrated in Fig. 2b. The tail of the inhibition function $h_j[a]$ increases with increasing expected processing difficulty A_j . The solid line illustrates inhibition, when saccade timing is adjusted to a low value A_1 . Foveal activations a_f below A_1 produce only minor modulations. However, when activations exceed the expected processing difficulty, $a_f(t) > A_1$, foveal inhibition decreases the rate considerably. If the expected processing difficulty is much smaller than the actual foveal activation—for example, $a_f(t) > A_3$ —the rate drops asymptotically toward zero, $h_3(a_f) \approx 0$. Two additional lines are depicted in Fig. 2, where foveal inhibition is adjusted to medium processing difficulty A_2 (dotted line) or high processing difficulty A_3 (dashed line). In both cases, activations below the adjusted value produce weak rate modulations, whereas

⁴ Note that, in our model, the maximum of the activation $a_f(t)$ reflects processing difficulty of the foveal item (see Engbert et al., 2005). A more detailed definition of the activation is given in the Supplementary Material.

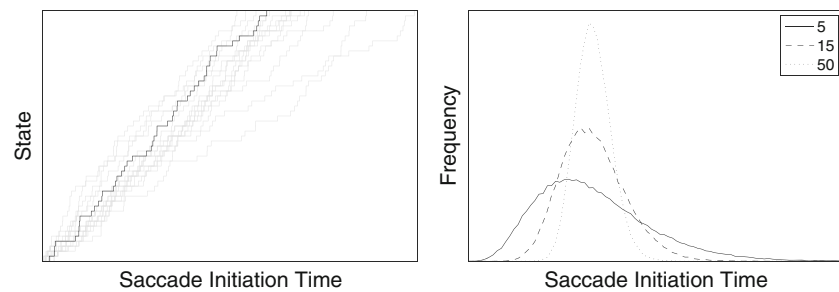


Fig. 1 Random-walk model for saccade timing. **a** Examples for realizations of the random walk with threshold value $N=50$. The random walk starts at $n(0)=0$ and terminates at the threshold value $n(t_p)=N$, which is

reached after the first-passage time t_{fp} . **b** Distribution of the relative frequency for three different thresholds $N = 5, 15, 50$. Each distribution represents 100,000 realizations of the first-passage time t_{fp}

activations exceeding the expected processing difficulty show noticeable inhibition values.

Global: adjustment of saccade timer

Because the third principle of our model is derived from a different notion than local control of fixation durations, we reiterate its motivation in short form. There is experimental support (Harris, Hainline, Abramov, Lemerise and Camenzuli, 1988; Hooge and Erkelens, 1998; Kolars, 1976) for the notion of random timing (local-I); that is, saccades are initiated after a random time interval. However, random timers pose the problem of how the mean value of a timer is determined. In general, it is presumed that, on average, saccades are initiated after a time interval adjusted to overall task demands (Kolars, 1976) or adjusted to the estimated foveal processing time of previously fixated stimulus elements (Hooge and Erkelens, 1998). Thus, the random timer is based on expected or previously experienced processing difficulty. In the following, we implement the latter principle, since there is experimental support for this concept from visual search tasks (Hooge and Erkelens, 1998).

In the ICAT model, saccade timing is updated gradually after each saccade initiation. Two parameters are affected by this adjustment. First, the time interval j between the initiation of two saccade programs has a predefined average duration T_j . This interval duration increases with increasing processing time of the previous fixations and is set to match the time needed to extract sufficient visual input from the fixated region. Second, the expected processing difficulty A_j reflects the anticipated maximum foveal activation $a_f(t)$ during the next timer interval j . The processing difficulty A_j is needed to scale the strength of foveal inhibition. We assume that both parameters depend on fixation history, which can be formulated as a discrete mapping of the form

$$A_{j+1} = g_A(A_j, A_{j-1}, \dots, A_{j-(d-1)}) \quad (7)$$

$$T_{j+1} = g_T(A_{j+1}). \quad (8)$$

Thus, change of A_{j+1} represents an autoregressive process of order d defined by the function $g_A(\cdot)$, while the saccade

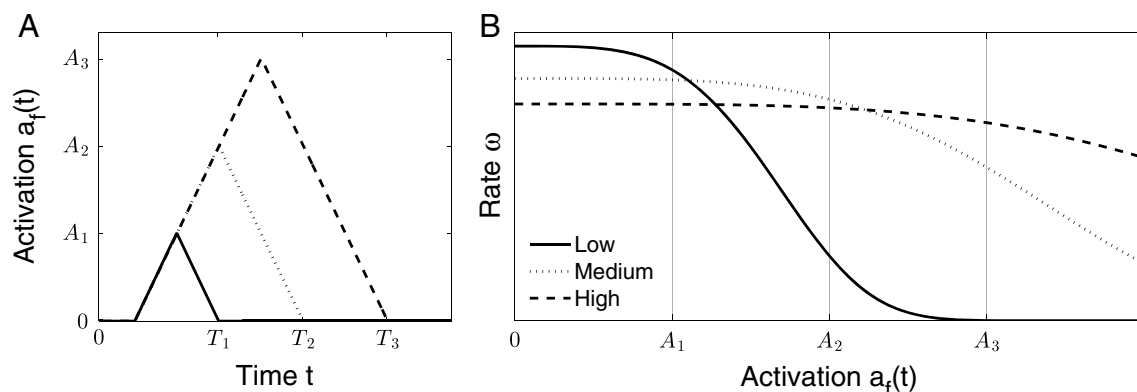


Fig. 2 **a** Temporal evolution of three activations with varying maximum processing difficulty (A_1, A_2, A_3). In our examples, activations remain zero until the onset of processing. In the beginning, processing activations evolve identically, but activations grow higher and processing lasts longer with increasing processing difficulty. **b** Foveal inhibition for three

different processing difficulties A_1, A_2 , and A_3 . For example, given a high foveal activation $a_f(t) \approx A_3$, the strength of the inhibition increases for processing difficulties A_1 to A_3 . Note that the time dependence of $w_f(t)$ is implicit, due to variation of the foveal activation $a_f(t)$

timer interval T_{j+1} is determined by a nonlinear mapping $g_T(\cdot)$ from the actual value of the expected processing difficulty. When the estimation is based on a large number of fixations d , the adaptivity of the timing process will be small, and average fixation durations will be relatively stable. Thus, timers with no variation in the mean timing interval are included in our model for $d \rightarrow \infty$.

In the simulation studies, as well as the experimental validation of our model, adaptation is an autoregressive process of order $d=1$, where A_{j+1} and T_{j+1} depend solely on the difficulty of the last fixated item. To ensure that processing has encountered the new maximum difficulty, activations in our simulation had to be in the second processing stage or later (declining activations in Fig. 2a) before saccade timing could adapt to a new expected difficulty. Otherwise, the expected difficulty remained unchanged.

Qualitative behavior of the saccade timer model

Before we discuss more details of our model of saccade generation, we explore the qualitative behavior of the saccade timing model and investigate the interplay of autonomous saccade initiation, foveal inhibition, and saccade timer adjustments. We use well-defined situations to examine the range of behaviors that can be observed when processing difficulty changes. In each of the presented examples, we illustrate the model's behavior over a sequence of five subsequent saccade initiations, in which processing demands change during the central saccade initiation. We describe single-trial simulations and discuss the time course of average saccade initiation intervals, as well as distributions of saccade initiation intervals. All parameter combinations used in our simulation studies are shown in Table 1.

Increasing processing demands

In Fig. 3a, we demonstrate the effect of increasing processing demands in a single run of the simulation. Effects can be observed on three levels: (1) foveal processing, (2) rate modulations of the random walk, and (3) the realized random walk. Foveal analysis corresponds to processing during a task and strongly depends on task-specific assumptions. In our model, low activations generally indicate low processing difficulty, and activations increase with increasing processing demands. High processing demands will generate both high amplitudes of activations and long periods of high activations (see Fig. 2a). In our example, processing demands are low during the first two saccade initiations ($n-2$, $n-1$), increase during the n th saccade initiation, and remain high on the last two saccade initiations ($n+1$, $n+2$). As a result, activations of foveal processing are relatively small during the first two saccade initiations and increase afterward (solid line in Fig. 3a, upper panel). Since saccade timing is adjusted to

low processing demands (dashed line in the upper panel), almost no rate modulation is observed during the first two saccade initiations (central panel). An increase of processing demands during the n th saccade initiation immediately reduces the rate of the random walk. After adjustment of the saccade timer to higher processing demands at the beginning of saccade initiation $n+1$, overall rate decreases, and modulations by foveal inhibition vanish.

Since each simulation is a stochastic realization of our model, we generated 10,000 simulations using the same parameter combinations to assess overall performance (Fig. 3b). Mean saccade initiation intervals are shortest during the first two saccade initiations, where saccade timing is adapted to low processing demands, and longest during the last two saccade initiations, where saccade timing is adjusted to high processing demands. When processing difficulties increase during the n th saccade initiation, saccade initiation intervals lengthen, on average. For this parameter combination, the prolongation of saccade initiation intervals by foveal inhibition is only a fraction of the increase in saccade initiation intervals observed after readjustment of the saccade timer. The distributions are shifted toward longer mean saccade initiation intervals (Fig. 3c).

Decreasing processing demands

In the next example, we examine saccade timing in the opposite case of decreasing processing difficulty (Fig. 4). The time courses of foveal analysis, expected processing demands, rate modulation, and corresponding random walks are shown in Fig. 4a. Activations resulting from foveal analysis reflect high processing demands during the first two saccade initiations and decrease during the n th saccade initiation. Since saccade timing is adjusted to high processing demands, almost no rate modulation is observed during the first two saccade initiations. A further decrease of processing demands during the n th saccade initiation does not affect rate. Hence, the observed rate remains almost constant during the first three saccade initiations. At the beginning of saccade initiation $n+1$, saccade timing is adjusted to lowered processing demands, and the rate of the random walk increases.

Mean saccade initiation intervals evolve correspondingly (Fig. 4b). Durations are longest when saccade timing is adjusted to high processing demands. When processing demands decrease, mean saccade initiation intervals are not immediately reduced. Only after a delayed adjustment of saccade timing at the beginning of saccade initiation $n+1$ do durations decrease significantly. Distributions of saccade initiation intervals are identical for high and low processing demands, when saccade timing is adjusted to high processing demands (Fig. 4c). After adjusting saccade timing to lower processing demands, the distribution shifts toward shorter saccade initiation intervals.

Table 1 Model parameters for qualitative simulations (columns with multiple values refer to symbols with low/high processing demands)

Simulation	Processing			Saccade Timer			
	Maximum A_{low}/A_{high}	Onset ^a	Rate ^b	States N_t	Duration ^a T_j	Foveal α	Inhibition β
Figs. 3 & 4: Single trial	40/70	50	0.66	50	300/340	0.03	6.00
Fig. 5: Foveal inhibition I							
- Delayed adjustment (solid line)	40/70	50	0.66	50	300/340	0.001	6.00
- Partial adjustment (dashed line)	40/70	50	0.66	50	300/340	0.03	6.00
- Perfect adjustment (dotted line)	40/70	50	0.66	50	300/340	0.06	6.00
- Overcompensation (dash-dotted)	40/70	50	0.66	50	300/340	0.10	6.00
Fig. 6: Foveal inhibition II							
- Delayed adjustment (solid line)	40/70	50	0.66	50	300/340	0.06	6.00
- Partial adjustment (dashed line)	40/70	50	0.66	50	282/310	0.40	1.40
- Perfect adjustment (dotted line)	40/70	50	0.66	50	255/262	0.60	0.25
Figure 7: Adaptation	40/70	50	0.66	50	300/340	0.06	6.00
Equation(s)	3 & 7			3	3 & 8	5	5

^a All durations are presented in milliseconds.

^b Activation change per millisecond during processing

Local control: modulations by foveal inhibition

Since foveal inhibition depends both on the time course of activations and on the set of parameters, a broad range of behaviors can be observed when processing demands change. The relationship between foveal inhibition and average

saccade initiation intervals is illustrated for four parameter combinations in Fig. 5. The left panel of Fig. 5a displays rate modulations generated by foveal inhibition when saccade timing is adjusted to low processing demands. The equivalent curves for saccade timing adapted to high processing demands are shown in the right panel. For low activations, rates are

Fig. 3 Illustration of saccade timer model: Processing demands increase during the n th saccade initiation. **a** Levels of the model: Foveal analysis and expected processing demands (top panel: solid and dashed lines, respectively), rate modulations (central panel), and the realized random walk (bottom panel). **b** Mean saccade initiation intervals before ($<n$), during (n), and after ($>n$) the critical fixation. **c** Distribution of saccade initiation intervals before ($<n$), during (n), and after ($>n$) the critical fixation

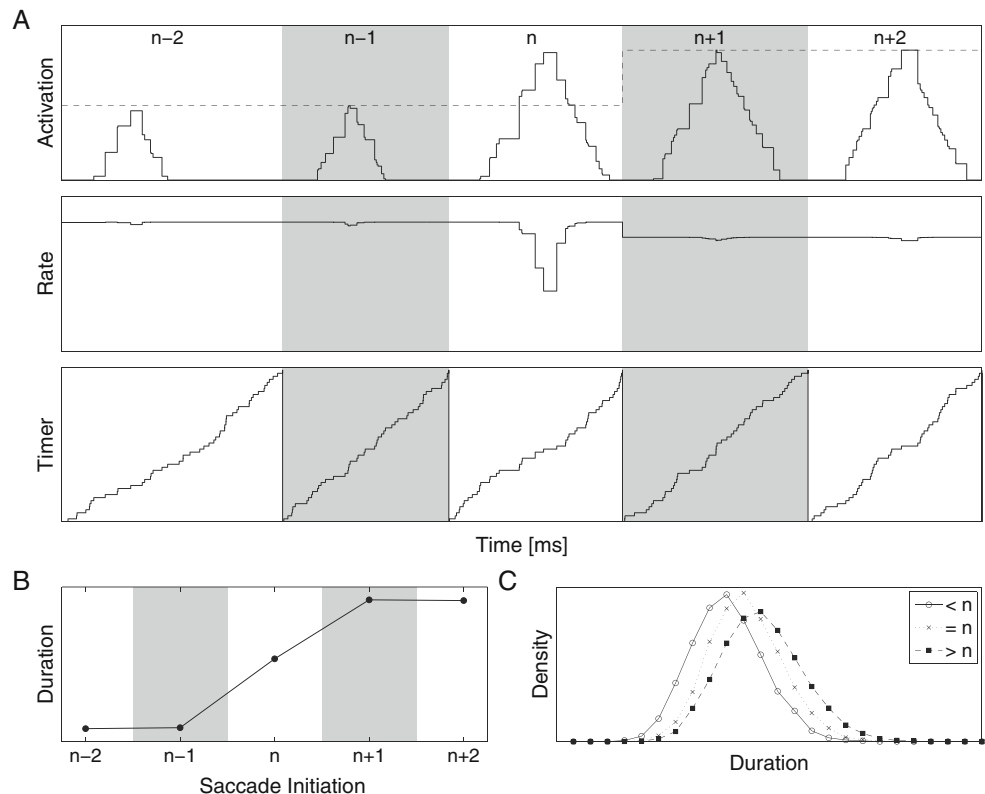
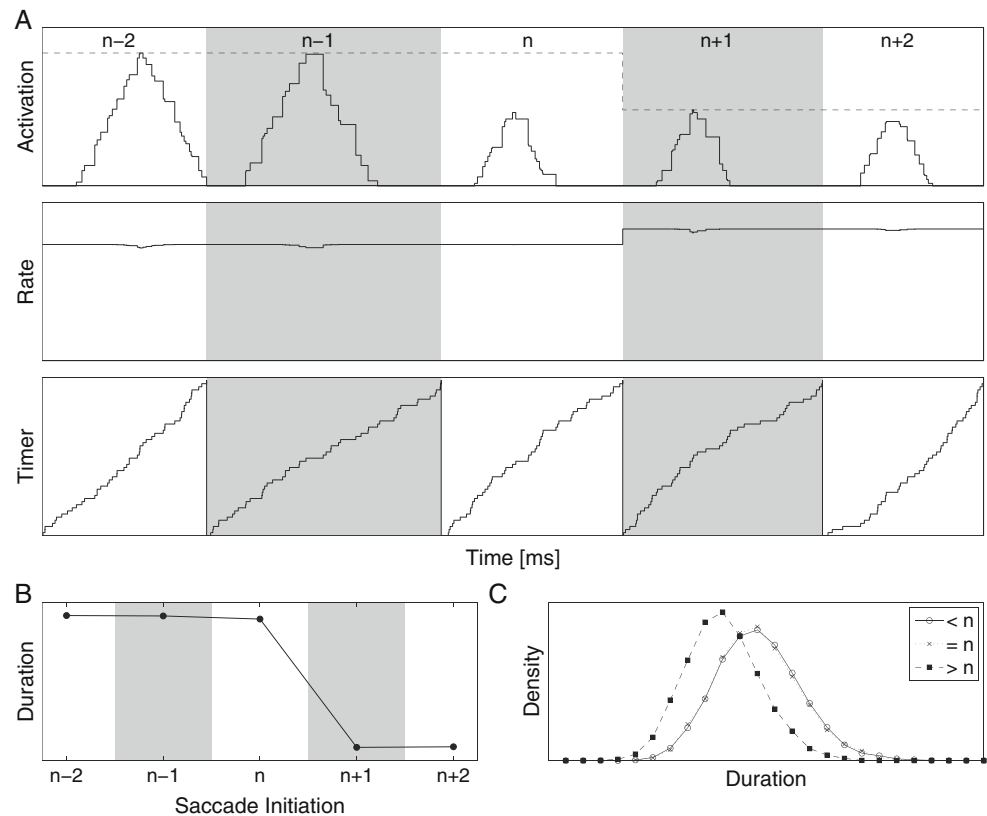


Fig. 4 Illustration of saccade timer model: Processing demands decrease during the n th saccade initiation. **a** Levels of the model: Foveal analysis and expected processing demands (top panel: solid and dashed line, respectively), rate modulations (central panel), and the realized random walk (bottom panel). **b** Mean saccade initiation intervals before ($<n$), during (n), and after ($>n$) adjustment. **c** Distribution of saccade initiation intervals before ($<n$), during (n), and after ($>n$) adjustment



higher when saccade timing is adapted to low processing demands (left panel), as compared with when saccade timing is adapted to high processing demands (right panel). Thus, elementary steps will, on average, be shorter when saccade timing is adapted to low processing demands.

In both panels of Fig. 5a, activations within the white area evolve from processing demands below the expected maximum, while activations in the gray area emerge when processing demands are higher than expected. Obviously, when activations are below the threshold for which they are adapted, rates are almost constant and barely differ between parameter combinations (white area). However, strong rate modulations are observed when activations exceed the threshold (gray area). The exact characteristics depend on the parameters chosen and range from an almost unaffected rate (solid line) to strong modulations (dash-dotted line). When saccade timing is adapted to high processing demands, neither low nor high activations induce modulations of the random walk's rate. Nonetheless, some parameter combinations are more sensitive to increasing activations and show faint modulations for very high activations.

For each parameter combination, average durations of five successive saccade initiations are displayed in Fig. 5b. Mean saccade initiation intervals are based on 10,000 simulations, and the time course of activations used in the simulations corresponds to the time course of activations in the single-run simulations (Figs. 3 and 4). Across different parameter

combinations, average saccade initiation intervals are similar before a change ($\leq n-1$) and after adjusting the saccade timer to new processing demands ($\geq n+1$). When processing demands increase (left panel), durations increase according to the strength of foveal inhibition during the n th saccade initiation interval. The increase can be compared with later well-adjusted saccade initiation intervals ($\geq n+1$). When foveal inhibition is absent (solid line), mean saccade initiation intervals remain unaffected, and a delayed adjustment of saccade initiation intervals is observed. With stronger foveal inhibition, saccade initiation intervals increase. For some parameter settings, durations increase somewhat (dashed line). In addition, a perfect adjustment can be seen (dotted line), or the prolongation might even be stronger than expected by well-adjusted saccade timing (dash-dotted line). Even though mean saccade initiation intervals might resemble adjusted saccade timing for some parameter combinations, the rate of a random walk is differentially affected during the n th saccade initiation and later well-adjusted saccade timing. For decreasing processing demands, evolution of average saccade initiation intervals is identical for all parameter combinations. The duration of the n th saccade initiation interval remains long and decreases afterward ($\geq n+1$). Such an asymmetric control of fixation durations—that is, an immediate increase and a delayed decrease—has been reported during reading (Kennison and Clifton, 1995) and visual search (Hooge et al., 2007). The increase, however, varied between experiments. While

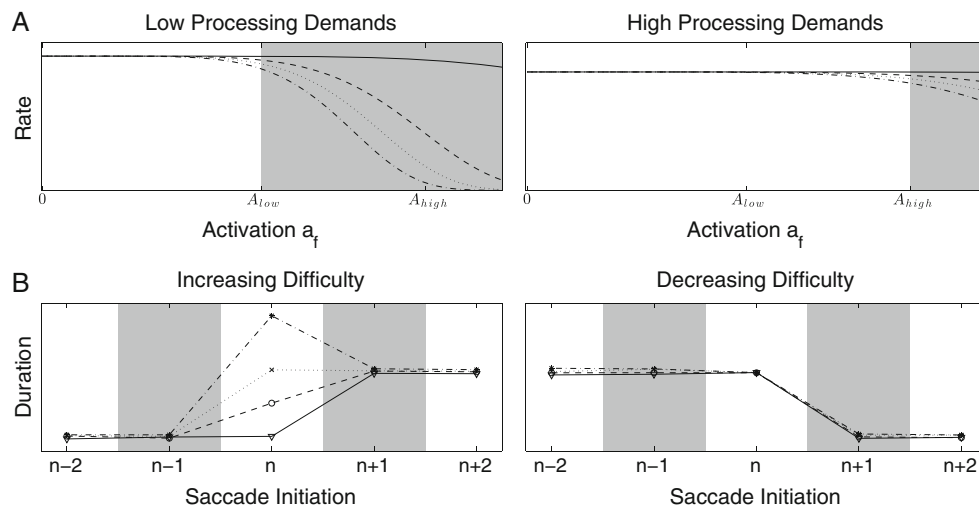


Fig. 5 Foveal inhibition: Increasing processing difficulty. Rate modulations and mean saccade initiation intervals of four parameter combinations. **a** Rate modulations when saccade timing is adjusted to low processing demands (A_{low} ; left panel) and high processing demands (A_{high} ; right panel). In both plots, the gray area shows activations above the expected maximum. **b** Mean durations over five successive saccade

initiations when processing demands increase (left panel) and decrease (right panel). Four different forms of foveal inhibition are observed when processing demands increase during the n th saccade initiation: Delayed adjustment (solid line), partial adjustment (dashed line), perfect adjustment (dotted line), and overcompensation (dash-dotted line)

Kennison and Clifton observed some sort of overcompensation, Hooge et al. reported a partial adjustment.

In the previous example, we demonstrated a range of qualitative behaviors on saccade initiation intervals for increasing processing demands, while the temporal evolution of saccade initiation intervals was identical for decreasing processing demands. Next, we present three parameter combinations that differentially affect saccade initiation intervals when processing demands decrease (Fig. 6). The solid line resembles rate modulations of the previous examples. The rate is almost unaffected by activations below the threshold to which saccade timing is adjusted (Fig. 6a: left panel, white area; right panel, entire area). Activations exceeding this threshold cause strong rate modulations (left panel, gray area). Saccade initiation intervals for this parameter combination increase immediately when processing demands increase and remain long during the n th saccade initiation when processing demands decrease (Fig. 6b). The dashed line shows strong modulations for the entire range of activations. Rate modulations are almost identical for saccade timing adjusted to low and high processing demands. In both cases, the rate is high when processing demands are minimal and decreases rapidly when activations increase. Rate modulations are insensitive to the height of activations and quickly reach an asymptote. Since the duration of heightened activations is longer for high processing demands (see Fig. 2a), saccade initiation intervals immediately adapt to new processing demands both when difficulty increases and when it decreases. Such parameter combinations will be estimated when fixation durations are controlled by a cognitive trigger. The third parameter combination (dotted line) shows rate modulations across the entire range of activations. The modulation,

however, is stronger when processing is adjusted to low processing demands. For this parameter combination, average saccade initiation intervals shorten immediately when processing demands decrease. As compared with well-adjusted saccade initiation intervals, the reduction is only partial. It is important to note that all parameter combinations used in Fig. 6 led to a similar prolongation of fixation durations when processing demands increased. In the first case, foveal inhibition affected a few time steps strongly (solid line). In the second case, foveal inhibition affected almost all time steps during processing, but to a much weaker degree (dashed line). And the third case represents a mixture of the two other cases (dotted line). Thus, even though fixation durations increased by a similar amount, the prolongation was caused by only a few very long time steps (solid line), by many moderately prolonged time steps (dashed line), or by a mixture of both (dotted line).

Global control: temporal adjustment of random timing and foveal inhibition

In the preceding examples, we focused on local effects of foveal inhibition for various parameter combinations. However, an important concept in our model is the continuous adjustment of saccade timing, which has been assumed only implicitly in previous indirect control theories. In our model, adjustments depend on expected processing demands (Equation 7) and affect saccade timing at two levels. First, saccades are initiated autonomously, but the average duration T_j of this random time interval increases with increasing expected processing demands (Equation 8). Second, strength of foveal inhibition scales with expected processing demands A_j

(Equation 6). Here, we investigate the functional role of both adjustments by deactivating the adaptive processes in our model simulations. For deactivated processes, parameters remained constant for the entire simulation—that is, $T_{j+1} = T_j$ (cf. Equation 8) and $A_{j+1} = A_j$ (cf. Equation 7). In summary, we simulated four versions of our model: the complete model with two active adaptive processes, a model without adaptation, and two models where only one of the processes adapted. Since fixation durations across tasks can be described by asymmetric control, we use the same parameters as in Fig. 5 (perfect adjustment). With two adaptive processes, rate modulations behave as illustrated earlier (Fig. 7a, solid line). When saccade timing is adapted to low processing demands (left panel), the rate of the random walk is modulated by activations above the adjusted level (gray area). When saccade timing is adapted to high processing demands (right panel), the overall rate is reduced and remains unmodulated by a larger range of activations (white area). Corresponding saccade initiation intervals show an asymmetric control pattern with immediate prolongations and delayed reductions, when processing demands change (Fig. 7b).

Without adaption (Fig. 7, dash-dotted line), rate modulations are independent of previous processing demands, which leads to identical curves in the left and right panels. Since high activations always cause rate modulations in the model, the influence of foveal inhibition is symmetric, and fixation durations immediately increase and decrease, when processing demands change. Hence, saccade timing without adaptation resembles control by cognitive triggers.

In the next simulation, the mean timer interval T_j adapts without simultaneous adaptation of foveal inhibition (Fig. 7, dashed line). Rates of the random walk are generally reduced when saccade timing is adapted to high processing demands (right panel), and rate modulations are strong both when adapted to low and when adapted to high processing demands. As a consequence, average saccade initiation intervals are symmetrically modulated in two steps. In a first step, foveal inhibition immediately prolongs and reduces saccade initiation intervals (saccade initiation n). In a second step, saccade initiation intervals are further modulated, since the autonomous timer adapts with a temporal delay (saccade initiation $n+1$).

Finally, if the scaling factor of foveal inhibition A_j adapts without adaptation of the timer (dotted line), rate modulations are observed only when processing is adjusted to low processing demands. As a consequence, only a single fixation is prolonged when processing demands increase, and no fixation when processing demands decrease. As revealed by our simulations, adjustments of the mean saccade timer interval T_j lead to delayed and sustained changes in saccade initiation intervals, while adjustments of the scaling factor A_j determine the local prolongation of single saccade initiation intervals.

Asymmetric control of saccade initiation is generated only when both processes adapt simultaneously over time.

Immediacy and lag effects

The majority of eye movement studies focused on average durations of first fixations, single fixations, and gaze durations (Rayner, 2009). As was demonstrated above, our model is able to generate differences in mean fixation durations via foveal inhibition and, thus, is generally in agreement with immediate effects of the current fixation location on corresponding durations. Simulations of immediacy effects in reading can be found in the Supplementary Information. Most interesting, even multimodal distributions are well captured by the model. Lag effects, where processing difficulty of the fixated object spills over to the next fixation, have also been observed (Rayner and Duffy, 1986). During reading, fixations on word $n+1$ are longer when the preceding word n is a low-frequency word, as compared with a high-frequency word (Henderson and Ferreira, 1990; Kennison and Clifton, 1995; Schroyens et al., 1999). Interestingly, the lag-frequency effect—that is, the modulation of the fixation duration on word $n+1$ —is sometimes even larger than the immediacy effect on word n (Kliegl, Nuthmann, and Engbert 2006; Schad, Nuthmann, and Engbert 2010). Lag effects in ICAT may be generated by two mechanisms. First, if processing has advanced sufficiently, the saccade timer is adjusted to previous processing demands, which, in turn, affects subsequent fixation durations. Second, if processing is slower, foveal inhibition may have a delayed impact on the next fixation. Depending on the exact time course of processing, the current fixation, the next fixation, or even both may be prolonged. Hence, our model generates effects on subsequent fixations, as observed in gaze durations, and accounts for shifts of effects from first-fixation durations to later fixations—for example, when preview of an item is prevented (Inhoff and Rayner, 1986; Reingold et al., 2012; Sereno and Rayner, 2000). In general, mean fixation durations of one or more successive fixations may be replicated by the ICAT model.

Labile saccade programming

Until now, we focused on the duration between the initiation of two adjacent saccade programs, while neglecting the role of saccade programming and execution. In ICAT, a random timer is constantly active, and saccade programs are initiated whenever a certain threshold is reached. Saccade programs are often assumed to consist of two stages: a labile stage, which can be subject to cancelation, and a nonlabile stage, which is resistant to further changes. The labile stage has a mean duration T_{lab} and is canceled if a second saccade program is initiated during this stage. The canceled saccade program is replaced by a new labile saccade program, which, in turn, may

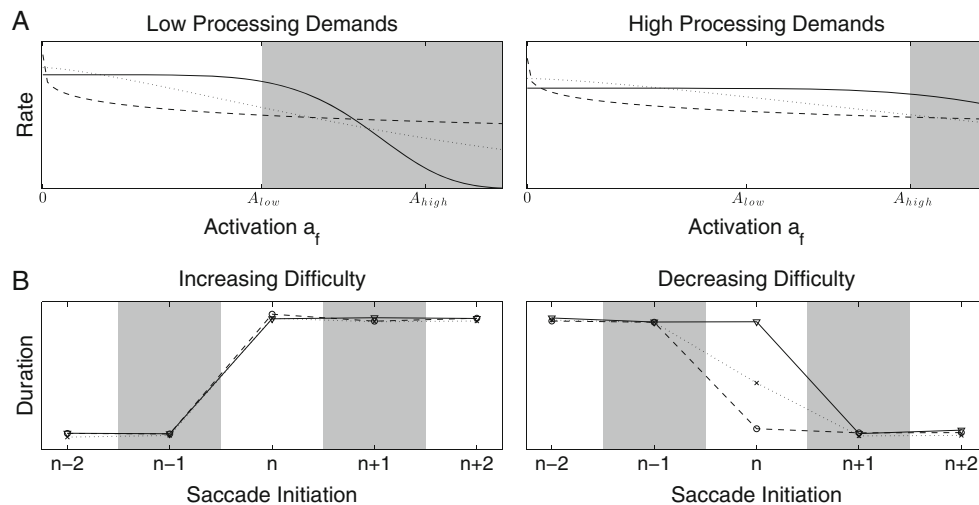


Fig. 6 Foveal inhibition with decreasing processing difficulty: Rate modulations and mean saccade initiation intervals of three parameter combinations. **a** Rate modulations when saccade timing is adjusted to low processing demands (A_{low} ; left panel) and high processing demands (A_{high} ; right panel). In both plots, the gray area shows activations above the expected maximum. **b** Mean durations over five successive saccade

initiations when processing demands increase (left panel) and decrease (right panel). Three different forms of foveal inhibition are observed when processing demands decrease during the n th saccade initiation: delayed adjustment (solid line), partial adjustment (dotted line), and perfect adjustment (dashed line)

be canceled by another new saccade program. Only after transition from the labile stage to the nonlabile stage is a saccade imminent and the current fixation terminated.

The main motivation for two stages of saccade programming derives from the double-step paradigm in saccade generation (Becker and Jürgens, 1979), which revealed that saccades to a first saccade target can be canceled by the appearance of a second saccade target up to 250 ms after presentation of the first. Later presentations led to a sequence of fixations on both targets. In our model, this is captured by a “point of no return” at the transition from a labile to a nonlabile stage. The assumption of two stages of saccade programming in models

of eye movement control during reading was introduced by Reichle et al. (1998) and has been applied to other models of eye movement control during reading (Engbert and Kliegl, 2001; Engbert et al., 2002) and scene perception (Nuthmann et al., 2010). While the assumption of discrete processing stages during saccade programming has been very influential in models of eye movement control, there is some evidence for continuous flow of information in the oculomotor system (Bichot, Rao and Schall, 2001).

In an earlier section, we presented the concept of a random walk to generate intervals between two saccade initiations. Here, we take advantage of the stochastic simulation of

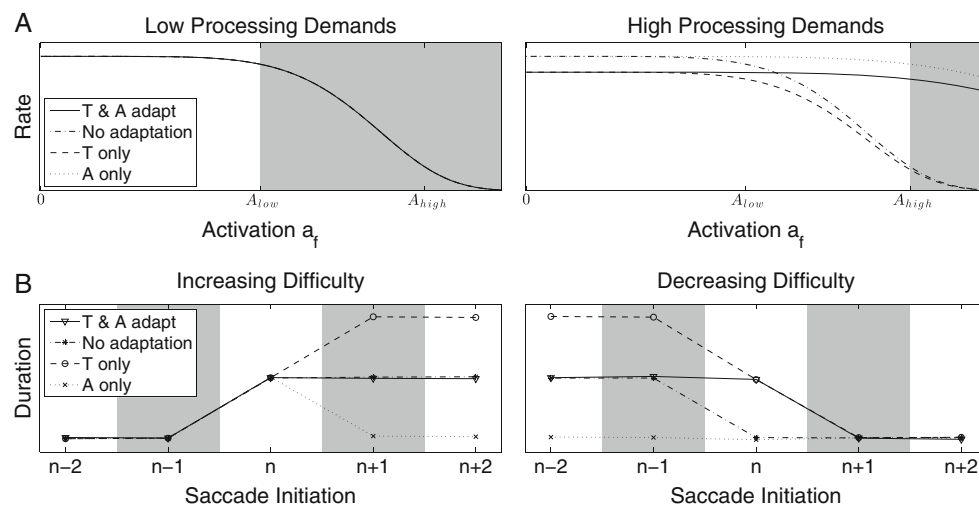


Fig. 7 Saccade timer adjustments: Role of adjusting the average saccade timer interval T_f and the expected processing difficulty A_f . **a** Rate modulations when saccade timing is adjusted to low processing demands (A_{low} ; left panel) and high processing demands (A_{high} ; right panel). In both plots,

the gray area shows activations above the expected maximum. Note that the four functions are superimposed on each other in the left panel. **b** Mean durations over five successive saccade initiations when processing demands increase (left panel) and decrease (right panel)

Markov processes (Gillespie, 1978) to implement a coherent framework of stochasticity at multiple levels. Each component of saccade control—that is, saccade initiation, labile and nonlabile saccade programs, and saccade execution—can be described by separate random-walk processes. In order to get a better estimate of fixation durations, we added a random walk for the labile saccade programming stage that can be canceled by the initiation of a new saccade program. Durations of nonlabile saccade programs and durations to execute saccades depend on factors like saccade amplitude and were excluded at this level.

The dynamical state of the ICAT model is defined by a vector $S_m = (m_1, m_2)$ representing the states of two random walks—that is, $m_1 = 0, 1, 2, \dots, N_t$ for saccade timing and $m_2 = 0, 1, 2, \dots, N_o$ for the labile saccade program. Two transitions are possible from each state $S_m = (m_1, m_2)$ to state $S_n = (m_1 + 1, m_2)$ or $(m_1, m_2 + 1)$. Thus, while multiple processes may be active simultaneously, each transition affects only one random walk.

Mathematically, we have to compute the transition probability W_{nm} of the model from state S_m at time t , having arrived there at time $t - \tau$, to an adjoined state S_n . Following Gillespie (1978), we can sample a realization of the time step τ from the total transition probability W_m . The time step is drawn from an exponential waiting time distribution (see Equation 2)—that is,

$$\rho(\tau) = W_m \exp\{-W_m \tau\} \quad \text{with} \quad W_m = \sum_n W_{nm}, \quad (9)$$

where W_m is the sum of all transition rates W_{nm} from the current state S_m to an adjoined state S_n . Since saccade initiation and labile saccade programming are two independent one-step processes, the total transition probability W_m at time t is given by

$$W_m = w_j + w_{lab} \quad (10)$$

where w_j is the time-dependent transition probability of the random walk implemented in the ICAT model of saccade timing (see Equation 5) and w_{lab} corresponds to the transition probability of the labile saccade program.

While the transition rate w_j of the saccade timer is always nonzero, the random walk of the labile stage is active only after saccade initiation. When the random walk of the saccade timer reaches a threshold, the labile program starts, $w_{lab} > 0$, and terminates when reaching the oculomotor threshold, $w_{lab} = 0$. The rate of the random walk is given by

$$w_{lab} = \begin{cases} \frac{N_o}{T_{lab}} & \text{during labile stage} \\ 0 & \text{otherwise} \end{cases} \quad (11)$$

where N_o is the number of states of oculomotor random walks and T_{lab} denotes the average duration of the labile stage. If the

labile stage is canceled by a new saccade program, the state of this random walk begins anew—that is, $m_2 = 0$.

After sampling a time step τ , a transition has to be selected. Probabilities of possible transitions are given by relative transition probabilities (Gillespie, 1978)—that is,

$$p_n = \frac{W_{nm}}{W_m}. \quad (12)$$

In summary, the algorithm proposed by Gillespie (1978) allows one to simultaneously simulate multiple independent random walks (for a further extension, see the Supplementary Material). The computation is performed in three iterative steps. First, a total transition probability W_m is computed by summing the rates of all random walks. Second, the time until the next transition τ is determined by drawing a pseudorandom number from an exponential distribution (Equation 9). Since the exponential distribution and corresponding random number depends on the total transition probability, time steps τ decrease with increasing number of active random walks. Third, only one random walk moves during each transition. Probability of a step is given by relative transition probabilities p_n of each random walk (Equation 11). After a transition, the process starts anew. When a random walk reaches a threshold, random walks are reset and may even be stopped. The procedure ensures that distributions of durations for each class of random walks are independent and correspond to the rates used during the simulation.

A histogram of 1,000,000 simulated fixation durations is shown in Fig. 8 (bold line). As in regular fixation durations, the distribution has a pronounced peak and is skewed to the right. The rightward skew is generated by fixation durations that have been prolonged by one or more cancellations (thin lines). While fixation durations with no cancellations of the labile stage are shortest and have the smallest variance (solid line, $N = 913,953$), average fixation duration and variance increases with increasing number of cancellations. Overall, 9% of fixation durations were affected by cancellations. As can be seen from the plot, long fixation durations are primarily generated by saccade cancellations. In agreement with this, simulations in the Supplementary Information were also run without a cancellation mechanism. While simulations captured effects in mean fixation durations, long fixation durations were generally underestimated. Cancellations of existing saccade programs seem to be an important factor for the variability in fixation durations.

Combining temporal control in ICAT with spatial eye movement control

Thus far, we have focused on the temporal control of eye movements. For a more rigorous validation of ICAT, we incorporated the principles for the temporal control of eye

movements with a set of rules for the spatial control of eye movements. These rules were taken from a model of eye movement control during reading (i.e., SWIFT; Engbert et al., 2005). An overview of the full model architecture is given in Fig. 9. Saccade initiation is controlled by the ICAT principles, illustrated in the leftmost part of the figure. Saccades are triggered by an autonomous saccade timer (local-I principle). After a random time interval, a new saccade program is initiated, which consists of two stages. During the labile programming stage, saccades may still be modified or even canceled. Note that foveal inhibition affects both the autonomous saccade timer and the programming of saccades during the labile programming stage. When passing into a nonlabile programming stage, a saccade target is selected, and saccade execution is inevitable. Saccade targets will be chosen from an activation field defined as a set of activations. The activation field represents the processing of the visual input. Temporal dynamics in the activation field strongly depend on the current fixation position and change after each saccade correspondingly. As was stated earlier, foveal processing feeds back to the ICAT model. Thus, foveal activations are used to inhibit the initiation of new saccades (local-II principle) and adjust saccade timing to new processing demands (global principle). The full model generates a sequence of fixations with realistic temporal control and spatial control—that is, fixation durations and fixation locations. We limit the description to the full model, since the focus of this work was on the temporal control of eye movements. A detailed description of the full model can be found in the Supplementary Material.

Experimental validation

Using two variants of a simple scanning task (for a related task, see Hooge and Erkelens, 1998), we investigated the control of fixation durations in our model when equipped with a realistic oculomotor machinery. We limit our presentation to the experimental results and simulations of fixation durations. A detailed description of the full model, as well as spatial simulation results, can be found in the Supplementary Material.

In the first experiment, participants searched for a ring in an array of Landolt-Cs. Fixation duration adjustments were investigated with a stepwise change of processing demands during the trial. In the second experiment, the same set of stimuli was used in a gaze-contingent task to limit fixation duration adjustments to foveal processing. In both tasks, we examined the model's performance on mean fixation durations, as well as fixation duration distributions. In addition, we explored fixation durations before different saccade types in Experiment 1. All analyses reported here were limited to target-absent trials.

Task

Participants were instructed to scan sequences of 11 symbols from the left to the right on a computer display and to search for a target stimulus (see the Supplementary Material for experimental details). We used a ring as target and Landolt-Cs as distractors. The sequence of symbols in each condition is displayed in Fig. 10. We varied target–distractor similarity by gap size and expected higher fixation durations on symbols with small gaps, due to higher processing difficulty. As baseline conditions, we measured eye movements when the entire sequence was composed of distractors with large gaps (low difficulty) or small gaps (high difficulty). In the remaining conditions, gap size changed at an intermediate position of the sequence. As a result, participant's performance had to adapt from distractor difficulty faced with at the beginning of a trial to the difficulty presented after the stepwise change. To reduce its predictability, changes occurred between the fifth and eighth symbols of a sequence. In our analysis, trials were merged relative to this change. In the remainder, we will refer to *relative position 1* as the first symbol with new gap size.

Experiment 2 was the same as Experiment 1, except for a gaze-contingent display manipulation (McConkie and Rayner (1975). On each trial, the fixated symbol was visible to a participant, while all other symbols were masked by Xs. As soon as the eyes moved to the next stimulus, the previously fixated symbol was masked, and the newly fixated symbol revealed. The gaze-contingent display technique was used to analyze the specific influence of foveal processing on the adjustment of fixation durations, since preview of upcoming symbols was prevented. By applying this manipulation, modulations of fixation durations can unambiguously be attributed to fixated stimulus elements.

Experiment 1: stepwise processing change during visual search

Mean fixation durations

Average durations of the first fixation on a stimulus are plotted in the left panel of Fig. 11. Stimulus position 1 corresponds to the first distractor with new gap size in conditions with varying processing demands (cf. Fig. 10, fixation sequence). Since the absolute position of a change varied across trials, fixation durations in baseline conditions were selected accordingly. First-fixation durations are typically higher on more demanding stimulus elements (black solid vs. gray solid line) than on easy stimulus elements. When processing demands increase, an immediate prolongation of fixation durations is observed (gray dashed line). Decreasing processing demands, by contrast, produce a delayed response (black dashed line). While fixation durations on the first stimulus remain almost as long as those in the difficult baseline condition, first-fixation

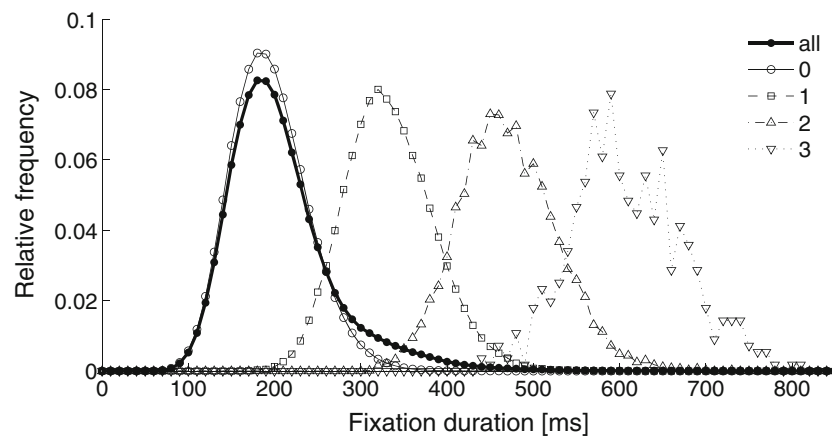


Fig. 8 Histograms of saccade initiation times with a bin width of 10 ms (all, $N=1,000,000$), saccade initiations without cancellation of the labile saccade program (0, $N=913,953$), with one cancellation (1, $N=78,658$), with two cancellations (2, $N=6,785$), and with three cancellations (3, $N=$

558). Four and five successive cancellations occurred rarely and were observed 45 times and once, respectively. Distributions were normalized relative to the included number of fixations

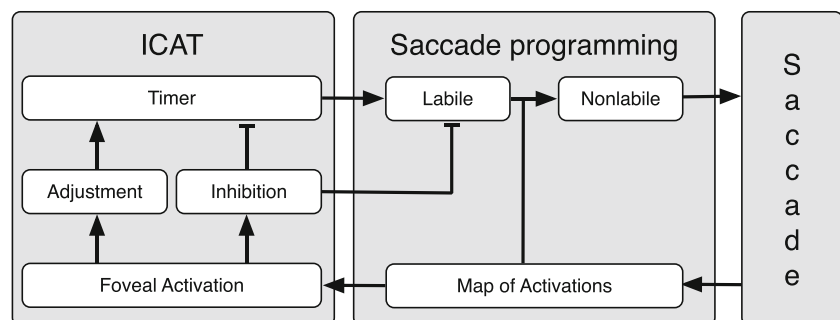
durations on subsequent stimulus elements slowly approach the low-difficulty baseline.

Simulation results are shown in the central panel of Fig. 11. The overall pattern is captured by the model. Since the model was fitted to the performance of the average participant, variability of population means and confidence intervals were somewhat smaller in our simulations. In order to assess the quality of simulations, we plotted simulated data against experimental data in the right panel of Fig. 11. Filled dots show the relation between simulated data and the data points used to estimate model parameters (first half of participants). Gray squares represent the second half of participants predicted by the same simulations; however, corresponding data were not used for parameter estimation. Data points from the first and second halves are connected by horizontal lines. The unity line ($y=x$) was added to facilitate comparisons of experimental and simulated data. Data points based on a perfect prediction would line up on this reference line. Furthermore, we calculated correlation coefficients (Spearman rank correlation) between the first and the second halves of the data (R_d), between simulated data and the first half (R_1), and between simulated data and the second half (R_2). The correlation coefficient R_d is an estimate of the reliability of the experimental data and gives an upper boundary of how well the model might predict

experimental data. The value R_1 indicates how well the model predicts the data used for parameter estimation. The value R_2 should not be considerably smaller than R_1 since deviations indicate an overfit of the model to the first half of participants. As can be seen in Fig. 11, the first and second halves of the experimental data are quite similar and show a high correlation ($R_d=.87$). The correlation between simulated and experimental data are of similar magnitude, with a slightly better fit of the first half of participants ($R_1=.93$, $R_2=.81$). We provide correlational plots for all analyses to give an estimate of the quality of the simulated data. Generally, we did not observe overfitting of the data set used for parameter estimation.

Two aspects of the experimental data are worth highlighting. First, in the decreasing difficulty condition, fixation durations on the last difficult stimulus (relative position 0) are increased relative to the high-difficulty baseline. Thus, even though the upcoming change decreases processing demands, fixation durations rise. A similar finding, the *parafoveal-on-foveal (PoF) effect*, has been reported in word recognition (Kennedy, Pynte and Ducrot, 2002) and reading (Inhoff, Starr and Shindler, 2000), where properties of the next (parafoveal) word modulate fixation durations on the fixated (foveal) word. The increased fixation duration at position 0 lends support to such a PoF effect in our data. However, in a

Fig. 9 Model overview: The ICAT principles incorporated into a general model of eye movement control



a) Low Difficulty	○	○	○	○	○	○	○	○
b) High Difficulty	○	○	○	○	○	○	○	○
c) Increasing	○	○	○	○	○	○	○	○
d) Decreasing	○	○	○	○	○	○	○	○
Relative position	-3	-2	-1	0	1	2	3	4

Fig. 10 Sequence of fixated symbols. Processing demands change at relative position 1 in the two experimental conditions. **a** Baseline condition with low difficulty: All stimulus elements have wide gaps. **b** Baseline condition with high difficulty: All stimulus elements have narrow gaps. **c** Experimental condition with increasing difficulty: At relative position 1, gap size decreases and remains narrow. **d** Experimental condition with decreasing difficulty: At relative position 1, gap size increases and remains wide

first-order approximation, we did not include this effect in our model, since its origin remains nebulous. Further research is needed to determine whether this is a real effect on saccade initiation intervals or just a consequence of additional saccade programming time because of, for example, readjusting average saccade length. Irrespective of the origin of the effect, our experimental results demonstrate that parafoveal processing is a crucial factor influencing fixation durations.

Second, saccade timer adjustments in our model depend on the last stimulus. Thus, a stepwise adjustment of fixation durations might be expected. Nonetheless, we observed a gradual decline of fixation durations across several stimulus elements in both experiment and model (dashed black line). Since eye movements are not constrained to strictly serial, left-to-right movements, items can be skipped without receiving a fixation. On some occasions, skippings will move the eyes from a simple stimulus to a difficult one that, in this case, already is the second or third stimulus after a change. Since saccades are more likely to skip a single stimulus, the proportion of skippings landing on the second stimulus is higher than

the proportion of skippings landing on the third stimulus. When analyzing the m th fixation duration relative to the first fixation after a change, fixation durations show the expected modulations (Fig. 12). Increasing processing demands immediately prolong fixation durations, while decreasing processing demands lead to a reduction of fixation durations with a temporal delay. Both experimental and simulated data are well-adjusted on the second fixation after a change. Thus, the slow decline in Fig. 11 results is due to a mixture of forward saccades and skipping saccades.

Fixation duration distributions

Histograms of fixation durations with a bin width of 25 ms are shown in Fig. 13 for experimental (left panels) and simulated (central panels) data. Distributions depict the first-fixation duration on a new stimulus (position 1), the preceding stimulus (position 0), and the two subsequent stimulus elements (positions 2 and 3). Experimental variability is well-captured by simulations of the full model. Each distribution was calculated by collapsing data across participants. We used the same fixations as in Fig. 11, and fixation duration distributions evolve accordingly. On all stimulus elements, fixation duration distributions are shifted toward longer fixations in the high-difficulty baseline (black solid line), as compared with the low-difficulty baseline (gray solid line). At position 1, fixation duration distributions immediately resemble the high-difficulty baseline when processing difficulty increases (gray dashed line). Decreasing processing difficulty (black dashed line), by contrast, generates a delayed (position 1) and gradual adjustment toward the low-difficulty baseline (positions 2, 3). Inspection of the correlation plots reveals that

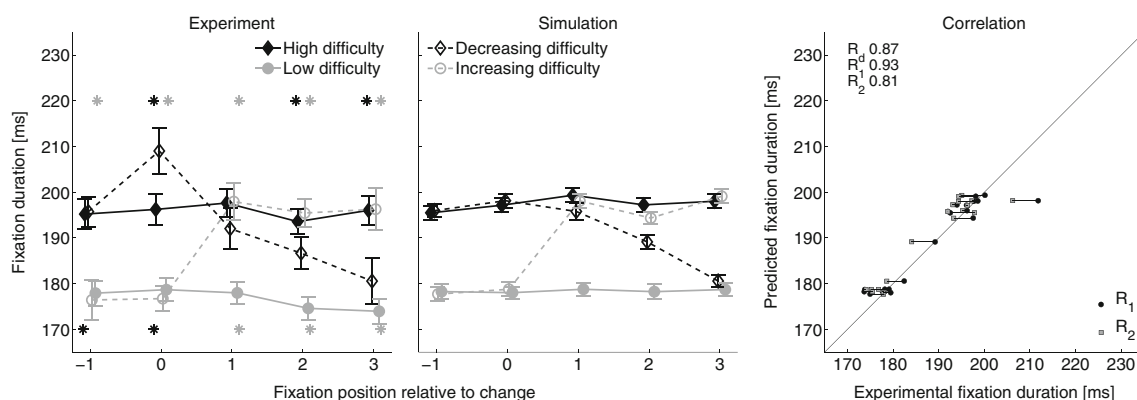


Fig. 11 Mean first-fixation durations on a stimulus relative to a change in processing difficulty. Left panel: Experimental data. Central panel: Simulation results. Solid lines represent baseline conditions without a change in gap size. In other conditions, gap size changed at position 1 (dashed lines). Trials starting with stimulus elements with a small gap are plotted in black, and trials starting with a large gap are plotted in gray. Error bars indicate confidence intervals based on Cousineau (2005). Asterisks show significant paired-sample t -tests ($p < .05$) between experimental

conditions (top row, decreasing difficulty; bottom row, increasing difficulty) and baseline conditions (gray asterisks, low difficulty; black asterisks, high difficulty). Right panel: Comparison of simulations with first and second halves of the experimental data. R_d gives the correlation between first and second halves of the experimental data. R_1 and R_2 indicate correlations between simulated data and first half and second halves, respectively

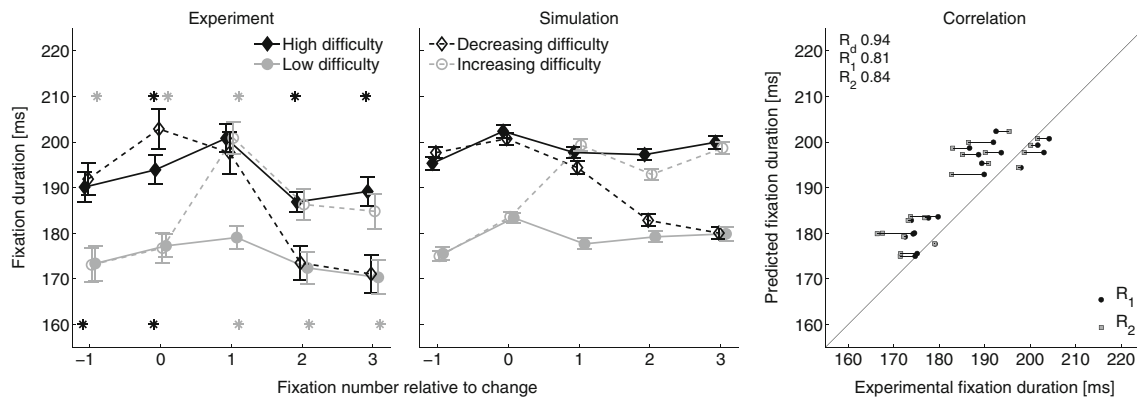


Fig. 12 Mean fixation durations of the n th fixation relative to a change in gap size. Left panel: Experimental data. Central panel: Simulation results. Solid lines represent baseline conditions without a change in gap size. In other conditions, gap size changed at position 1 (dashed lines). Trials starting with stimulus elements with a small gap are plotted in black, and trials starting with a large gap are plotted in gray. Error bars indicate confidence intervals based on Cousineau (2005). Asterisks show significant paired-sample t -tests ($p < .05$) between experimental conditions (top

row, decreasing difficulty; bottom row, increasing difficulty) and baseline conditions (gray asterisks, low difficulty; black asterisks, high difficulty). Right panel: Comparison of simulations with first and second halves of the experimental data. R_d gives the correlation between first and second halves of the experimental data. R_1 and R_2 indicate correlations between simulated data and the first half and second half, respectively

both halves of the experimental data are well predicted by our model simulations.

Skippings benefits

In well-structured tasks like reading or in the current experiment, processing order of objects is intrinsically given, and saccades may be classified according to their target. In particular, the distinction between forward saccades (i.e., movements from the currently fixated object n to the neighboring object $n+1$) and skippings (i.e., saccadic movements skipping the next object and moving gaze to a more distant object $\geq n+2$) is impossible in less structured tasks but very informative in regard to eye guidance. Since skippings generate larger saccade amplitudes, more information needs to be processed on fixations encompassing a skipping. Interestingly, the investigation of fixation durations has led to the observation of skipping benefits—that is, shorter fixation durations before skipping the next object than before fixating the next object. Skipping benefits have been reported in scanning tasks (Trukenbrod and Engbert, 2012) and during reading, where they are mediated by word frequency or word length (Kliegl and Engbert, 2005). Furthermore, when looking for compensatory effects, Trukenbrod and Engbert (2012) demonstrated that at least some skippings are not accompanied by additional processing time on neighboring fixations.

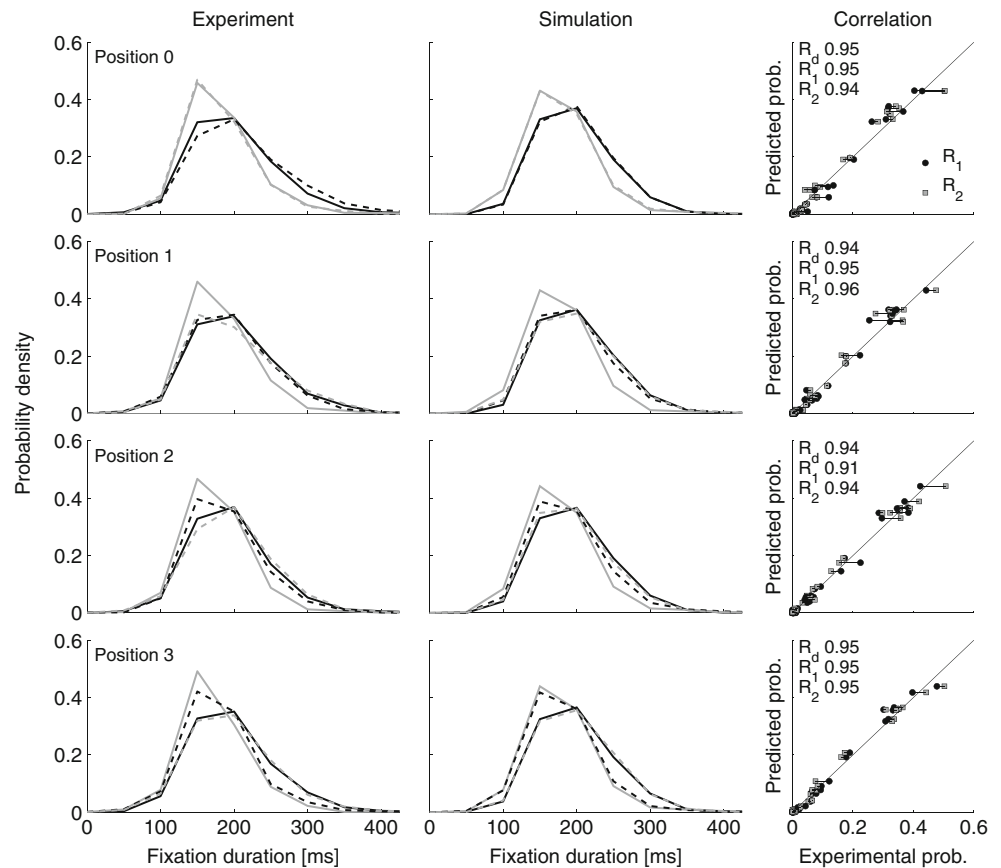
Fixation durations before refixations, forward saccades, and skippings taken from the two baseline conditions (high difficulty, low difficulty) are plotted in Fig. 14. Shortest fixation durations were observed before refixations both in the experiment and in simulations. Skippings were further classified as saccades skipping one stimulus, landing on stimulus $n+2$ (skipping 1), and saccades skipping two stimuli, landing

on stimulus $\geq n+3$ (skipping 2). Our experiment revealed skipping benefits that were attenuated in the low-difficulty condition. Interestingly skipping more than one symbol resulted in shorter fixation durations than just skipping a single stimulus. Our simulations generate slightly longer fixation durations when the next stimulus is skipped. This effect, however, reverses when more than one stimulus is skipped and is attenuated in the low-difficulty condition. Thus, saccades initiated by a timer that is modulated by processing but not triggered by processing is suitable to generating skipping benefits. Since our analysis was limited to the control conditions with constant processing demands, skipping benefits are not a result of temporal adjustments of the ICAT saccade timer.

Summary

The main purpose of Experiment 1 was to test the mechanisms of saccade timing underlying the ICAT model within a full model of eye guidance. Our simulation results demonstrate that the effects of asymmetric control of fixation durations are compatible with realistic spatial behavior of our model. While average fixation durations can immediately be prolonged when processing demands increase, a shortening of fixation durations is observed only on subsequent fixations. Variability produced by our model is comparable to the variability observed experimentally. In addition, analysis of skipping benefits revealed that saccade timing as implemented in our model does not necessarily generate prolonged fixation durations prior to skippings, contrary to models assuming sequential attention shifts (Engbert and Kliegl, 2011).

Fig. 13 First-fixation duration distributions. Top panel: Histograms before a change (relative position 0). Second panel: Histograms immediately after a change (relative position 1). Bottom panels: Histograms after adjustment (relative positions 2, 3). Solid lines represent baseline conditions without a change in gap size. In other conditions, gap size changed at position 1 (dashed lines). Trials starting with stimulus elements with a small gap are plotted in black, and trials starting with a large gap are plotted in gray. Left panels: Experimental data. Central panels: simulation results. Right panels: Comparison of simulations with first and second halves of the experimental data. R_d gives the correlation between first and second halves of the experimental data. R_1 and R_2 indicate correlations between simulated data and the first half and second half, respectively



Experiment 2: gaze-contingent display presentation

In Experiment 2, we developed a more rigorous test of foveal timing. We tested the same conditions and presented the same stimulus elements as in Experiment 1; however, parafoveal preprocessing was precluded by masks (using an “X” as mask). As soon as participants moved the gaze to a new object, the previously fixated stimulus was covered by the mask, and the newly fixated stimulus was revealed. Since the task could be solved successfully only by fixating each stimulus element, we asked participants to scan arrays from left to right and to fixate each stimulus. Trials with skippings or regressions were excluded from further analyses.

Mean fixation durations

Results from the second experiment are plotted in the left panel of Fig. 15. As in Experiment 1, durations of the first fixation on a stimulus were higher in the high processing difficulty condition (black solid line) than in the low-difficulty condition (solid gray line). When processing demands decreased (black dashed line), fixation durations did not immediately adjust to the new stimulus element. Instead, fixation durations remained unchanged with respect to the difficult baseline condition. When processing difficulty

increased (gray dashed line), an immediate but disproportionate prolongation of fixation durations was observed. Immediately after the increase, fixation durations were longer than would be expected on the basis of the baseline condition consisting of difficult stimulus elements.⁵ In both conditions, fixation durations returned to the corresponding baseline on the second stimulus element after a change. The major difference from Experiment 1 was the absence of the PoF effect at position 0 when processing demands decreased. This observation underlines the parafoveal origin of the PoF effect in Experiment 1.

Results from our simulations are shown in the central panel of Fig. 15. Note that fixation durations across participants decrease with increasing position of the symbol. In our simulations, we did not seek to account for this linear trend. Mean fixation durations were fit to mean durations at *relative position 1*. The qualitative pattern is in good agreement with the detrended experimental data. Fixation durations are longer when processing difficulty is high (black solid lines vs. gray solid lines). When processing demands decrease, fixation durations remain long on the first stimulus with a large gap size and decrease only after the second fixation (dashed black

⁵ A mask prevented preview of unfixated stimulus elements. Hence, processing time was the same in both conditions.

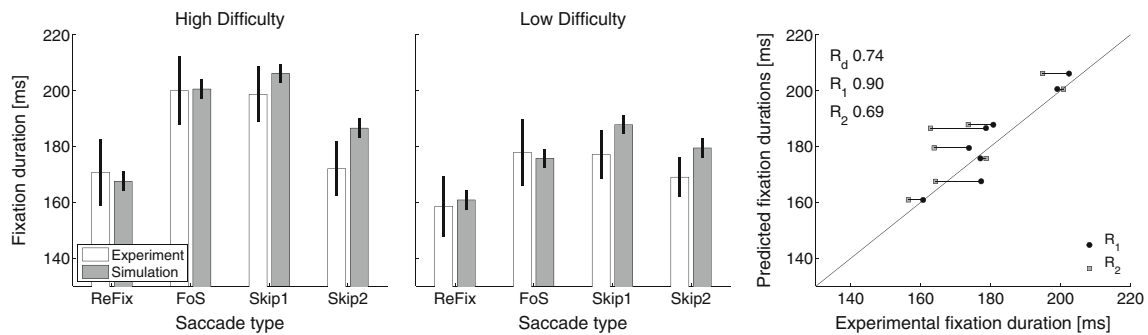


Fig. 14 Experimental and simulated fixation durations before refixating an object, making a forward saccade toward the next stimulus, and skipping of the next (skipping 1) and next but one (skipping 2) stimuli. Error bars indicate confidence intervals based on Cousineau (2005). Left panel: Fixations in the high-difficulty condition. Central panel: Fixations

in the low-difficulty condition. Right panel: Comparison of simulations with the first and second halves of the experimental data. R_d gives the correlation between the first and second halves of the experimental data. R_1 and R_2 indicate correlations between simulated data and the first half and second half, respectively

line). Most interesting, for increasing processing demands, the model generates an immediate and disproportionate prolongation on the first difficult stimulus element and returns to the baseline on the second stimulus element (gray dashed line).

Fixation duration distributions

Histograms of the first-fixation durations on a symbol (position 1), before a change (position 0), and after a change (position 2) are plotted in Fig. 16 (left panels, experiment; central panels, simulation; right panels, correlation plots). Distributions of fixations on difficult symbols were shifted toward longer fixation durations (black solid line vs. gray solid line). This shift can be observed at all positions in the baseline condition. The empirical pattern is reproduced by the model simulations. When processing demands changed during a trial, distributions were differentially modulated by

increasing and decreasing difficulty. Before a change, distributions matched the corresponding baseline conditions. Immediately after a change, fixation duration distributions were almost unaffected by decreasing processing difficulty (black dashed line). At the same time, increasing processing difficulty strongly modulated the shape of the fixation duration distribution (gray dashed line). Prolongation of fixation durations was accompanied by inflated variability. Thus, longer fixation durations after a change did not result from a shift of the distribution but, rather, from a prolongation of long fixation durations (i.e., >250 ms). Irrespective of the change, distributions on the second symbol after a change did not differ from the baseline conditions. Model simulations successfully reproduce the observed pattern across conditions and, most important, recover the increased skew of fixation duration distributions when processing demands increase.

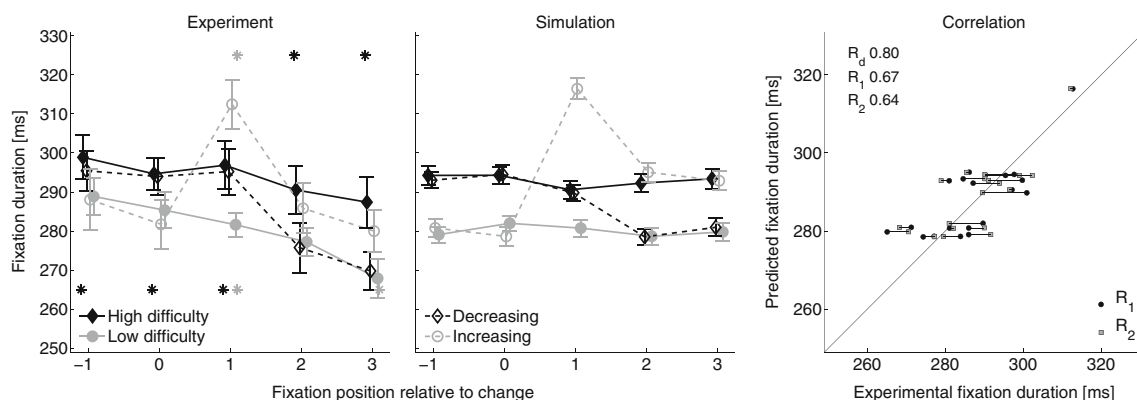


Fig. 15 Mean first-fixation durations on a stimulus relative to a change in gap size. Left panel: Experimental data. Central panel: Simulation results. Solid lines represent baseline conditions without a change in gap size. In other conditions, gap size changed at position 1 (dashed lines). Trials starting with stimulus elements with a small gap are plotted in black, and trials starting with a large gap are plotted in gray. Error bars indicate confidence intervals based on Cousineau (2005). Asterisks show significant paired-sample t -tests ($p < .05$)

between experimental conditions (top row, decreasing difficulty; bottom row, increasing difficulty) and baseline conditions (gray asterisks, low difficulty; black asterisks, high difficulty). Right panel: Comparison of simulations with first and second halves of the experimental data. R_d gives the correlation between the first and second halves of the experimental data. R_1 and R_2 indicate correlations between simulated data and the first half and second half, respectively

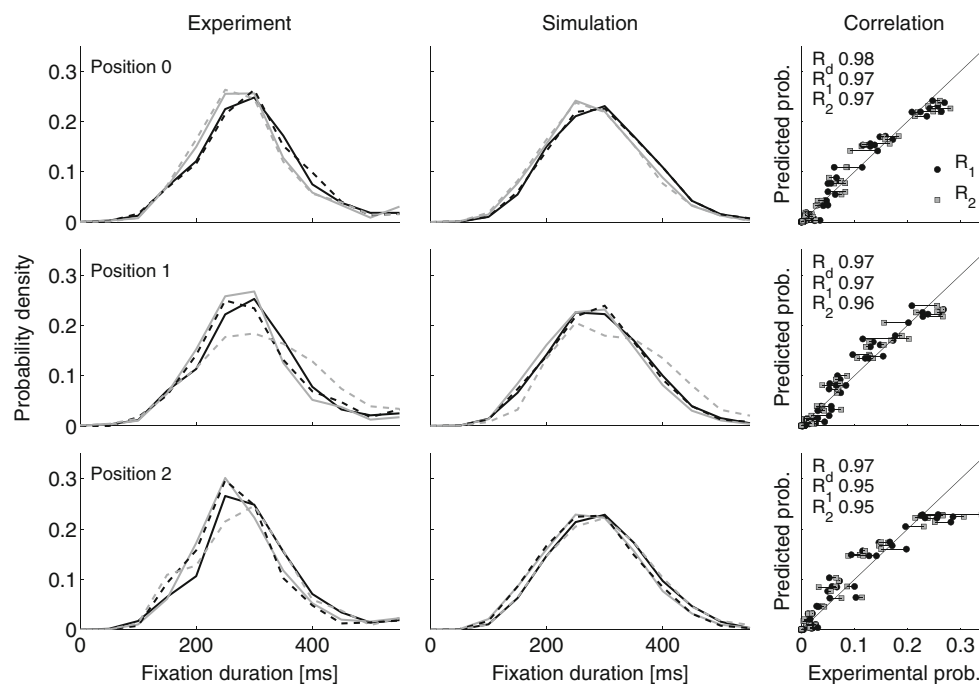


Fig. 16 First-fixation duration distributions. Top panel: Distributions before a change (relative position 0). Central panel: Distributions immediately after a change (relative position 1). Bottom panel: Distributions after adjustment (relative position 2). Solid lines represent baseline conditions without a change in gap size. In other conditions, gap size changed at position 1 (dashed lines). Trials starting with stimulus elements with a

small gap are plotted in black, and trials starting with a large gap are plotted in gray. Left panel: Experimental data. Central panel: Simulation results. Right panels: Comparison of simulations with first and second halves of the experimental data. R_d gives the correlation between first and second halves of the experimental data. R_1 and R_2 indicate correlations between simulated data and the first half and second half, respectively

Summary

Experiment 2 was a gaze-contingent experiment focusing on the control of fixation durations solely by foveal information. The results confirmed an asymmetric control of fixation durations as proposed by our model. While increasing processing demands may immediately prolong fixations, decreasing processing demands will affect fixation durations only on subsequent fixations. Furthermore, simulations of a full eye movement model captured temporal as well as spatial aspects of eye movements. Thus, control principles proposed in the ICAT model can be combined with target selection principles without disturbing the spatiotemporal dynamics of eye movements.

General discussion

We proposed and analyzed a new computational model for the control of fixation durations. We argued that the model explains experimental findings across tasks like reading, visual search, and scene perception within a unified framework. Our model is based on three core principles. First, autonomous saccade timing initiates new saccade programs after random time intervals independently of processing during a fixation. Second, average durations of autonomous saccade timing are

adjusted to expected foveal processing demands. Expectations are built up by the preceding fixation history. Third, foveal inhibition prolongs the initiation of a saccade program when processing is highly demanding. Foveal inhibition may have an immediate effect on fixation durations.

In a second part we evaluated performance of the model in two versions of a scanning task. Saccade initiation was incorporated into an existing model of eye movement control during reading (i.e., SWIFT; Engbert et al., 2005). Simulations confirm that our model is able to reproduce the development of average fixation durations and corresponding distributions while simultaneously maintaining a realistic spatial control of eye movements. Furthermore, skipping benefits—that is, shorter fixation durations before skipplings than before forward saccades—were replicated with ICAT.

Temporal adjustment of saccade timing

One outstanding characteristic of our model is the dynamic implementation of saccade initiation intervals based on the history of encountered processing demands. This is a crucial improvement with respect to previous theories about random saccade timing (Engbert et al., 2005; Hooge and Erkelens, 1998; Kolars, 1976), where saccade initiation intervals were assumed to be constant during a trial while matching overall task demands. In the latter case, saccade timing can be

adjusted only when the difficulty is known and will not adapt to new processing demands during a trial. During everyday activities, processing demands continuously vary within and across tasks with both gradual and abrupt changes. Our saccadic system is constantly required to adapt to new situations. It seems that previously faced processing difficulty is a useful predictor for the time required for task-dependent analysis. However, fixation history and expectations were confounded in our experiment. Simulating reading data that investigated effects of contextual constraints (see Supplementary Material for a simulation of the data set from Staub, 2011) revealed the importance of expectations independent of word properties. We conclude that expectations in saccade timing can be distinguished from fixation history.

Fixation duration control by parafoveal and peripheral processing

The current version of ICAT predicts modulations of fixation durations due to immediacy and lag effects—that is, by processing of the currently fixated region and the previously fixated region. Another important source of variability is related to processing in the parafovea (and maybe periphery)—in particular, processing at the upcoming fixation location. PoF effects—that is, modulations of fixation durations by the parafoveal stimulus—have been demonstrated in reading (e.g., Kennedy et al., 2002; Kliegl et al., 2006) and visual search (Trukenbrod and Engbert, 2012; Vlaskamp and Hooze, 2006). Currently, ICAT lacks the ability to reproduce this effect, which can be seen in the simulation studies of Experiment 1 (Figs. 11 and 12). There are two major reasons why we decided not to address the problem of the observed PoF effect in a first version of our model.

First, PoF effects are highly contentious see, e.g., Drieghe, 2011, for a discussion of PoF effects in reading, and the mechanisms that generate PoF effects are not well understood. It has been suggested that PoF effects are a consequence of mislocated fixations and reflect processing at the intended saccade target. Since an easy stimulus was at the intended saccade target in Experiment 1, the mislocated fixations account predicts a PoF effect in the opposite direction—that is, a shortening of fixation durations. The PoF effect in our experiment, however, led to longer mean fixation durations than fixations at any other fixation location. Hence, we dismiss this interpretation as the source of the PoF effect in our paradigm. Our data were compatible with two other interpretations. (1) PoF effects might reflect a delayed decision to initiate the next saccade program. This interpretation suggests parafoveal inhibition in the ICAT model—that is, a rate modulation by parafoveal processing. If this interpretation is valid, subsequent versions of ICAT could be equipped with an inhibitory mechanism that is based on foveal and parafoveal processing (cf. Laubrock, Cajar, and Engbert, 2013). (2) PoF effects

might be a consequence of differences in saccade programming. The regular arrangement of our stimuli might have generated a stereotyped behavior with a preferred saccade amplitude for the difficult stimulus elements. When the first easy stimulus element lengthens this preferred saccade amplitude, an additional programming cost might occur that prolongs fixation durations. In that case, the decision to program a new saccade remains unchanged by parafoveal processing, and PoF effects arise from a prolongation of saccade programming. In line with this interpretation, the PoF effect in Experiment 1 was accompanied by a decrease in the probability of generating forward saccades.

Second, the PoF effect is rather small when compared with the size of immediacy and lag effects. Simulations with SWIFT have demonstrated that the dynamics of the computational model may generate small PoF effects. Hence, PoF effects might be inherent in the dynamics of the computational model, and the implementation of a mechanism for PoF effects at an early stage might hinder the exploration of the full potential of ICAT.

As a final remark, there are several potential model extensions that have recently been explored using a similar mixed control mechanism. Schad and Engbert (2012), for example, investigated a dynamic attentional span in SWIFT. The size of the attentional span determined the area from which information was extracted, so that attention was restricted to the fixated word in some cases, while attention was extended to the parafovea in other cases. Given that foveal inhibition is determined by processing across the entire attentional span, parafoveal effects can arise. In a variant of the CRISP model (Nuthmann et al., 2010), Laubrock et al. (2013) investigated fixation duration control in scene perception using images filtered either in the fovea or in the parafovea/periphery. According to their model, processing in the fovea interacts with processing in the parafovea/periphery, and the ratio of foveal and parafoveal/peripheral activations determines the strength of inhibition.

Although the current version of ICAT does not generate PoF effects, their existence does not disagree with the general architecture of ICAT. Nonetheless, future models including ICAT will need to address PoF effects, which constitute an important benchmark for eye movement models.

Cognitive triggers versus indirect control of fixation durations

Across tasks, there have been enduring debates as to whether fixation durations are controlled by cognitive triggers or via modulated indirect control. This problem is particularly difficult to solve in complex tasks like reading, where fixation durations are affected by multiple factors. One major advantage of the proposed model is that it allows one to estimate the influence of processing on a moment-to-moment basis. Since immediate modulations of fixation durations can be achieved

only by foveal inhibition, parameters of foveal inhibition uncover to what degree fixation durations depend on momentary processing. At one extreme, fixation durations will be unaffected by foveal inhibition. At the other extreme, foveal inhibition decreases the rate of the random walk on each time step. Hence, estimated parameters indicate whether fixation durations in a given task can be accounted for by a pure indirect control mechanism or determine to what degree fixation durations are modulated by ongoing processing. Interestingly, all simulations reported here and in the Supplementary Material were based on parameter combinations leading to an asymmetric control. Even though some effects might seem like evidence for cognitive trigger theories (as frequency modulations during reading), asymmetric control seems to be sufficient to replicate the observed data patterns.

SWIFT and CRISP models

Our model extends the concept of random timing first proposed in SWIFT (Engbert et al., 2005), a model of eye movement control during reading, and CRISP (Nuthmann et al., 2010), a model of eye movement control in scene viewing. Both frameworks agree on core assumptions about saccade timing. Saccades are initiated after random time intervals by an autonomous saccade timer. Thus, an indirect control mechanism triggers new saccades. When high processing demands are encountered, foveal inhibition delays saccade initiation. Both frameworks represent mixed control strategies, since saccades are generated after random durations but can be modulated by ongoing processing. The proposed model, however, contains substantial improvements.

First, temporal adjustments of saccade timing are explicitly implemented in our model. Saccade timing is a dynamic process, and both autonomous initiation and foveal inhibition change over time as a function of fixation history. As was mentioned earlier, this represents a new framework for models of eye movement control. However, more research is needed to determine the number of fixations influencing saccade timing in different tasks. In SWIFT, the mean duration of the autonomous timer is constant, which may be seen as an extreme case where adjustments depend on an infinite number of preceding fixations. In this case, local changes in processing demands will not affect random saccade timing. Instead, the average duration of the autonomous saccade timer and the strength of foveal inhibition remain unchanged during a task. Our model may help to estimate the maximum number of fixations influencing saccade timing and the sensitivity to local changes in processing demands during reading.

Second, continuous-time, discrete-state random walks control saccade initiation intervals and saccade programming. Mathematically, this implementation has at least two major advantages, as compared with the procedure used in SWIFT, where a set of coupled ordinary differential equations is

discretized by the Euler method for the computation of numerical solutions. Simulations of our model with a Monte Carlo procedure proposed by Gillespie (1978) are mathematically exact and time efficient, since each iteration step (with variable length) reflects a state change and numerical inaccuracies due to discretization are avoided. While the CRISP model uses a single random walk, our implementation demonstrates how multiple random walks can be simulated simultaneously.

Third, as in the CRISP model, foveal inhibition is implemented on a moment-to-moment basis. By using continuous-time, discrete-state random walks for autonomous saccade timing, foveal inhibition can be implemented as a continuous process. In our model, rates of a random walk are immediately modulated by ongoing processing. Depending on the strength of foveal inhibition, a varying number of time steps are affected. As a consequence, foveal inhibition is able to replicate both patterns of mean fixation durations and fixation duration distributions of various shapes (e.g., multimodal).

Conclusions

Using a computational model, we studied principles of fixation duration control. First, random timing and adjustment to high processing difficulty by foveal inhibition are suggested as local control principles. Additionally, we introduced a global control for the adjustment of fixation durations to specific task constraints that are independent of the current processing difficulty. We demonstrated the viability of our model in controlling fixation durations in tasks with sequences of fixations. The model reproduced both average fixation duration patterns and fixation duration distributions.

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