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ICAT: A Computational Model for the Adaptive Control of Fixation Durations

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Contents

Introduction	4
Global control of fixation durations	5
Theories of local control of fixation durations	5
Fixations durations across tasks	7
Modeling motivation & goals	11
Core Principles of the model	12
ICAT: A model of saccade initiation intervals	13
Local-I: A random-walk model of saccade generation	13
Local-II: Foveal inhibition	15
Global: Adjustment of saccade timer	16
Qualitative behavior of the saccade timer model	17
Immediacy & lag effects	26
Labile saccade programming	26
Combining temporal control in ICAT with spatial eye movement control	28
Experimental validation	30
Task	30
Experiment 1: Stepwise processing change during visual search	31
Experiment 2: Gaze-contingent display presentation	36
General Discussion	38
Temporal adjustment of saccade timing	40
Fixation duration control by parafoveal and peripheral processing	40
Cognitive triggers vs. indirect control of fixation durations	41
SWIFT and CRISP models	42
Conclusions	43
References	43

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. (192 words)

Introduction

Visual perception is a highly dynamic process during which saccadic eye movements continually scan the environment (Findlay & 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 & Walker, 1999). Due to this independence, a research strategy focussing 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 importantly, this research generated detailed computational models that account for the selection of fixation locations during reading (e.g., Engbert, Nuthmann, Richter, & Kliegl, 2005; Reichle, Pollatsek, Fisher, & Rayner, 1998; Engbert & Krügel, 2010), scene perception (e.g., Itti & Koch, 2001; Kienzle, Franz, Schölkopf, & Wichmann, 2009; Torralba, Oliva, Castelhano, & Henderson, 2006), and visual search (e.g., Najemnik & Geisler, 2005; Rutishauser & 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; Reichle, Rayner, & Pollatsek, 2003, for an overview). To fill this gap, Rayner (2009) suggested that experimental paradigms developed to analyze fixation durations in reading should be more widely adopted 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 & Pollatsek, 1981) to analyze the control of fixation durations in scene viewing (Henderson & Pierce, 2008; Henderson & Smith, 2009). Based on 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: (i) Local control depending on processing difficulty of the item in foveal vision affects the current fixation and (ii) 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 (Hooze & 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 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, & 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, & Ruthruff, 1992), visual search (Näsänen, Ojanpää, & Kojo, 2001), and reading (Bowers & Reid, 1997). Low and high pass filtering of an image changes average fixation durations (Groner, Groner, & Mühlenen, 2008; Mannan, Ruddock, & Wooding, 1995) and stimulus difficulty modulates fixation durations, which increase with increasing target-distractor similarity (Hooge & Erkelens, 1998; Jacobs, 1986; Vlaskamp, Over, & Hooge, 2005) and increasing complexity of stimulus elements (Gould & Dill, 1969).

Second, task specific processing (Rayner, Li, Williams, Cave, & Well, 2007) affects average fixation durations that last about 225 to 250 ms during reading, 275 to 325 ms during scene perception, and 180 to 275 ms during visual search (for a review see Rayner, 2009). These modulations do not solely depend on stimulus properties and remain when the same stimulus material is viewed under differing instructions. During scene perception, fixations are longer when memorizing a scene in comparison to searching the same scene (Henderson, Weeks, & Hollingworth, 1999; but see Castelano, Mack, & Henderson, 2009). During reading, average gaze durations decrease when skimming a text (Just & Carpenter, 1987) or repeatedly reading the same text (Hyönä & Niemi, 1990) and lengthen during elaborate processing (Bohn & 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, & Liversedge, 2011). Interindividual differences persist across tasks revealing the tendency of a preferred average fixation duration in participants (Castelano & 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 extensively been investigated. Resulting theories can be classified into three categories which assume control by a cognitive trigger (direct control), indirect control, or mixed control (Rayner & McConkie, 1976; Rayner, 1977; Rayner & Pollatsek, 1981). *Cognitive trigger* theories assume that the decision to move the eyes is solely based on processing of visual input during a fixation. Saccades might be triggered by the decision 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 (submitted) suggested that the trigger to initiate a new saccade program is synchronized with the trigger that shifts attention to the next object.¹ 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, which 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, & 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 (Fove & Simpson, 2002; Lamme & Roelfsema, 2000; Poghosyan & Ioannides, 2007). Third, programming of saccades takes 150 to 175 ms (Becker & Jürgens, 1979; Rayner, Slowiaczek, Clifton, & Bertera, 1983; Rayner, 1998; note that Schall & Thompson, 1999, give slightly smaller estimates of 100 to 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 (Hooge & Erkelens, 1998; Kolers, 1976), i.e., 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 reaching a deadline independent of current processing (cf., Engbert & 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 assumes that fixation durations are primarily terminated by indirect control where processing

¹More 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 its simplest form, where saccade programs are initiated by processing of input from the foveated area.

may prolong the current fixation by inhibiting the execution of the next saccade (Engbert et al., 2005; Feng, 2009; Yang & 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 & 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, subject's eyes were guided by foveal information (Hooze & 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 fixations was too short to select the correct saccade target. Hooze and Erkelens (1998) concluded that saccades are initiated after random time intervals independent 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 & Erkelens, 1999) and/or parafoveal analysis (Vlaskamp & Hooze, 2006), 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' (1996, 1998) displays, the arrangement of stimulus elements by Greene and Rayner (2001) was denser and permitted parafoveal preview of neighboring symbols. As a consequence, Greene and Rayner (2001) observed a higher proportion of correctly directed saccades and argued for a tight link between processing and eye-movement control in dense compared to sparse displays. However, Greene and Rayner (2001) observed a fraction of saccades that were not directed toward the target as predicted by indirect control theories (Hooze & 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 like stimulus, preview, and visibility, saccadic performance may vary from unselective to almost perfectly selective across tasks (see Trukenbrod & 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 (Hooze & Erkelens, 1999).

Contrary to indirect control, some studies observed immediate fixation duration modulations during visual search. Fixation durations increase with complexity of stimulus

elements (Salthouse & Ellis, 1980) and increasing target-distractor similarity of fixated objects (Becker & Williams, 2011; Trukenbrod & Engbert, 2007; Williams & Pollatsek, 2007). Hence, rejection of distractors has been suggested as the event triggering saccades in visual search (Becker & Williams, 2011; Rayner, 1995).

Recently, Hooze, 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. Hooze et al. (2007) 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: (i) Immediate prolongations of fixation durations can be seen when processing demands increase (local control), but (ii) 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 extensively been investigated 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 & Carpenter, 1980) and predictability (Ehrlich & Rayner, 1981) are two of the most important factors influencing eye-movement behavior at various levels. Both first-fixation durations as well as gaze durations are elevated on low-frequency (Inhoff & Rayner, 1986; Kennison & Clifton, 1995; Kliegl, Grabner, Rolfs, & Engbert, 2004; Rayner & Duffy, 1986) and unpredictable words (Inhoff, 1984; Rayner & Well, 1996; Zola, 1984). 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, & Raney, 1996). Second, inspection of first-fixation durations revealed that frequency (Staub, White, Drieghe, Hollway, & Rayner, 2010; Reingold, Reichle, Glaholt, & Sheridan, 2012) 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 like in normal reading (Rayner, Liversedge, White, & Vergilino-Perez, 2003; Liversedge et al., 2004; Rayner, Liversedge, & White, 2006; 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 only be triggered 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 & Rayner, 1986; Sereno & 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 subject. 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, & 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 fixations durations remained highly variable. More importantly, 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 ms 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 (Hooze 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 fixating a high-frequency word, fixation durations were affected by word frequency of the target word. Fixations were longer on low-frequency words than on high-frequency words (285 ms 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 ms 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 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 & Hollingworth, 1998; for reviews see Henderson & Hollingworth, 1999; Henderson & Ferreira, 2004). Henderson (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 towards it, prolongs subsequent fixation times even when viewers remain unaware of the change (Henderson & Hollingworth, 2003; Hayhoe, Bensinger, & 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 (Diepen & 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 & Liebelt, 1981; Loftus & Mackworth, 1978; Henderson et al., 1999). The effect typically shows up in gaze durations by refixating implausible objects more often but is fragile on the level of first-fixation durations (Henderson & Hollingworth, 1998). As an exception, De Graef, Christiaens, and d’Ydewalle (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 & McConkie, 2001) and visual search (Hooze 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 & Ferreira, 2004) and only rarely reflected in individual fixation durations (Henderson & 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 & Henderson, 2009).

Fixation durations in the SOD paradigm. A paradigm with gaze-contingent stimulus presentation (Rayner, 1975) has been used to investigate eye movements during reading (Ishida & Ikeda, 1989; Morrison, 1984; Rayner & Pollatsek, 1981), visual search (Vaughan, 1982; Vaughan & Graefe, 1977), and scene perception (Henderson & Pierce, 2008; Henderson & 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 importantly, 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 is 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 & Pierce, 2008; Morrison, 1984; Shioiri, 1993). It has been suggested that short fixation durations are pre-programmed during preceding fixations and that the number of pre-programmed fixations increases with increasing SOD. For example, Morrison (1984) reported 15 to 30 percent anticipatory saccades when the stimulus was delayed for 200 ms, but 50 to 75 percent for delays of 350 ms. Since pre-programmed saccades are specified during the preceding fixation, the proportion of pre-programmed saccades should not increase with increasing SOD (at least for fixation durations beyond 200 ms).

Even though results from the SOD paradigm have widely been 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 & Pierce, 2008; Henderson & Smith, 2009; Rayner & 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 based on 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 & 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, & 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 neither been 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 primarily focused on average fixation duration patterns. Nonetheless, fixation durations are highly variable and may under some circumstances lead to multimodal distributions (c.f., simulations of Yang & McConkie, 2001, in supplementary material). A model for the control of fixation durations needs to account for both mean fixation durations and their distributions.

²Saccade 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.

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 & 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 little time is left for processing of the visual input.

Indirect control combined with inhibition allows to initiate saccades independent of a specific processing event. *In such a framework, cognitive processing and saccade programming occur simultaneously and mostly independent of each other.*³ 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 & Jürgens, 1979). Thus, additional processing time can easily be obtained by refixating the target area.

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 only serve the purpose of processing needs, 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 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 is updated according to the specific fixation history.

What is the functional relevance of these three principles? While principles Local-II and Global are needed to tune the scanning rate of the saccadic system to the cognitive processing difficulty, principle Local-I 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

³In contrast, since saccades can only be initiated after successful processing of the foveated region, the eyes remain at an already processed location for about 150 ms in cognitive trigger theories.

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 & Wing, 1996). Interestingly, the addition of a stochastic drift process to the timer provided a significant improvement of the Wing-Kristofferson model (Collier & Ogden, 2004). From the perspective developed here, such a stochastic drift can be interpreted as a free-running adaptive mechanism which 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 (i) oculomotor aspects of saccade generation and of (ii) 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 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 intervals⁴ are implemented as a stochastic process, since random-walk models have successfully been used to account for variability of reaction times in behavioral decision processes (Ratcliff, 1978; Smith & Ratcliff, 2004, for a review on two-choice decisions) and eye-fixation durations (e.g., Engbert & 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 $t_{fp} = \min\{t | n(t) = N\}$. 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

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

Exponentially distributed waiting times were [sampled by computing](#) a log-transform of a

⁴In 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.

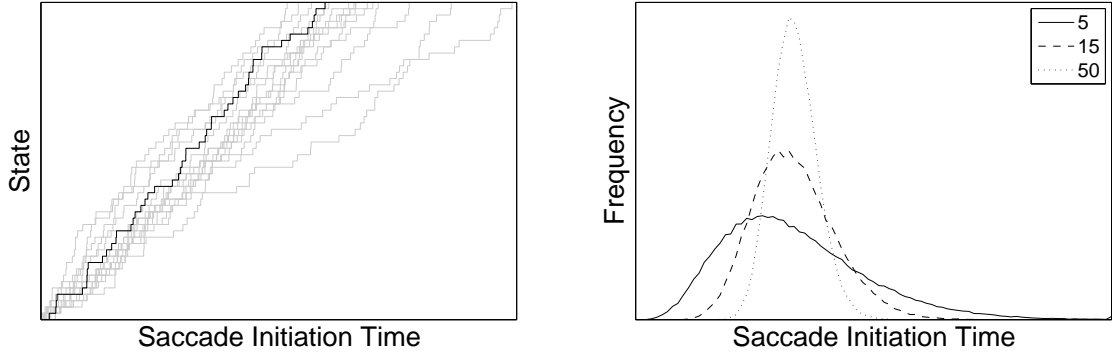


Figure 1. Random-walk model for saccade timing. (a) Examples for realizations of the random walk with threshold value $N = 50$. The random walk starts at $n(0) = 0$ and terminates at the threshold value $n(t_{fp}) = N$, which is reached after the first-passage time t_{fp} . (b) Distribution of the relative frequency for three different thresholds $N = 5, 15, 50$. Each distribution represents 100,000 realizations of the first-passage time t_{fp} .

uniformly distributed random number ξ , i.e.,

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

where ξ is drawn from independent and identically-distributed pseudo-random 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 compared to 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 Figure 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 pre-defined 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 Figure 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 , saccade initiation intervals are more variable and skewed, while large N_t 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_f , 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 .⁵ Throughout the paper, we use a very general form of the inhibition function $h_j[\cdot]$, i.e.,

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

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 scale the strength of inhibition. Values of the inhibition function $h_j[\cdot]$ range from 0 to 1, so that foveal inhibition by Eq. (4) decreases the transition probability (compared to the baseline \hat{w}) resulting in prolonged intervals of the saccade timer.

Putting together Eqs. (4,5), we obtain the time-dependence of the random walk's transition probability,

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

Modulations of the transition probability by foveal inhibition are illustrated in Figure 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 towards zero,

⁵Note 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 Supplementary Material.

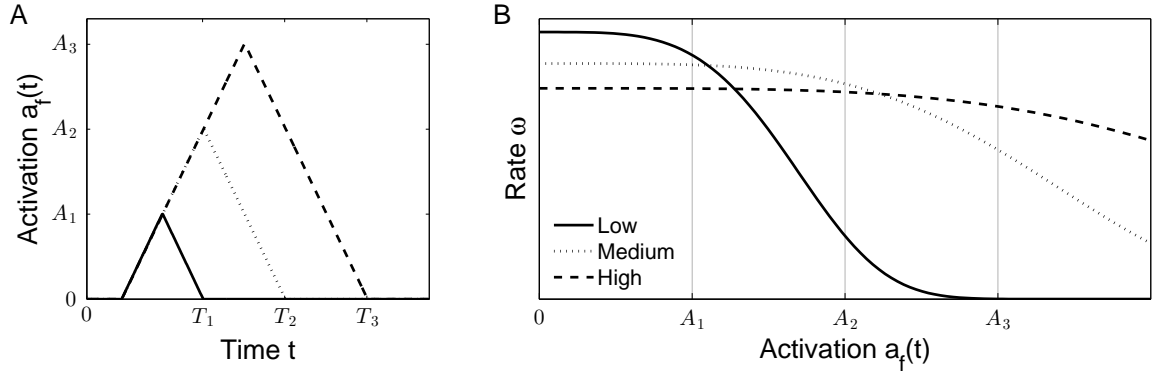


Figure 2. (a) Temporal evolution of three activations with varying maximum processing difficulty (A_1, A_2, A_3). In our examples activations remain zero until the onset of processing. In the beginning processing activations evolve identically but activations grow higher and processing lasts longer with 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 $w_j(t)$ is implicit, due to variation of the foveal activation $a_f(t)$.

$h_3(a_f) \approx 0$. Two additional lines are depicted in Figure 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 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 re-iterate its motivation in short form. There is experimental support (Harris, Hainline, Abramov, Lemerise, & Camenzuli, 1988; Hooze & Erkelens, 1998; Kolers, 1976) for the notion of random timing (Local-I), i.e., 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 (Kolers, 1976) or adjusted to the estimated foveal processing time of previously fixated stimulus elements (Hooze & 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 (Hooze & 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 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} solely depend 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 models 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 Figure 3a we demonstrate the effect of increasing processing demands in a single run of the simulation. Effects can be observed on three levels: On the level of (i) foveal processing, (ii) rate modulations of the random walk, and (iii) 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 as well as 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 afterwards (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 re-adjustment 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 Figure 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 $n + 1$, 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.

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 Figure 5. The left panel of Figure 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 higher when saccade timing is adapted to low processing demands (left panel) compared to 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 Figure 5a, activations within the white area evolve from processing

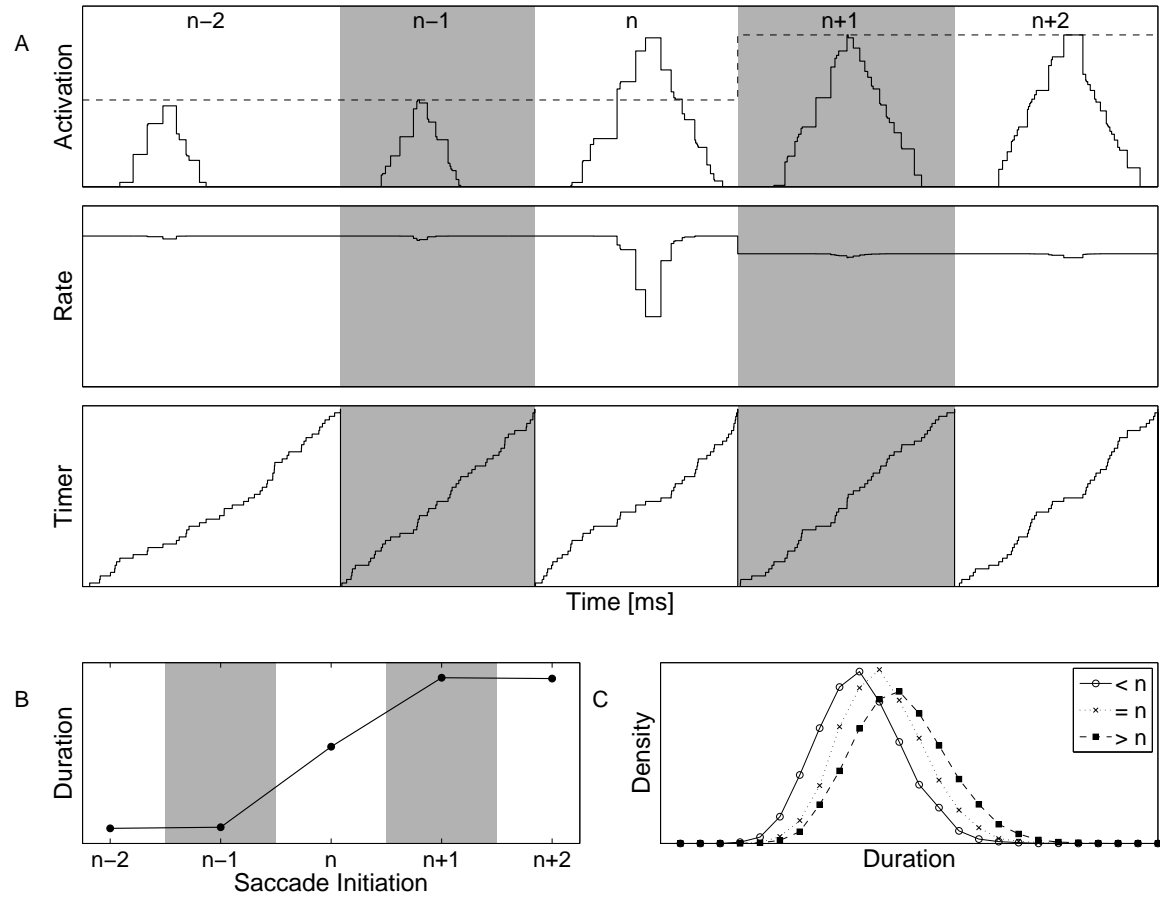


Figure 3. Illustration of saccade timer model: Processing demands increase during the n th saccade initiation. Levels of the model: Foveal analysis and expected processing demands (top panel: solid and dashed line, respectively), rate modulations (central panel), and the realized random walk (bottom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

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 Inhibition α β	
Figure 3 & 4: Single trial	40/70	50	0.66	50	300/340	0.03	6.00
Figure 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
Figure 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 ms.^b Activation change per millisecond during processing.

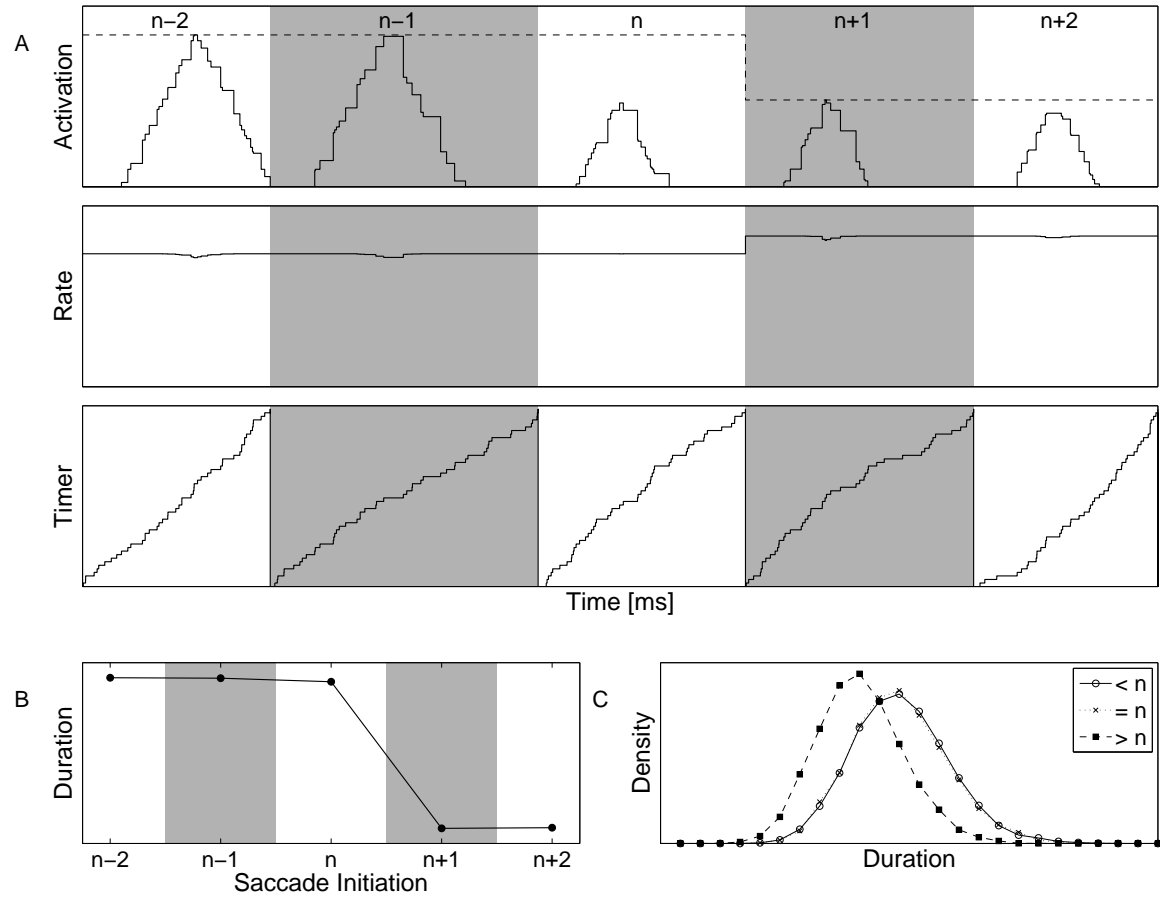


Figure 4. Illustration of saccade timer model: Processing demands decrease during the n th saccade initiation. Levels of the model: Foveal analysis and expected processing demands (top panel: solid and dashed line, respectively), rate modulations (central panel), and the realized random walk (bottom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

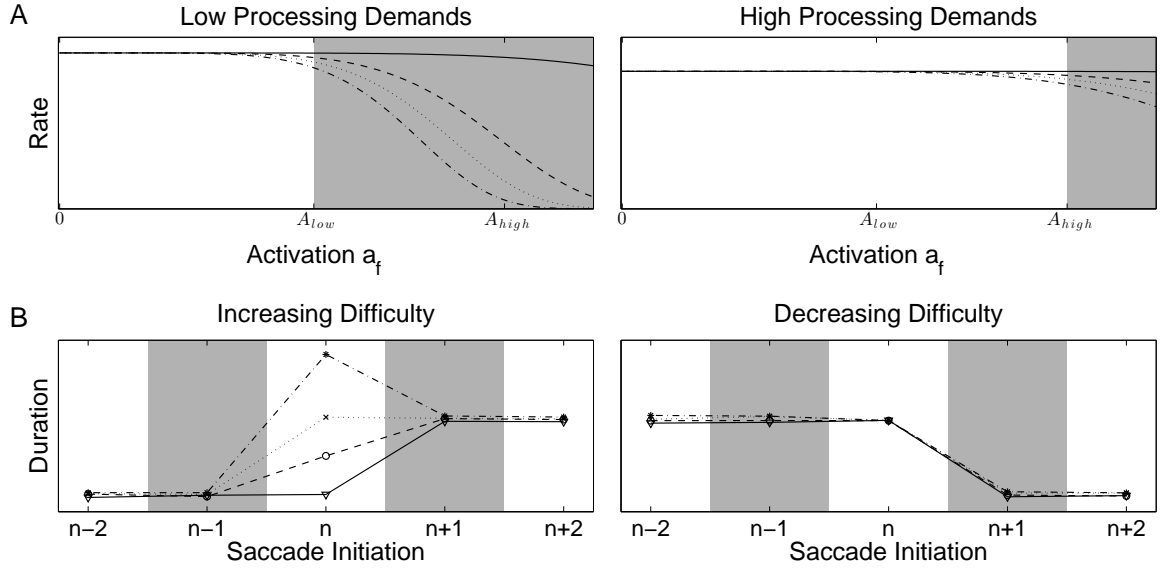


Figure 5. Foveal inhibition: Increasing processing difficulty. Rate modulations and mean saccade initiation intervals of four parameter combinations. a) Rate modulations when saccade timing is adjusted to low processing demands (A_{low} ; left panel) and high processing demands (A_{high} ; right panel). 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).

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 Figure 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, 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 to 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 afterwards ($\geq n + 1$). Such an asymmetric control of fixation durations, i.e., an immediate increase and a delayed decrease has been reported during reading (Kennison & Clifton, 1995) and visual search (Hooge et al., 2007). The increase, however, varied between experiments. While Kennison and Clifton (1995) observed some sort of overcompensation, Hooge et al. (2007) 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, 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 decreases. Such parameters 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. Compared to well adjusted saccade initiation intervals, the reduction is only partial. **It is important to note that all parameter combinations used in Figure 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

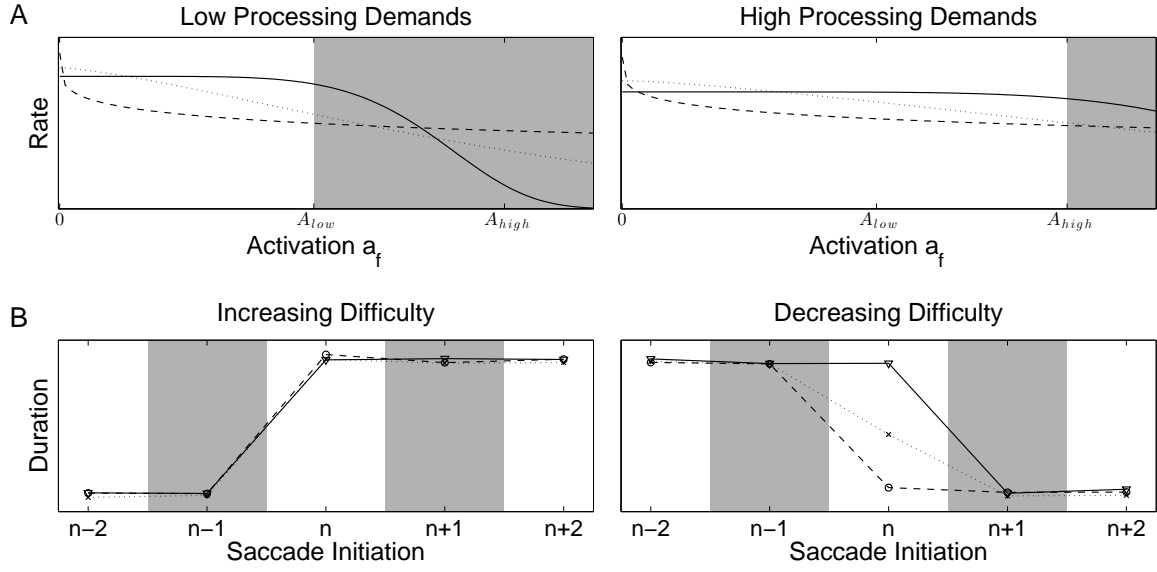


Figure 6. Foveal inhibition: Decreasing processing difficulty. Rate modulations and mean saccade initiation intervals of three parameter combinations. a) Rate modulations when saccade timing is adjusted to low processing demands (A_{low} ; left panel) and high processing demands (A_{high} ; right panel). 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).

fixation durations increased by a similar amount, the prolongation was caused either by only a few very long time steps (solid line), by many moderately prolonged time steps (dashed line), or 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 only been assumed implicitly in previous indirect control theories. In our model adjustments depend on expected processing demands (Eq. (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 (Eq. (8)). Second, strength of foveal inhibition scales with expected processing demands A_j (Eq. (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, i.e., $T_{j+1} = T_j$ (cf., Eq. (8)) and $A_{j+1} = A_j$ (cf., Eq. (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

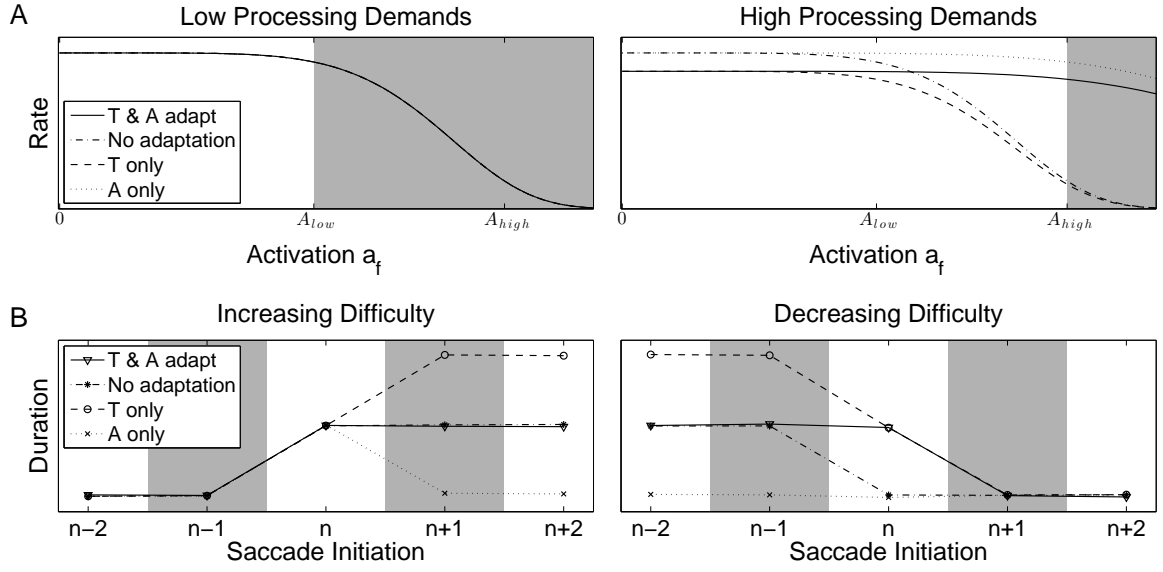


Figure 7. Saccade timer adjustments. Role of adjusting the average saccade timer interval T_j and the expected processing difficulty A_j . a) Rate modulations when saccade timing is adjusted to low processing demands (A_{low} ; left panel) and high processing demands (A_{high} ; right panel). In both plots the gray area shows activations above the expected maximum. Note, 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).

be described by asymmetric control, we use the same parameters as in Figure 5 (perfect adjustment). With two adaptive processes, rate modulations behave as illustrated earlier (solid line, Fig. 7a). When saccade timing is adapted to low processing demands (left panel), 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), 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 (dash-dotted line, Fig. 7), rate modulations are independent of previous processing demands, which leads to identical curves in the left and right panel. 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 (dashed line, Fig. 7). 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 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 only observed, 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 only generated, when both processes adapt simultaneously over time.

Immediacy & lag effects

The majority of eye-movement studies focused on average durations of first fixations, single fixations, and gaze durations (Rayner, 2009). As 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 interestingly, 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 & Duffy, 1986). During reading, fixations on word $n + 1$ are longer when the preceding word n is a low-frequency word compared to a high-frequency word (Henderson & Ferreira, 1990; Kennison & Clifton, 1995; Schroyens, Vitu, Brysbaert, & d'Ydewalle, 1999). Interestingly, the lag-frequency effect, i.e., the modulation of the fixation duration on word $n + 1$, is sometimes even larger than the immediacy effect on word n (Kliegl, Nuthmann, & Engbert, 2006; Schad, Nuthmann, & 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, either 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 & Rayner, 1986; Reingold et al., 2012; Sereno & 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 non-labile 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 be canceled by another new saccade program. Only after transition from the labile stage to the non-labile stage a saccade is imminent and the current fixation is terminated.

The main motivation for two stages of saccade programming derives from the double-step paradigm in saccade generation (Becker & 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 non-labile 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 & 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, & 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 Markov processes (Gillespie, 1978) to implement a coherent framework of stochasticity at multiple levels. Each component of saccade control, i.e., saccade initiation, labile and non-labile 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 non-labile 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, i.e., $m_1 = 0, 1, 2, \dots, N_t$ 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 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 Eq. (2)), i.e.,

$$\rho(\tau) = W_n \exp \{-W_n\} \quad \text{with} \quad W_n = \sum_{n \neq m} W_{nm}, \quad (9)$$

where W_n 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 $W_n(t)$ at time t is given by

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

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

While the transition rate $w_j(t)$ of the saccade timer is always non-zero, the random walk of the labile stage is only active after saccade initiation. When the random walk of the

saccade timer reaches a threshold, the labile program starts, $w_{lab}(t) > 0$, and terminates when reaching the oculomotor threshold, $w_{lab}(t) = 0$. The rate of the random walk is given by

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

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, i.e., $m_2 = 0$.

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

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

In summary, the algorithm proposed by Gillespie (1978) allows to simultaneously simulate multiple independent random walks (for a further extension see Supplementary Material). The computation is performed in three iterative steps. First, a total transition probability $W_n(t)$ is computed by summing the rates of all random walks. Second, the time until the next transition τ is determined by drawing a pseudo-random number from an exponential distribution (Eq. (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 $p_n(t)$ of each random walk (Eq. (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 Figure 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 cancelations (thin lines). While fixation durations with no cancelations 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 cancelations. Overall, 9% of fixation durations were affected by cancelations. As can be seen from the plot long fixation durations are primarily generated by saccade cancelations. In agreement with this, simulations in the Supplementary Information were also run without a cancelation mechanism. While simulations captured effects in mean fixation durations, long fixation durations were generally underestimated. Cancelations 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

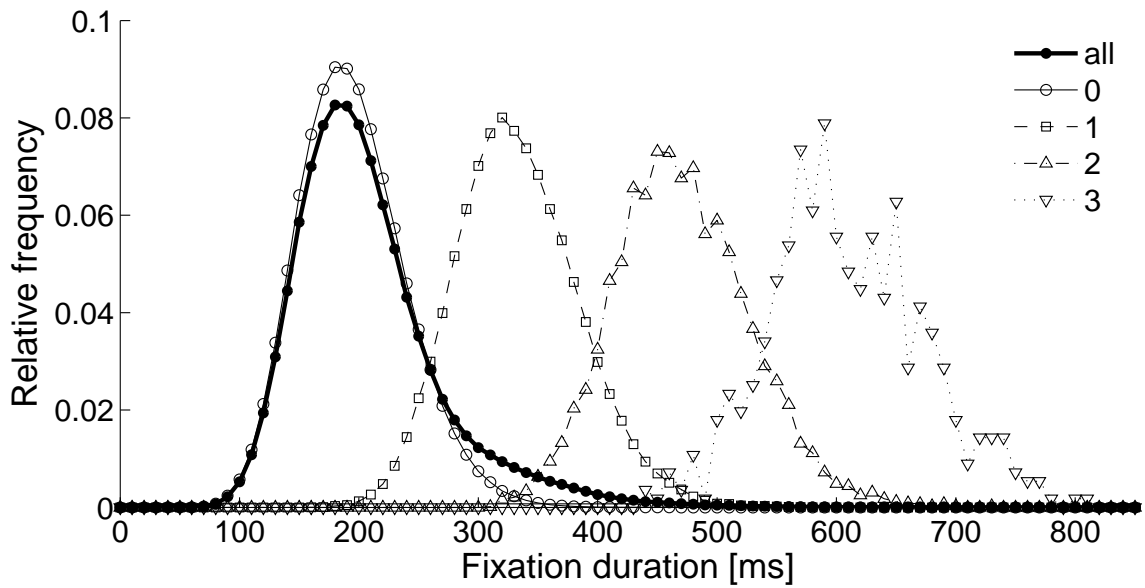


Figure 8. Histograms of saccade initiation times with a binwidth of 10 ms (all, $N = 1,000,000$), saccade initiations without cancellation of the labile saccade program (0, $N = 913,953$), with one cancellation (1, $N = 78,658$), two cancellations (2, $N = 6,785$), three cancellations (3, $N = 558$). 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.

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 Figure 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 (principle Local I). 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, foveal inhibition affects both the autonomous saccade timer and the programming of saccades during the labile programming stage. When passing into a non-labile 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 stated earlier, foveal processing feeds back to the ICAT model. Thus, foveal activations are used to inhibit the initiation of new saccades (principle Local II) and adjust saccade timing to new processing demands (principle Global). The full model generates a sequences of fixations with realistic temporal control and spatial control, i.e., 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.

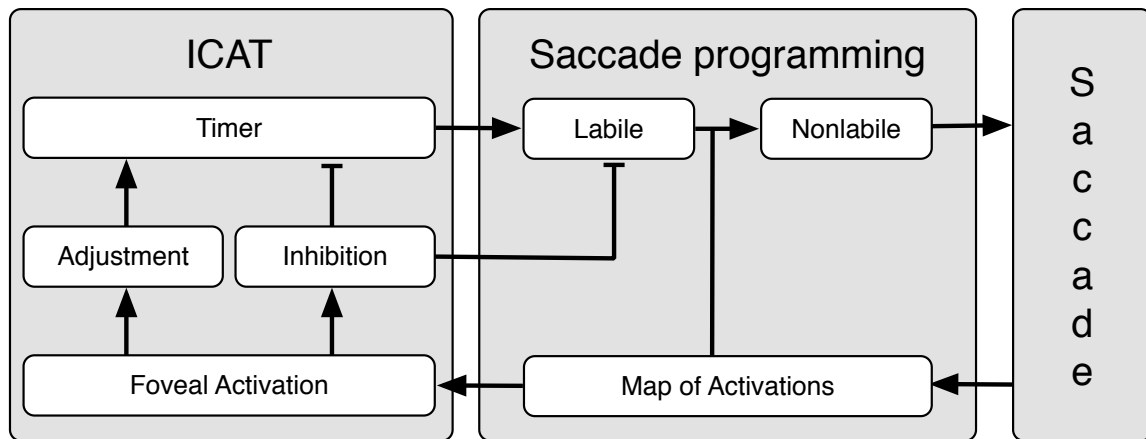


Figure 9. Model overview. The ICAT principles incorporated into a general model of eye-movement control.

Experimental validation

Using two variants of a simple scanning task (see Hooge & Erkelens, 1998, for a related task), 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, respectively.

In the first experiment, participants searched for a ring in an array of Landolt-Cs. Fixation duration adjustments were investigated by 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 eleven symbols from the left to the right on a computer display and to search for a target stimulus (see 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 Figure 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

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

Figure 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.

the fifth and eighth symbol 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 & Rayner, 1975). In 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 Figure 11. Stimulus position 1 corresponds to the first distractor with new gap size in conditions with varying processing demands (cf., Fig. 10). 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 the difficult baseline condition, first-fixation durations on subsequent stimulus elements slowly approach the low difficulty baseline.

Simulation results are shown in the central panel of Figure 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, respectively, were somewhat

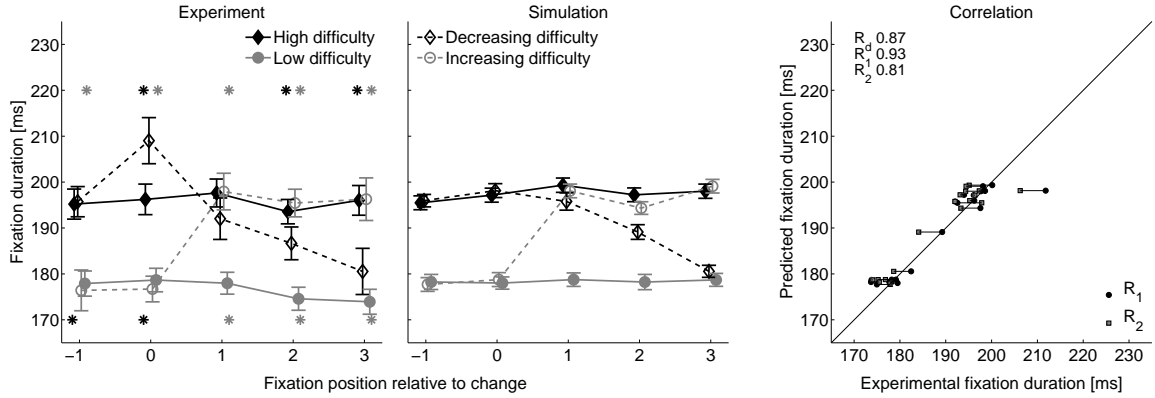


Figure 11. Mean first-fixation durations on a stimulus relative to a change in processing difficulty. Left panel: Experimental data; central panel: Simulation results. Solid lines represent baseline conditions without a change in gap size. In other conditions gap size changed at position 1 (dashed lines). 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 ($p < 0.05$) 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 half of the experimental data. R_d gives the correlation between first and second half of the experimental data. R_1 and R_2 indicate correlations between simulated data and first half and second half, respectively.

smaller in our simulations. In order to assess the quality of simulations, we plotted simulated data against experimental data in the right panel of Figure 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 half 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 half 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 Figure 11 first and second half 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 effect*, has been reported in word recognition (Kennedy, Pynte, & Ducrot, 2002) and reading (Inhoff, Starr, & 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 parafoveal-on-foveal effect in our data. However, in a 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 n 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 Figure 11 is due to a mixture of forward saccades and skipping saccades.

Fixation duration distributions. Histograms of fixation durations with a binwidth of 25 ms are shown in Figure 13 for experimental (left panels) and simulated data (central panels). 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 & 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 Figure 11 and fixation duration distributions evolve accordingly. On all stimulus elements fixation duration distributions are shifted towards longer fixations in the high difficulty baseline (black solid line) compared to 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 towards the low difficulty baseline (positions 2, 3). Inspection of the correlation plots reveals that both halves of the experimental data are well predicted by our model simulations.

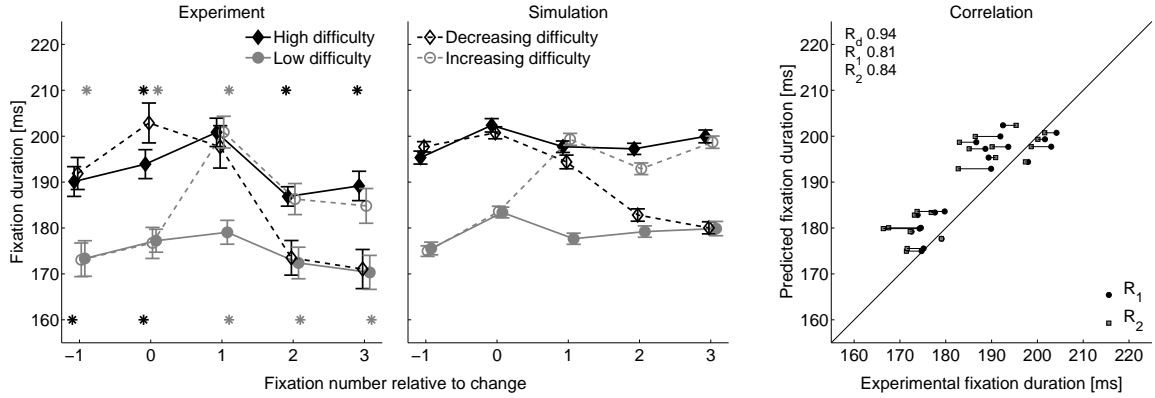


Figure 12. Mean fixation durations of the n th fixation relative to a change in gap size. Left panel: Experimental data; central panel: Simulation results. Solid lines represent baseline conditions without a change in gap size. In other conditions gap size changed at position 1 (dashed lines). Trials 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 ($p < 0.05$) 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 half of the experimental data. R_d gives the correlation between first and second half of the experimental data. R_1 and R_2 indicate correlations between simulated data and first half and second half, respectively.

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, i.e., shorter fixation durations before skipping the next object than before fixating the next object. Skipping benefits have been reported in scanning tasks (Trukenbrod & Engbert, 2012) and during reading, where they are mediated by word frequency or word length (Kliegl & 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 Figure 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

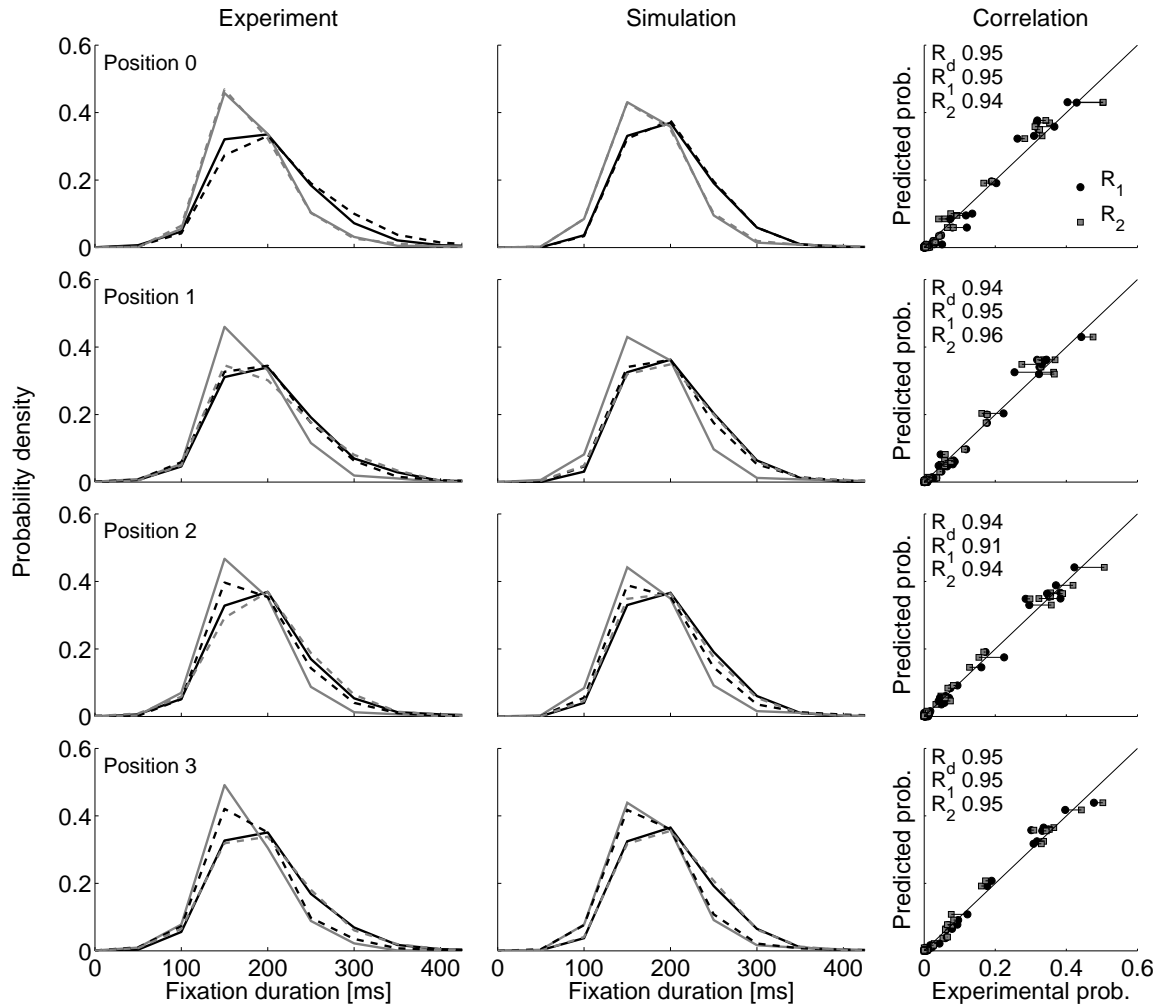


Figure 13. First-fixation duration distributions. Top panel: Histograms before a change (relative position 0); second panel: Histograms immediately after a change (relative position 1); bottom panels: Histograms after adjustment (relative positions 2, 3). Solid lines represent baseline conditions without 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 half of the experimental data. R_d gives the correlation between first and second half of the experimental data. R_1 and R_2 indicate correlations between simulated data and first half and second half, respectively.

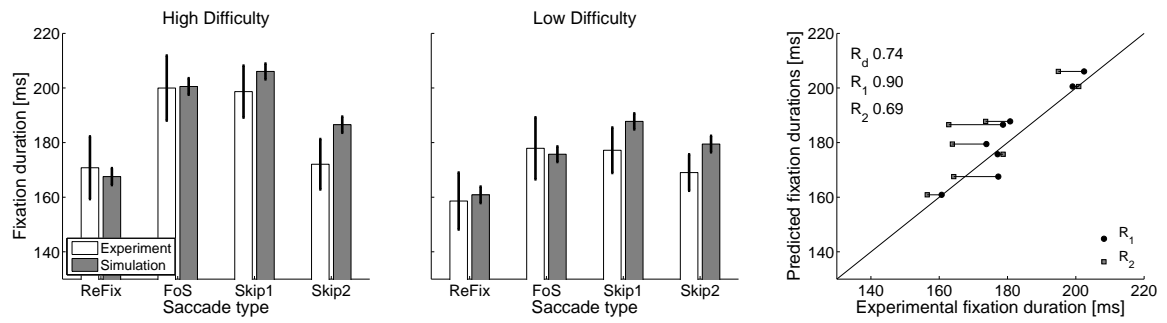


Figure 14. Experimental and simulated fixation durations before refixating an object, making a forward saccade towards the next stimulus, and skipping of the next (skipping 1) and next but one stimulus (skipping 2). Error bars indicate confidence intervals based on Cousineau (2005). Left panel: Fixations in the high difficulty condition; central panel: Fixations in the low difficulty condition; right panel: Comparison of simulations with first and second half of the experimental data. R_d gives the correlation between first and second half of the experimental data. R_1 and R_2 indicate correlations between simulated data and first half and second half, respectively.

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 generate 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 effects of asymmetric control of fixation durations is 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 only observed 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 skipplings, contrary to models assuming sequential attention shifts (Engbert & Kliegl, 2011).

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 only be solved

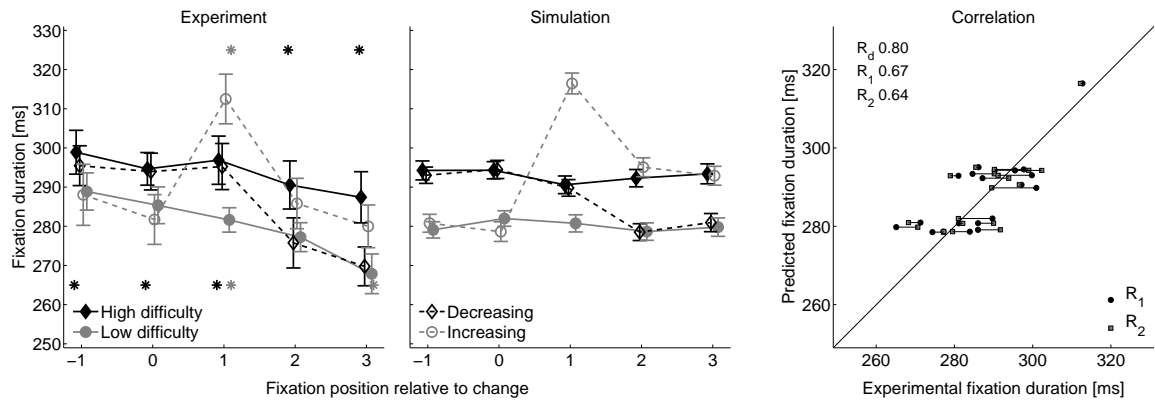


Figure 15. Mean first-fixation durations on a stimulus relative to a change in gap size. Left panel: Experimental data; central panel: Simulation results. Solid lines represent baseline conditions without a change in gap size. In other conditions gap size changed at position 1 (dashed lines). Trials 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 ($p < 0.05$) 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 half of the experimental data. R_d gives the correlation between first and second half of the experimental data. R_1 and R_2 indicate correlations between simulated data and first half and second half, respectively.

successfully 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 Figure 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 expected by the baseline condition consisting of difficult stimulus elements.⁶ In both conditions fixation durations returned to the corresponding baseline on the second stimulus element after a change. The major difference to Experiment 1 was the absence of the Parafoveal-on-Foveal 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 Figure 15. Note that

⁶A mask prevented preview of unfixed stimulus elements. Hence, processing time was the same in both conditions.

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 line). Most interestingly, 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 (position 0), and after a change (position 2) are plotted in Figure 16 (left panels: Experiment; central panels: Simulation; right panels: Correlation plots). Distributions of fixations on difficult symbols were shifted towards 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 importantly, recover the increased skew of fixation duration distributions when processing demands increase.

Summary. Experiment 2 was a gaze-contingent experiment focussing 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 only affect fixation durations 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 spatio-temporal 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 an unified framework. Our model is based on three core principles. First, autonomous saccade timing initiates new saccade programs after random time intervals independent of processing during a fixation. Second, average

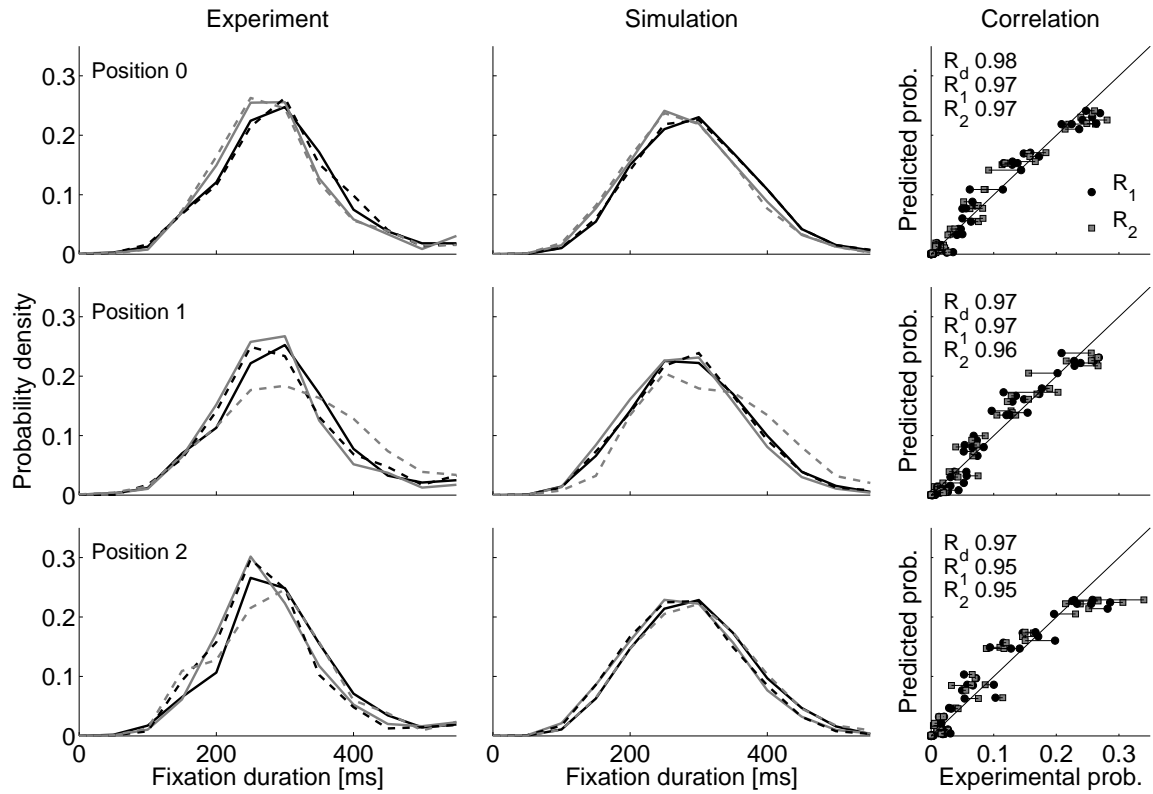


Figure 16. First-fixation duration distributions. Top panel: Distributions before a change (relative position 0); central panel: Distributions immediately after a change (relative position 1); bottom panel: Distributions after adjustment (relative position 2). Solid lines represent baseline conditions 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 half of the experimental data. R_d gives the correlation between first and second half of the experimental data. R_1 and R_2 indicate correlations between simulated data and first half and second half, respectively.

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, i.e., 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; Hoge & Erkelens, 1998; Kolers, 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 only be adjusted 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 Information, simulation of the Staub, 2011, data set) 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, i.e., 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. Parafoveal-on-foveal (PoF) effects, i.e., 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 & Engbert, 2012; Vlaskamp & Hoge, 2006). Currently, ICAT lacks the ability to reproduce this effect, which can be seen in the simulation studies of Experiment 1 (Fig. 11 & 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, i.e., 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: a) PoF effects might reflect a delayed decision to initiate the next saccade program. This interpretation suggests parafoveal inhibition in the ICAT model, i.e., 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 (c.f., Laubrock, Cajar, & Engbert, 2013). b) 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 of the probability to generate forward saccades.

Second, the PoF effect is rather small when compared to the size of immediacy and lag effects. Simulations with SWIFT have demonstrated that the dynamics of the computational model may generate small POE effects. Hence, PoF effects might be inherent to 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 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 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 vs. indirect control of fixation durations

Across tasks, there have been enduring debates 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 to estimate the influence of processing on a moment-to-moment basis. Since immediate modulations of fixation durations can only be achieved by foveal inhibition, parameters of foveal inhibi-

tion uncover to which 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 which 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 encountering high processing demands, 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 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 compared to 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 to control fixation durations in tasks with sequences of fixations. The model reproduced both average fixation duration patterns as well as fixation duration distributions.

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