Modulation of Saccadic Curvature by Spatial Memory and Associative Learning

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Summary

The way the eye travels during a saccade typically does not follow a straight line but rather shows some curvature instead. Converging empirical evidence has demonstrated that curvature results from conflicting saccade goals when multiple stimuli in the visual periphery compete for selection as the saccade target (Van der Stigchel, Meeter, & Theeuwes, 2006). Curvature away from a competing stimulus has been proposed to result from the inhibitory deselection of the motor program representing the saccade towards that stimulus (Sheliga, Riggio, & Rizzolatti, 1994; Tipper, Howard, & Houghton, 2000). For example, if participants are instructed to perform a saccade towards a defined target stimulus and to ignore a simultaneously presented nearby distractor stimulus, a saccade landing on the target typically exhibits curvature away from the distractor (e. g. Doyle & Walker, 2001).

The present thesis reports how trajectories of saccadic eye movements are affected by spatial memory and associative learning. The final objective was to explore if the curvature effect can be used to investigate associative learning in an experimental paradigm where competing saccade targets are retrieved from associative memory rather than being sensory events. The thesis incorporates manuscripts on the following working steps to accomplish this objective: The first manuscript presents the computer software that was written in order to derive measure of saccadic curvature from the recorded eye movement traces. The second manuscript replicates and extends prior reports on the effect of (non-associative) spatial working memory on saccade deviations (Theeuwes, Olivers, & Chizk, 2005). The third manuscript uses a novel associative learning task to demonstrate that changes in saccadic curvature during associative learning comply with the acquisition and extinction of competing associations as predicted by the Rescorla-Wagner model (Rescorla & Wagner, 1972), originally put forward to explain classical conditioning in animals.

Zusammenfassung

Die Trajektorie einer sakkadischen Blickbewegung weist im Allgemeinen eine leichte Krümmung auf. Empirische Befunde sprechen dafür, dass eine gekrümmte Trajektorie vor allem dann resultiert, wenn in der visuellen Peripherie mehrere Reize als potentielle Sakkadenziele dargeboten werden, und so um die Selektion als Sakkadenziel konkurrieren (Van der Stigchel, Meeter, & Theeuwes, 2006). Eine Abweichung der Trajektorie in Gegenrichtung zur Position eines konkurrierenden Reizes ist durch die Inhibition des Motorprogramms erklärt worden, das die Sakkade in Richtung des konkurrierenden Reizes repräsentiert (Sheliga, Riggio, & Rizzolatti, 1994; Tipper, Howard, & Houghton, 2000). Ist eine Versuchsperson zum Beispiel instruiert eine Blickbewegung auf einen als Zielreiz definierten Stimulus hin auszuführen, und dabei einen gleichzeitig dargebotenen Distraktorreiz zu ignorieren, so krümmt sich die Sakkade weg von der Distraktorposition (e. g. Doyle & Walker, 2001).

Die vorliegende Dissertation untersucht Einflüsse des räumlichen Gedächtnisses und des assoziativen Lernens auf die Krümmung von Sakkadentrajektorien. Die übergeordnete Fragestellung ist, ob der Krümmungseffekt Aufschluss über assoziative Lernprozesse gibt, wenn im Lernexperiment Konkurrenz nicht zwischen sensorischen Reizen sondern zwischen konfligierenden Vorhersagen des Sakkadenziels besteht. Die Dissertation umfasst folgende Manuskripte, die einzelne Arbeitsschritte auf dem Weg zur Beantwortung dieser Frage dokumentieren: Das erste Manuskript dokumentiert die Computersoftware, die im Rahmen der Dissertation zur Parametrisierung der aufgezeichneten Blickbewegungssignale programmiert wurde. Das zweite Manuskript repliziert und erweitert empirische Befunde über den Einfluss des räumlichen Arbeitsgedächtnisses auf die Auslenkung von Trajektorien (Theeuwes, Olivers, & Chizk, 2005). Das dritte Manuskript beschreibt ein neues assoziatives Lernparadigma und demonstriert, wie die Krümmung von Sakkadentrajektorien im Lernverlauf durch die Akquisition und Extinktion von Gedächtnisinterferenz moduliert wird. Die beobachtete Modulation steht im Einklang mit den Vorhersagen der Theorie von Rescorla und Wagner (1972) zur Erklärung von klassischer Konditionierung im Tierbereich.

Table of Contents

1 Proposed Manuscripts

I. Koenig, S., & Lachnit, H. (2010a). A Software Package for the Analysis of Eye Movement Trajectories with MATLAB. Manuscript in preparation for submission to Behavior Research Methods

II. Koenig, S., & Lachnit, H. (2010b). Revisiting the Memory-Based Saccadic Curvature Effect. Manuscript submitted to Journal of Experimental Psychology: Human Perception and Performance.

III. Koenig, S., & Lachnit, H. (2010c). Curved Saccade Trajectories Reveal Conflicting Predictions in Associative Learning. Manuscript submitted to Journal of Experimental Psychology: Learning, Memory and Cognition.

2 Saccadic Curvature, Spatial Memory and Associative Learning

Introduction

When human participants perform a saccadic eye movement towards a target stimulus while at the same time a distractor stimulus at a different location competes for selection as the saccade target, the trajectory of a saccade landing on the target will curve away from the distractor. It has been proposed, that this deviation is caused by inhibition of the motor program representing the saccade towards the distracting stimulus (Sheliga, Riggio, & Rizzolatti, 1994; Tipper, Howard, & Houghton, 2000). Converging empirical evidence has provided support for the idea of such inhibitory deselection employed in order to reduce interference during saccade programming (Aizawa & Wurtz, 1998; McPeek, Han, & Keller, 2003; McPeek, 2006; McSorley, Haggard, & Walker, 2006, 2009; Walker, McSorley, & Patrick Haggard, 2006). The series of experiments reported in the present thesis used trajectory deviations in saccadic eye movements in order to investigate interference in spatial memory. Recent evidence has suggested a close link between spatial working memory and the oculomotor system (Theeuwes, Olivers, & Chizk, 2005). It has been demonstrated that saccade trajectories not only deviate away from distracting stimuli (Doyle & Walker, 2001) but also deviate away from *remembered* distractor locations (Theeuwes et al., 2005). However, experiments on the memory-based effect still are sparse (Theeuwes, Van der Stigchel, & Olivers, 2006; see also Godijn & Theeuwes, 2004) and profound knowledge currently is missing.

The experiments reported in the present thesis further investigated the memory-based curvature effect (a) in order to gain insights into the exact nature of the memory representation that interacts with oculomotor programs, and (b) to explore whether the effect can be used as a method to indicate associative memory and learning processes. Experimental results referring to the first question extend prior research on saccade deviations and spatial working memory (Theeuwes et al., 2005, 2006) but discount the current belief that curvature is caused by active rehearsal of a distractor location. With respect to the second question the thesis will present an innovative, new learning task that can be used in order to investigate memory interference acquired during associative learning. Before I turn to a detailed outline of these contributions, the remaining parts of the introduction will briefly summarize our current knowledge about saccadic curvature in general and will outline when and why saccades deviate away from distractor stimuli in the first place.

Saccadic Eye Movements

In humans, visual perception comes in two different qualities. Perception at the point of gaze (within the central 1° of the visual field) is characterized by high visual acuity as well as color perception and corresponds to a dense packing of cone receptors in the fovea centralis. In contrast, peripheral vision exhibits degraded acuity and is achromatic. Saccadic eye movements or *saccades* are fast, jerk-like rotations of the eyeball that redirect the point of gaze in order to select a stimulus for foveal processing. Orienting gaze can also be achieved by using head and body movements, but in humans the saccade represents the major mechanism for deploying overt visual attention. In every day life we behave in a world cluttered with visual information and typically perform several saccades per second (varying with the exact situation). Selecting the target for the next eye movement thus may constitute the most frequently made decision during our life time.

Curvature of Trajectories

Saccades have been characterized as *ballistic* movements, because they are not guided by visual feedback during the eye rotation (Carpenter, 1988; Becker, 1991). A saccade rather “catapults” the eye to its new position, reaching rotational velocities of up to 800°/s (depending on the distance the eye traveled). These high velocity movements are accompanied by a suppression of visual thresholds starting from 50ms prior to movement onset and enduring throughout the saccade (Latour, 1962; Findlay & Gilchrist, 2003). Despite the obvious need to keep saccade duration as short as possible, the exact way the eyes travels during a saccade typically does not follow a straight line but rather shows some curvature instead.

Early studies reported curved trajectories primarily with reference to oblique saccades, which in general show more curvature than vertical or horizontal saccades (Yarbus, 1967; Bahill & Stark, 1975; Viviani, Berthoz, & Tracey, 1977). Subsequent experiments provided evidence, that saccadic curvature is caused by interference induced by multiple conflicting stimuli competing for selection as the saccade target (for review see Van der Stigchel, Meeter, & Theeuwes, 2006). For example, if rhesus monkeys are trained to saccade to an odd colored target presented amongst a variable number of homogeneous distractors, the trajectories of saccades towards the target exhibit pronounced curvature and variability as compared to a distractor absent condition that presents the target only (Arai, McPeek, & Keller, 2004).

Covert Visual Attention

Saccadic curvature attracted major interest because it provided empirical evidence for a common physiological substrate of covert attention and eye movements as proposed in the *premotor theory of attention* (Rizzolatti, Riggio, Dascola, & Umiltá, 1987; Rizzolatti, Riggio, & Sheliga, 1994). In a now classic series of experiments Sheliga and colleges (Sheliga, Riggio, & Rizzolatti, 1994, 1995; Sheliga, Riggio, Craighero, & Rizzolatti, 1995) provided human subjects with the task to saccade to a target location while covertly attending to a different location. Under these circumstances saccade trajectories consistently curved away from the attended location. The authors' interpretation was as follows: If covert visual attention influences eye movement trajectories, attention is not some supra-modal function of the brain but rather seems to share the same neural circuits involved in oculomotor control. One explanation for the fact that saccades deviated *away* from the attended location was formulated in the *suppression hypothesis* of saccadic curvature (Sheliga et al., 1994). From the perspective of the premotor theory, allocating covert visual attention towards some peripheral stimulus basically is the same as programming an eye movement towards that location that then is withheld from execution because participants are instructed to keep central fixation and not to move their eyes. Curvature away from the attended location is supposed to result from the suppression of the saccade program towards that location.

Various experiments in succession to the studies on attention and eye movements have shed further light on the saccadic curvature effect (for review see Van der Stigchel et al., 2006; Van der Stigchel, 2010). For example, it has been demonstrated, that the deliberate, endogenous allocation of attention is no necessary precondition for saccades to deviate away from a location. Rather it seems that any distractor stimulus presented together and in conflict with the saccade target can cause the eyes to deviate away, even if the distractor is irrelevant, and task demands do not require any deliberate orienting (Doyle & Walker, 2001).

Population Coding

Why do saccades curve away from distractors? Theoretic accounts build on two major principles: population coding of saccade programs and inhibition of distractor related activity. The first principle refers to the assumption that the internal representation of a saccade is *population coded* by the activity of multiple neurons in oculomotor structures such as the superior colliculus (Tipper, Howard, & Houghton, 2000). The superior colliculus (SC) has been shown to contain a topographic representation of oculomotor space in retinal coordinates (Sparks & Hartwich-Young, 1989). In the intermediate layers of the SC *burst neurons* show activity time locked to saccade onset while *buildup neurons* show increasing activity in an interval preceding the saccade. The movement fields of these neurons correspond to saccades of defined directions and amplitudes. For example, a neuron is maximally active for a rightward saccade (direction = 0°) with an amplitude of 10° and activity of that neuron gradually falls off for saccades with increasing deviation from this optimal saccade metric. The map is topographic, because on a rostral-to-caudal axis neighboring neurons code for saccades of similar amplitudes and on a medial-to-lateral axis neighboring neurons code for similar directions. Programming a saccade elicits widespread activity involving a population of neurons, with maximum activity at the location that codes for the exact saccade metrics, and a gradual decline of activity with increasing distance to that activity peak. Furthermore, the map is retinotopic because, within the SC, motor layers and sensory layers form a sandwich structure in which motor fields map onto receptive fields. A peripheral stimulus of defined position elicits maximum neural activity at a defined location in the sensory map and a saccade towards that stimulus elicits activity at the same location in the motor map.

Distractor Inhibition

The second mechanism to explain curvature originally was offered as the *suppression hypothesis* by Sheliga et al. (1994; see above). These authors assumed, that the instruction to perform a saccade towards a defined target location while avoiding a saccade towards a competing location resulted in inhibition of the competing motor program. A similar idea was formulated in Tipper et al.'s (2000) *reactive inhibition* account. If the competing saccades are represented by population codes as outlined in the previous section, these population codes might overlap. Suppression of the distractor related motor program then inhibits some neurons coding for the target location as well and as a result the peak of the target related activation gradient shifts away from the distractor location. This peak-shift in target related activity is proposed to cause initial deviation away from the distractor. Curvature back towards the actual target position at the end of the saccade has been suggested to be caused by cerebellar control mechanisms (McSorley, Haggard, & Walker, 2004; Quaia, Lefevre, & Optican, 1999).

Behavioral evidence for the existence of a *reactive inhibition* process as proposed by Tipper et al. (2000) comes from studies showing a positive correlation between saccadic latency and curvature away (McSorley, Haggard, & Walker, 2006, 2009; Walker, McSorley, & Haggard, 2006). If the target and the distractor stimulus appear at unpredictable locations, and thus any selection process cannot start prior to their simultaneous onsets, short latency saccades directed at the target curve *towards* the distractor location while long latency saccades curve *away*. This effect is in line with the hypothesis, that inhibition of the distractor site quickly builds up after the distractor evoked some activity initially. Neurophysiological findings also seem to support the inhibition account. Micro-electrode recordings in the monkey's frontal eye field and superior colliculus have shown that for saccades that curve away from a distractor stimulus activity at the distractor site is suppressed below the level of activity observed during straight saccades. On the contrary, for saccades that curve towards a lateral distractor, activity at the distractor site appears to be elevated (McPeek, Han, & Keller, 2003; McPeek 2006).

In summary, the empirical evidence outlined above strongly supports the view that saccade deviations away from distractor stimuli are caused by the inhibition of distractor related saccade programs. The experiments outlined above have shed light on the saccade target selection process when two *sensory* stimuli compete for selection. As suggested by the premotor theory (Rizzolatti et al., 1987, 1994), distractor induced saccade deviations strongly support the idea that exogenous and endogenous attention is represented at the level of oculomotor control. As reported at the outset of the introduction, these findings were extended by two recent experiments of Theeuwes et al. (2005, 2006; see also Theeuwes, Belopolsky, & Olivers, 2009) that yielded evidence in favor of a close link between the oculomotor system and spatial working memory. In these experiments any distractor stimulus was absent at the time of saccade execution, but rather saccades deviated away from *remembered* locations where a distractor stimulus had been presented some time prior to the saccade. Besides the experiments of Theeuwes et al., further empirical evidence on the influence of spatial memory on saccade deviations currently is missing. The starting point of the experiments in the present thesis thus was to further develop our current understanding of the exact nature of the memory representation that interacts with oculomotor programs. In a second step, we examined the memory-based deviation effect on a completely new terrain, and explored how the saccade target selection process, and in turn saccadic curvature, was affected by the acquisition of conflicting predictions in associative learning. The next chapter will give a detailed outline of the contribution of these experiments.

Outline of the Present Thesis

The thesis spans three manuscripts concerned with the curvature of saccade trajectories. The first manuscript (Chapter 3) describes the computer software that I have programmed in order to compute measures of saccade deviations from the recorded eye movements traces. This work was necessary because available analysis software did not provide appropriate response parameters. The software will be available to other researchers and hopefully contribute to a more standardized usage of trajectory measures. All experiments reported in the present thesis used the software for signal conditioning as well as response identification and parametrization.

The second manuscript (Chapter 4) is concerned with the nature of the memory representation that affects saccade trajectories. The predominant view has been that the memory trace elicited by brief presentations of a distractor stimulus is short-lived without active retention strategies such as rehearsal, and that any measurable effect on saccade trajectories disappears after 400-800ms (Godijn & Theeuwes, 2004). If, however, the activity elicited by the distractor is endogenously sustained because participants are instructed to remember the distractor's location, saccade trajectories exhibit distractor induced curvature 1000ms after distractor offset (Theeuwes et al., 2005, 2006). A series of three experiment was conducted to investigate the following questions: (1) Does the effect really rely on deliberate retention strategies such as rehearsal of the distractor location? (2) Which factors, besides active retention, contribute to the sustain of distractor interference? (3) Does the memory trace of the distractor that influences trajectories consist of sustained activation or rather suppression? In sum, the second manuscripts reports that distractor induced deviations can be observed over two seconds after distractor offset even if participants do *not* engage in any active retention strategies. We propose that memory-based curvature is caused by sustained inhibition rather than excitation of the distractor.

Up to now, there have been no prior reports on the modulation of saccadic curvature by associative memory and the third manuscript's major contribution is to demonstrate this associative curvature effect. The manuscript reports how trajectories acquired curvature as participants acquired conflicting predictions in an associative learning task. A connectionist network simulation revealed that observed changes in saccadic curvature complied with the acquisition and extinction of memory interference predicted by the Rescorla-Wagner model learning rule (Rescorla & Wagner, 1972; Wagner & Rescorla, 1972), originally put forward to explain classical conditioning in animals. It is proposed that measuring saccade deviations in this new oculomotor learning task provides a valuable, innovative method to investigate memory interference in associative learning.

Chapter 3: Computer Software

Chapter 3 documents the computer software that I have written in order to compute parameters of curvature from the recorded eye traces (Koenig & Lachnit, 2010a). Eye tracking manufacturers typically offer integrated systems that provide the eye tracking hardware together with a software program for post-experimental data analysis. However, the commercial software of major manufacturers at the time of writing does not provide any parameters of saccadic curvature (e. g. SR Research Ltd., Ontario, Canada and SensoMotoric Instruments GmbH, Berlin, Germany). Most published studies so far have relied on a combination of commercial programs to detect saccades in the recorded eye traces, and custom written software to compute parameters of curvature for the identified saccades. Many labs seem to use their own custom software and also use diverging measures of saccadic curvature (for a review see Ludwig & Gilchrist, 2002).

The Eye Movement Analysis (EMA) software described in Chapter 3 provides a collection of computer programs written in MATLAB© (The MathWorks, Natick, MA, USA). The EMA software imports recorded traces of vertical and horizontal eye position as well as pupil size and provides functions for signal denoising and artifact filters. Algorithms for the detection and parametrization of eye responses use the conditioned signal to compute responses parameters for saccades, fixations and blinks. Several functions focus on the identification and parametrization of saccade trajectories and the software implements different measures of saccadic curvature described by Ludwig and Gilchrist (2002). The software package contains the Matlab Eye Browser (MEB), a graphical user interface that can be used to to depict and edit recorded eye traces and that provides user-friendly, menu-based access to most functions in the software package.

The EMA software may contribute to a unified approach in measuring saccade trajectories while commercial software currently neglects these saccade parameters. All functions in the EMA library are written in the MATLAB programming language widely used as a data analysis tool in the field of neuroscience. The EMA program code provides a detailed documentation of how response parameters are computed. Also, the library should be easily extendible to adopt to custom needs. We hope that EMA's availability to other researches will help to standardize the use of saccadic curvature measures in the scientific literature.

Chapter 4: Empirical Study I

The three experiments described in Chapter 4 were conducted in order to shed further light on the processes involved in the memory-based curvature effect (Koenig & Lachnit, 2010b). In the original studies of Theeuwes et al. (2005, 2006) saccades curved away from a lateral location that contained no distractor stimulus at the time of saccade execution but a distractor stimulus was transiently presented 1000ms preceding the saccade. Curvature under these circumstances indicated how spatial memory for a distractor location interfered with the preparation and execution of an oculomotor response. In these experiments, at the start of a trial a stimulus was presented in the left or right visual field. While maintaining central fixation, human participants were instructed to remember the stimulus location in order to complete a test for the exact position at the end of the trial. Vertical saccades performed during the retention interval curved away from the side of distractor presentation. The link between spatial working memory and the oculomotor system that was suggested by Theeuwes et al., (2005, 2006) referred to the assumption that activity representing the distractor was endogenously sustained by participants in order to remember the distractor location and therefore became inhibited when the saccade to the target was to be executed in the retention interval.

The experiments reported in Chapter 4 were conducted in order to further investigate the nature of the memory representation that causes the eyes to deviate away from a remembered location. In Experiment 1 we measured saccadic curvature induced by perceptual distractors presented together with the saccade target in order to provide a reference condition for the memory-based effect investigated in Experiment 2 and 3. With these perceptual distractors we observed the following results: (1) In accord with the distractor inhibition account, in general, saccades deviated away from the lateral distractor. (2) The amount of deviation away increased with saccadic latency and short latency saccades in some conditions deviated *towards* the distractor. This result confirmed the idea that reactive inhibition (Tipper et al., 2000) was gradually applied to the distractor location after distractor onset. (3) The distractor had a more pronounced influence on saccade trajectories in a group with a small target-to-distractor distance as compared with a group presented with a far distractor. This observation was in line with the population coding hypothesis (Tipper et al., 2000) outlined in the introduction because more overlap in population coded motor programs can be expected if target and distractor are presented in close proximity.

In Experiment 2 we replicated the original memory-based curvature effect reported by Theeuwes et al. (2005). We demonstrated that saccades curved away from a location retained in memory when there was a test for the remembered location after saccade execution. As also reported by Theeuwes et al. (2005, 2006), and again in accord with the population coding hypothesis, a distractor presented in the same hemifield as the saccade target elicited more saccade deviations than a distractor in the opposite hemifield (supposedly because of more overlap in motor programs in the former condition). The memory representation that caused saccade deviations, however, remained somewhat unclear. On the one hand, it is possible that trajectory deviations were caused by the active rehearsal of the distractor location. Sustained endogenous activity then conflicted with the saccade goal in the retention interval and became inhibited right before the saccade was executed (as suggested by Theeuwes et al., 2006). On the other hand, the effect may have been caused by inhibition applied to the distractor location right after distractor onset, because participants were instructed not to look at the distractor, and also knew that the distractor never was the target of the subsequent saccade. If this inhibition was sustained until the time of saccade execution (possibly with some passive decay in the retention interval), saccades also would have deviated away from the distractor location. The next experiment was conducted to evaluate these different hypotheses.

In Experiment 3 a 150ms distractor stimulus was presented about two seconds prior to saccade execution. The vertical position of the distractor specified the direction of the subsequent saccade. For example, a distractor in the upper right quadrant instructed participants to prepare for a straight upward saccade two seconds later on, but yet to keep central fixation. The exact *lateral* position of the distractor within quadrant, or whether the distractor was actually presented to the left or right, however, was irrelevant to the task, and any encoding or “remembering” of the distractor location should have been automatic rather than caused by a deliberate process. At least, in this design participants were unlikely to actively sustain covert attention to the distractor in order to rehearse its exact location as suggested by Theeuwes et al. (2005, 2006) for the memory test condition in Experiment 2. In contrast, covert attention in this situation can rather be expected to shift towards the anticipated target location in preparation of the eye movement (Deubel & Schneider, 1996).

Despite the assumed absence of active rehearsal, vertical saccades, performed two seconds after the lateral stimulus had disappeared, curved away from the distractor location. Our results contradicted prior reports that any measurable distractor effect on saccade trajectories disappeared after an interval of 400-800ms (Godijn & Theeuwes, 2004) if participants do not engage in active retention. We interpreted these results in favor of an automatic stimulus encoding that elicited inhibition of the distractor related motor program right after distractor onset and gave rise to an inhibitory memory trace gradually decaying after distractor offset but still featuring enough residual inhibition to interfere with saccade execution two seconds later. In contrast to Theeuwes et al. (2005, 2006) our results thus suggested that sustained suppression rather than sustained activation caused the curvature effects in Experiment 2 and 3.

Chapter 5: Empirical Study II

The experiments reported in Chapter 4 confirmed and extended previous reports on the influence of (non-associative) spatial working memory on saccade trajectories. However, there have been no prior reports on whether saccade trajectories are influenced by *associative* learning and memory. The rationale behind the empirical study reported in Chapter 5 was to use the saccadic curvature effect in order to explore a new a method to investigate associative learning and memory (Koenig & Lachnit, 2010c).

We used a learning task that was designed in analogy to a classical conditioning experiment, where the conditioned stimulus (CS) is repeatedly paired with an unconditioned stimulus (US), and in the course of training the CS gradually acquires the potential to elicited a conditioned response (CR) that indicates the acquisition of a CS-US-association. Our oculomotor conditioning paradigm required human participants to learn which of several central cues (CS; pictures of fruit) predicted which of three different target locations (US) in the upper visual field. Saccades, elicited by the central cues and directed at the correct location in anticipation of the target, thus represented a conditioned response indicative of acquired cue-target associations. Also, depending on the training schedule, sometimes a cue not only predicted that the target could exclusively occur at one location, but also retrieved the prediction, that the target was somewhat less likely to occur at a different, competing location. For example, if participants learned that cue A was followed by an upper-right target (A+R), but the compound cue AX was trained with an upper-mid target (AX+M), referring to the Rescorla-Wagner (1972) model as the “most widely accepted description of associative changes during classical conditioning” (Gluck & Bower, 1988), cue A should acquire a strong A-R association but initially also should develop an interfering A-M association (where letters A and X denote pictures of different fruit and M and R denote the mid and right target position respectively). Note, that learning which includes the prediction of multiple competing outcomes is beyond the original scope of associative learning theories. In regular classical conditioning paradigms humans or animals typically predict the presence versus absence of one single outcome. It has been claimed that the Rescorla-Wagner theory might easily extend to the prediction of multiple outcomes such as in human category learning (Gluck & Bower, 1988; Shanks, 1991), however *direct* empirical evidence on one fundamental assumption of this claim is sparse. The assumption of this connectionist approach to classification learning is that, with training A+R, AX+M as in the example above, *both* associations, A-R and A-M, are adjusted simultaneously after the outcome has been presented. For example, learning in A+R trials is not restricted to an increase in the A-R association but the absence of outcome M should at the same time decrease any A-M associations acquired in AX+M trials. The major contribution of the manuscript in Chapter 5 is the demonstration that saccadic eye movements in fact yield information about these proposed concurrent weight changes. For example, in A+R trials the frequency and latency of a correct anticipatory saccade towards the trained location R yields a measure of the strength of the A-R association, and the curvature towards or away from outcome M of the very same saccade yields a measure of the concurrent A-M association. In the experiment reported in Chapter 5 we observed how these measures changed during training and provided information about the interference induced by concurrent, conflicting associations. The following paragraphs will give a brief overview over the main results.

Acquisition of conditioned responding was evident in a gradual increase in the frequency of correct anticipatory saccades as well as a gradual decrease in saccadic latency relative to cue onset. Most importantly, saccades acquired curvature away from the locus of associative interference. With reference to the previous example, during training A+R, AX+M, saccades elicited by A alone correctly landed on the upper-right location in anticipation of the target but also *curved* to the right, i. e. away from the incorrect prediction made by the A-M association acquired in AX+M trials. At the same time, correct vertical saccades elicited by AX curved to the left, i. e. away from the interfering right location predicted by the excitatory A-R association.

Simulations of a connectionist network model revealed that observed changes in curvature complied to changes in associative memory as predicted by the learning rule of the Rescorla-Wagner model (Rescorla & Wagner, 1972; Wagner & Rescorla, 1972). With reference to the prior example, the Rescorla-Wagner model predicts, that the A-M association interfering with the correct A-R association will initially be acquired but will subsequently extinguish in the course of further training. In compliance with this prediction, A-elicited saccades initially acquired curvature away from the incorrect upper-mid outcome but curvature then decreased again in the second half of training.

To explain the direction of the observed curvature effect, we propose a simple mechanism that takes two excitatory input signals referring to the correct and incorrect cue-target associations. The mechanism then inhibits the interfering (the weaker) prediction to an extent matching the strength of the associative weight that led to the incorrect prediction in the first place. The mechanism in general is compatible with the *reactive inhibition* account of saccadic curvature proposed by Tipper et al. (2000) and we accordingly suggest, that curvature observed in our experiment was caused by the reactive inhibition of an excitatory association. However, although observed saccadic curvature was in accord with the inhibition hypothesis, we found no evidence for the acquisition of *inhibitory associations* as predicted by the Rescorla-Wagner model. Extending our previous example, after training A+R, AX+M, X+M, cue X should have acquired a negative X-R association that inhibits the upper-right target whenever cue X is presented alone. Contrary to these predictions, in X+M trials saccades directed at the mid target did not curve away from the right target but rather exhibited no curvature at all. If an inhibitory X-R association was acquired, this inhibition (a) did not reach oculomotor structures or (b) was not active in X-alone trials. The latter explanation is consistent with the notion first put forward by Konorski (1948) stating that conditioned inhibitors may need an excitatory context in order to take effect.

In sum, the manuscript reports the first experimental evidence for the modulation of saccadic curvature by associative memory. To our knowledge, the experiment also offers the first direct evidence for concurrent weight changes in classification learning with multiple outcomes. The experimental paradigm may provide a valuable new method for accessing the set of concurrent associations that a cue acquires in the course of classification learning.

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3 A Software Package for the Analysis of Eye Movement Trajectories with MATLAB

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Abstract

The current paper presents the Eye Movement Analysis (EMA) software package, a library of MATLAB functions for the post experimental analysis of eye tracking data. The library imports records of vertical and horizontal eye position traces as well as pupil size data and provides basic functions for signal conditioning such as signal denoising and artifact filters. Several functions can be used to detect and parametrize eye responses such as saccades, fixations, blinks. The library includes a graphical user interface (GUI) that can be used to browse the eye record on a trial by trial basis. Most of the MATLAB functions can be called from this graphical interface without the need for using the MATLAB command prompt.

Our development focus thus far has been on the identification and parametrization of saccadic eye movements and most elaborate functions are devoted to this topic. The main focus has been on the analysis of saccade trajectories. Measures of saccadic curvature or the initial direction of saccade trajectories to data have been ignored by commercial software for eye movement analysis. We hope that EMA's availability to other researches will help to standardize the use of saccadic curvature measures in the scientific literature.

KEYWORDS: Eye Movements Analysis; Saccades; Fixations; Blinks

Introduction

Video based eye tracking systems currently are used as one of many standard techniques in the inventory of psychological and neuroscience methods (Duchowski, 2003) and eye movement research has led to valuable insights within the fields of attention, scene perception, reading, visual search and motor control (Findlay & Gilchrist, 2003). One typical scenario for the analysis of eye movements is the storage of continuous eye position traces during an experimental recording session and the post-experimental identification and parametrization of distinct ocular responses within the recorded traces. Commercial software for ocular response parametrization typically is developed as part of an integrated system that bundles the software with specific eye tracking hardware. Commercial software packages, however, often lack certain response parameters and accordingly the software that is presented in this paper was born out of our need to compute elaborate measures of saccadic curvature that have been discussed by Ludwig and Gilchrist (2002; see also Van der Stigchel, Meeter, & Theeuwes, 2006), but have not been included in any published software package yet. We hope that our software will provide a useful tool for other researchers as well and will contribute to a standardization of curvature measures that is currently missing.

The software consists of a function library called EMA (Eye Movement Analysis), that is programmed using the MATLAB (2007a, The MathWorks) scripting language, and its use should be straightforward for anyone moderately familiar with MATLAB. However, because the EMA library does include a graphical user interface (also programmed in MATLAB), most of the functions can be accessed in a user-friendly way using graphical menus. In the present paper, EMA's capabilities mainly are demonstrated from the perspective of this graphical user interface. The last section of the paper briefly discusses EMAs scripting capabilities and gives examples on how to batch process multiple eye records using underlying MATLAB functions.

The Matlab Eye Browser

The Matlab Eye Browser (MEB) provides a graphical user interface to the EMA library. The program can be used for data inspection and manual editing of ocular responses. Most functions in the EMA library can be called via graphical menus. You can invoke the browser by typing `>> MEB()` at the command prompt. Figure 1 shows MEB in action. The main screen consists of a plot of instantaneous gaze position in the calibration plane (upper left graph), time plots of gaze position, horizontal and vertical velocity, total velocity and acceleration, as well as information about file header, trial, and parametrization of eye events (mid to bottom left area).

Trial based navigation. The MEB program is designed to depict the eye data record on a trial-by-trial basis. If recording messages were sent to the eye tracker during the recording session to mark the start and end of each trial, EMA's import function will use this information to partition the record accordingly. If the record does not contain such information, the import function will partition the record into even intervals relying on user defined parameters of trial length and trial offset. Trials can be selected within the `Trials` panel at the left using the arrow buttons to choose the next or previous trial respectively or by entering the trial number. The left-most field depicts a text string coding for the trial type of the selected trial if such information had been sent to the eye tracker at the start of each trial during the recording session.

Gaze position plot. The upper-left graph plots instantaneous gaze position in the calibration plane where each dot in the scatter plot represents one sample. Gaze position is rescaled to move the origin to the center of the calibration plane while most video based eye trackers record gaze position with the origin at the top left corner. When importing data, one can specify new minimum and maximum values for the calibration plane to transform gaze position accordingly. In Figure 1 for example, gaze position has been scaled to units of millimeters (stimuli were presented on a 22” CRT-monitor measuring 400 x 300 mm).

Gaze position time plot. The upper-right graph shows traces of horizontal and vertical gaze position as a function of trial time. In this time plot gaze position traces are color coded to match the color of the horizontal (pink) and vertical (blue) axis within the gaze position plot to the left. As can be seen from Figure 1, the time plot contains a green and red vertical line which mark start (green) and end (red) of the section of the gaze position trace which will be depicted in the gaze position plot to the left. Vertical start and end markes can be moved in the time plot to select a new interval by performing a point, click and drag mouse action. Again, colors of the vertical start and end markers in the gaze position time plot correspond to colors in the gaze position plot to the left, where a green and red dot mark the start and end of the selected sequence of eye movements respectively.

Target stimulus positions. Eye tracking experiments may present distinct visual stimuli to elicit ocular responses. If the program is provided with information on target stimulus positions, MEB uses this information to overlay stimulus positions in the gaze position plot. Target positions are depicted as circles that contain an arbitrary single-character identifier. The example in Figure 1 depicts data from an experiment where a cued target was presented at one out of three possible locations in the upper hemifield. The trial code at the Trial panel shows that, in the current trial, the target appeared at the mid target position (coded as “2”). Besides the graphical representation of target positions EMA will use target information for response parametrization such as computing post saccadic error (see below).

Gaze velocity and acceleration. Component velocities *xy* and *yv,* as shown in the second time plot, are imported from traces stored by the eye tracker or computed from the gaze position signals in 2D or 3D velocity space (depending on the program settings). The third graph on the right depicts instantaneous gaze velocity computed as $gv=\sqrt{xv^2+yv^2}$, where *gv*, *xv* and *yv* denote total, horizontal and vertical gaze velocity respectively. Gaze acceleration as shown in the fourth time plot is computed as the first derivative of gaze velocity. The velocity graph mirrors the velocity signal at the x-axis to yield a better graphical representation (total gaze velocity in fact is of positive sign only).

Pupil size. Figure 2 depicts how the context menu (right mouse-click on graph) can be used to select the data source for a time plot. In this example MEB is configured to primarily depict pupil information and MEB thus may also be used for analyzing experiments that measure pupil size as their primary dependent variable. Time plots in Figure 2 are configured to show pupil size, velocity and acceleration as well as gaze position (from top to bottom).

File header. The mid-left panel called `File` depicts selected meta information stored in the file header such as the name of the experiment, the subject code, and date and time of the recording session.

Events and responses. The `Event` panel at the bottom left groups information about different kinds of events that can occur during the recording session. An event is defined by its start and end time during the recording session and may be related to either the occurrence of trials, stimuli or responses. One can select the type of event from the top-left pull-down menu of the `Event` panel. In Figure 1, for example, the entry SAC has been chosen from the pull-down menu to select saccadic responses to be depicted in the `Event` panel. Responses are numbered consecutively for the whole experimental session and one can choose a specific saccade by selecting the respective number from the scroll menu to the right. In Figure 1 saccade 318 that occurred in trial 36 has been selected. One can use the keyboards cursor keys to easily navigate trials and responses where up and down keys will select the previous and next response respectively while the left and right keys will select the previous and next trial. By selecting a specific response MEB will perform several actions: (1) The bottom-left list of response parameters will update to represent the selected saccade. (2) The green and red vertical markers in the time plots will jump to the start and end of the selected response respectively, and (3) the gaze position plot will be updated to show the trajectory of the selected response.

Basic Steps of Eye Movement Analysis

The following section will give a basic overview of the typical processing steps that are performed in order to yield a parametrization of ocular responses using MEB's graphical user interface.

Importing data

File formats. Currently, importing files from two different eye tracking manufactures, SensoMotoric Instruments as well as SR-Research, is supported. The EMA library should be easily extended with importing routines for other eye tracking systems since the EMA source code provides plain examples of how these routines are written. From the MEB program menu, the `File > Import` entry opens a file selection menu and lets one choose the desired file format.

Configuration scripts. You probably need to run a configuration script prior to file import that configures the import routine to correctly identify the trial structure within the data set. Furthermore, this configuration specifies settings such as stimulus positions in the experiment, how to rescale pupil and gaze position data, which filters to apply, how to delete blink related artifacts and which responses to process. A configuration script is executed from the program menu with the `File > Run` entry. Figure 3 gives an example of what a configuration script may look like. More detailed information is given in the program manual that is distributed with the software.

Signal conditioning

After importing the raw data several steps can be taken to enhance signal quality and as a result to facilitate the correct parametrization of ocular responses. Signal conditioning steps may include identification and exclusion of blink-induced signal distortions, heuristic filtering of gaze position traces, smoothing of gaze velocity as well as spline-interpolation and smoothing of pupil size.

Filtering artifacts. During a video based eye tracking session the recorded gaze position and pupil size signals are transiently lost with every eye blink. Closure of the lid at first causes a gradual decrease in recorded pupil size up to some point when the tracking algorithm completely loses the pupil. These artificial changes in pupil size at the outset of each blink also affect the recorded gaze position which is derived from the position of the pupil center. The same signal distortions occur when the lid gradually opens at the end of a blink. Figure 4a shows examples of these pre- and post blink signal distortions. Artifacts may become problematic for several reasons: (1) They may be mistaken for eye movements by the algorithm that detects saccades, (2) they may artificially fragment fixation periods into multiple fixations although in fact the gaze has been stable during the blink and (3) they may distort signal averages of pupil size. To deal with these artifact-induced problems, the EMA artifact filter deletes distortions from the gaze position traces and spline interpolates the pupil size signal as exemplified in Figure 4b. Blink induced signal distortions are detected by a combination of the following criteria: (1) pupil size falls below a defined minimum threshold; (2) pupil velocity and/or acceleration exceed critical values; (3) gaze position drops out of the valid calibration range; (4) gaze velocity and/or acceleration exceed critical values. After these first steps of artifact identification the routine additionally searches pre- and post artifact intervals of defined width to reapply the artifact filter with more liberal criteria. At last, every artifact-to-artifact interval that is shorter than some defined criterion is deleted from the record. The filter can be accessed with `Filter > Artifacts` from the MEB program menu.

Gaze position filter. Having deleted blink related artifacts from the gaze position record, two filters can be applied to the gaze position and gaze velocity signal, respectively. The EMA library contains a function that implements the heuristic filter algorithm described by Stampe (1993). For each sample in the gaze position signal a decision heuristic is applied to whether the sample is noisy or not and thus has to be replaced by either the preceding or following sample. This heuristic substitution rule leaves the signal with intervals of two successive samples that have identical values after the first filtering stage. These artificial two-sample flat-topped artifacts then are detected and corrected in a second filter stage. The mechanism is reapplied several times efficiently deleting different forms of noise that have been described to occur with video based eye tracking methods. Figure 5 demonstrates how the heuristic filter efficiently eliminates “ringing noise” (Stampe, 1993) at the end of saccades. The heuristic filter can be accessed from the MEB program menu under the `Filter > Gaze` entry.

Gaze velocity filter. The gaze velocity trace may be smoothed using a multiple-pass moving average filter. Filtering of gaze velocity drastically improves the quality of the gaze acceleration signal which is computed from gaze velocity. The filter cannot be accessed under any menu entry but is automatically applied whenever gaze velocity is (re-)computed and EMA is configured accordingly.

Pupil size filter. One part of the pupil filter has already been demonstrated as an effect of running the artifact filter. Blink intervals during which the eye tracker could not record the pupil size are spline interpolated to yield a continuous signal as shown in Figure 4b. Additionally a multiple-pass moving average filter can also be applied to further smooth the pupil size signal and at the same time enhance the quality of derived velocity and acceleration signals.

Response Identification and Parametrization

After signal conditioning you may run several routines to automatically search the eye traces in order to detect ocular responses. For each response a set of parameters is computed to quantify the response characteristics.

Artifacts. As described above, running the artifact filter will identify intervals where the tracker could not record any valid eye signal. For each of these intervals of temporary signal loss EMA computes parameters as listed in Table 1. You may use the artifact parameters in order to decide whether a trial has to be excluded from later analysis because there were too many blinks per trial (`itrl`), the duration of signal loss (`dur`) was too long, or because there was a change in gaze position during signal loss (`dltxy`, `amp`), i.e. the tracker lost signal during an eye movement, the artifacts actually masks a saccade and the trial hence has to be treated as missing in later statistical analysis. Besides this use for the identification of missings, artifacts may be interesting in their own right for every study concerned with the analysis of blink frequency. Computation of artifact parameters can be accessed with `Analyze > Artifacts` from the MEB program menu.

Saccades. Detection of saccades is carried out using a multiple-pass search algorithm. At first pass the algorithm identifies local gaze velocity peaks that exceed a critical minimum. Local maxima may consist of multiple samples forming a flat-topped peak but this peak must not exceed a critical width. Local minima enclosing a velocity peak are taken as preliminary saccade limits. The algorithm then merges adjacent saccades if the interval separating two saccades does not exceed a critical length and gaze velocity in the inter-saccade interval does not fall below a critical minimum. With this procedure multiple movement components may be merged within one single movement, effectively and flexibly dealing with pre- and post-saccadic glissades and overlapping saccades (Bahill & Stark, 1975). Finally, saccade limits are computed by searching from the first velocity peak backwards until eye velocity falls below some defined percentage (15% be default) of that first velocity peak to locate the start of the saccade. In the same way the function searches from the last velocity peak forward until eye velocity falls below some defined percentage of that last velocity peak (Fischer , Biscaldi, & Otto, 1993).

Parametrization of saccadic eye movements does include standard measures as listed in Table 2. Special attention has been devoted to measures of saccadic curvature as reviewed by Ludwig and Gilchrist (2002; see also Van der Stigchel & Theeuwes, 2006). These measures include initial curvature (Findlay & Harris, 1984, Van Gisbergen, Van Opstal, & Roebroek, 1987), initial average (Sheliga, Riggio, & Rizzolatti, 1995), maximum curvature (Smit & Van Gisbergen, 1990), as well as area curvature and second-order polynomial coefficients (Ludwig & Gilchrist, 2002). For some measures such as initial direction and direction at point of maximum curvature, angular deviations from the saccade targets rather than the saccade endpoint are automatically computed if stimulus positions are defined. Additionally, circular statistics such as mean vector length, mean angular deviation and straightness have also been included (Batschelet, 1981). Computation of saccade parameters can be accessed with `Analyze > Eye Events` from the MEB program menu.

Fixations. As a starting point for further analyses, intervals that are not scored as artifacts or saccades are identified as ocular fixations. The fixation detection algorithm then merges fixations if they are separated by artifacts or saccades that fall short of a critical duration and amplitude. A fixation interval thus may contain multiple microsaccades or blinks, depending on the program configuration. Parametrization of fixations includes basic measures as listed in Table 3. Computation of fixation parameters can be accessed with `Analyze > Eye Events` from the MEB program menu.

Pupillary Dilations. Since analyses of pupil size typically involve averaging pupil size traces of the same trial type rather than computing response parameters for single pupillary dilations (e.g. Reinhard, Lachnit, & Koenig, 2006, 2007) the EMA library currently does not include any routines for pupil size parametrization. After the pupil size trace has been blink-interpolated and possibly smoothed using the EMA library, computing signal averages of pupil size, however, is a straightforward standard procedure using Matlab's build-in functions.

Saving Your Data

When the analysis of a single recording session has been finished using the MEB program, the data set containing meta information, filtered signal data and computed response parameters is stored to hard disk using Matlab's native file format by choosing `File > Save` from MEB's program menu. Since statistical analyses of eye movements typically involve recording sessions from several subjects, EMA's export function is programmed to export multiple records at once. Exporting data includes specifying a number of data files in Matlab file format that then are converted to tab-delimited ASCII files. Multiple records are stacked into single tables that afterwards can be read by other statistical software packages such as SAS, SPSS, or R. EMA's export function will be described as part of the next section.

EMA Scripting Capabilities

MEB's graphical user interface calls functions in the EMA library that also can be evoked using the Matlab command prompt or as part of other Matlab functions or scripts. You might want to use these EMA scripting capabilities in order to batch process multiple eye records during import or export or if you want to expand EMA's standard response parametrization with the computation of custom response measures. EMA functions provide a consistent interface to the end user and all these functions operate on the same hierarchical data structure. This structure will be described in the following section.

Data Structures and Functions in the EMA Library

The `EMA_ED` class. The EMA library uses a custom Matlab data structure to store the different types of information contained in an eye movement record. A reference to a new, empty data structure is returned when calling the MEB program from the Matlab command prompt:

```
>> ED = MEB()

ED =
  EMA_ED handle

  Properties:
     SET: [1x1 EMA_SET]
     HDR: [1x1 EMA_HDR]
     SMP: []
     TRL: []
     SAC: []
     […]
```

This function call creates a data structure `ED` of class `EMA_ED` in the Matlab workspace which holds all the data that the MEB program will operate on. The properties (fields, substructures) of this data structure organize different aspects of the data set as listed in Table 4. When invoking the MEB program, the `EMA_ED` structure is empty except for some standard settings and header information stored within fields `SET` and `HDR`. Data fields will hold data as soon as a recording session is imported (`SMP`, `REC`, `TRL`, `MSG`) and response parameters have been computed (`ART`, `SAC`, `FIX`). At every time during the MEB session the underlying data structures can be accessed directly from the Matlab command prompt. For example as soon as saccades are computed, a table of saccade parameters can be accessed with

```
>> table_print(ED.SAC, 1:10)
        t1        t2  isac  itrl  dur   amp     dir     vel
  7464661  7464689     1     1   28  7.08  -90.44  377.12
```

```
7465122  7465136     2     1   14  0.50  -56.39   44.57
7465619  7465635     3     1   16  0.74  -78.98   74.67
7465638  7465651     4     1   13  0.58   96.57   58.78
7465983  7466005     5     1   22  0.53   93.05   37.29
7467454  7467488     6     1   34  6.30   91.19  258.71
7468533  7468567     7     1   34  6.12  -86.40  277.41
7468888  7468926     8     1   38  7.17   44.16  277.58
7469214  7469229     9     1   15  0.65   85.84   56.62
7474624  7474639    10     2   15  1.08  110.06  100.90
```

which will list the first ten saccades within the saccade table. The program manual provides a more detailed overview over the `EMA_ED` substructures and functions that operate on them.

Batch Processing Examples

Importing multiple records. If there are several eye records in an experiment you can use a simple MATLAB script to import, signal process, parametrize, and save multiple files without the need for manually going through all the processing steps using MEB's graphical menus. If the current working directory holds eye records of multiple subjects, these can be imported using the following script:

```
  FILE = dir('SBJ*.edf');
   % get list files to be imported

for i = 1:length(FILE)
   % cycle through files

     ED = EMA_ED();
       % init empty EMA_ED structure

     ED.SET.FILE.file = FILE(i).name;
     ED.SET.FILE.path = pwd();
       % set file name and path

     CONF_XYZ(ED);
       % run configuration script CONF_XYZ
       % see Figure 3 for an example
```

```
      EMA_import(ED);
        % import data
        % run signal conditioning as specified in CONF_XYZ
        % run response parametrization as specified in CONF_XYZ
      save([ED.INF.file, '.mat'], 'ED');
        % save processed record to file
  end
```

Depending on how many files are to be processed and which processing steps have to be computed for each file, batch processing of many files may take several minutes. By default, all functions in the EMA library do print verbose information about the processing status to the Matlab command prompt, so that one can always monitor processing progress.

Exporting multiple records. The library's export function is `EMA_export`. It is called to create ASCII files from the `EMA_ED` fields that can be used with statistical software such as SAS, SPSS, or R. Calling

```
>> EMA_export('SBJ*.mat', {'SAC', 'FIX'}, 'EXP_XYZ-');
```

from the Matlab command prompt will export tables holding saccade parameters (`SAC`) and fixations (`FIX`) for all data files “`SBJ*.mat`“ in the current working directory. Two tab-delimited ASCII files “`EXP_XYZ-SAC.dat`” and “`EXP_XYZ-FIX.dat`” will be created, that contain stacked saccade and fixation data of all data files in the current directory.

Conclusion

The current article presents the EMA library of Matlab functions designed to facilitate the post-experimental analysis of eye movement data. The library contains functions for importing records of different eye tracking systems, signal conditioning including artifact- and heuristic filters as well as parametrization artifacts, fixations and saccades. The library includes the Matlab Eye Browser (MEB) as a graphical user interface, that can be used to browse eye records on a trial-by-trial basis to inspect data sets and manually adjust responses. EMA's scripting capabilities provide the basis for automatized batch processing of multiple eye records. The program's primary use thus far has been the computation of measures of saccadic curvature and trajectory deviations. The library, however, is designed to be easily extendible to adopt to other fields of application.

In sum, we hope that our software's availability to other researches will help to standardize the use of saccadic curvature measures in the scientific literature. Two recent studies (Koenig & Lachnit 2010a, 2010b) have successfully demonstrated EMA's application to the analysis of saccadic curvature induced by (a) perceptual distractors, (b) spatial working memory and (c) associative learning.

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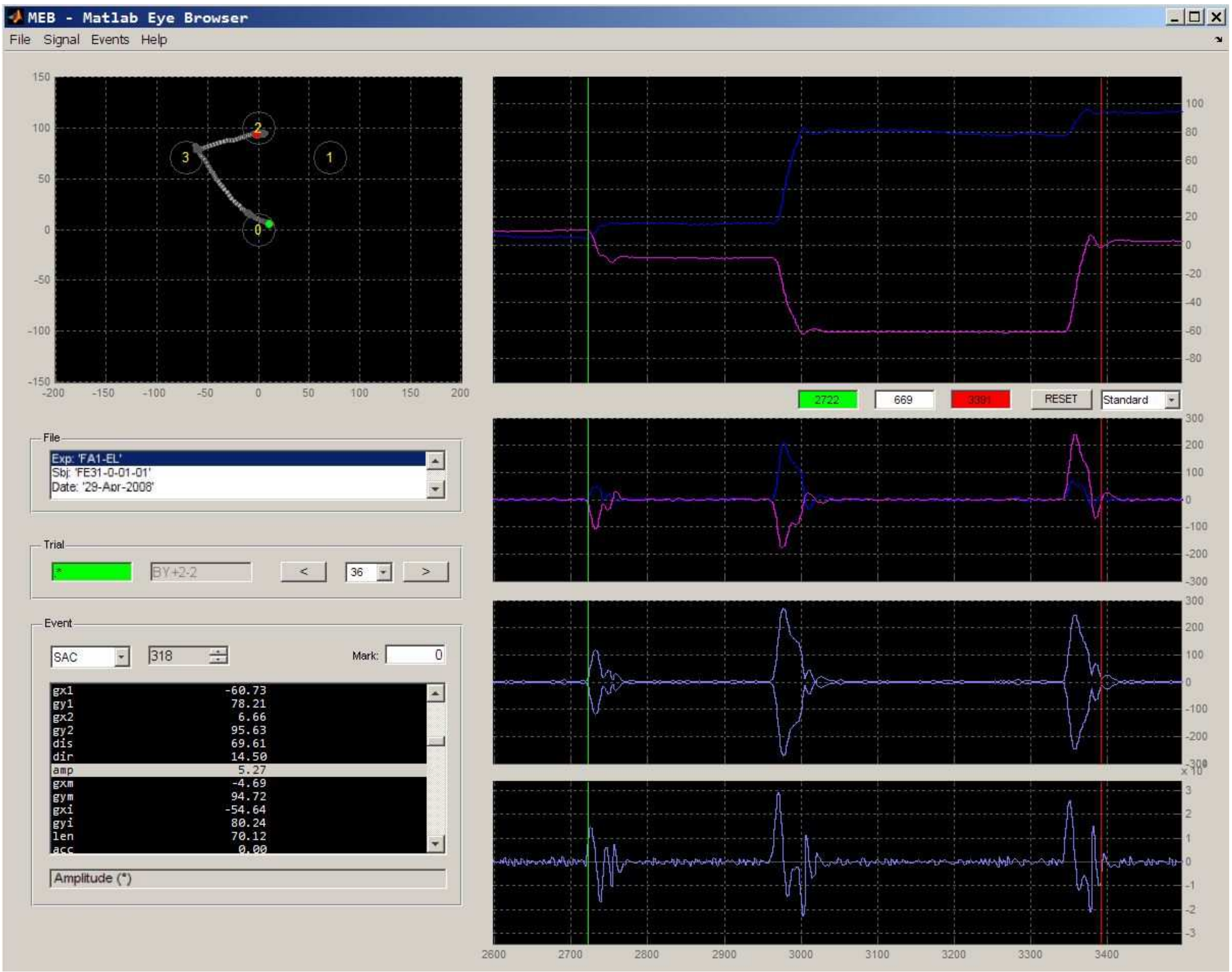
Figures



Exp: 'FA1-EL'
Sbj: 'FE31-0-01-01'
Date: '29-Apr-2008'

Trial: * | BY+2-2 | 36

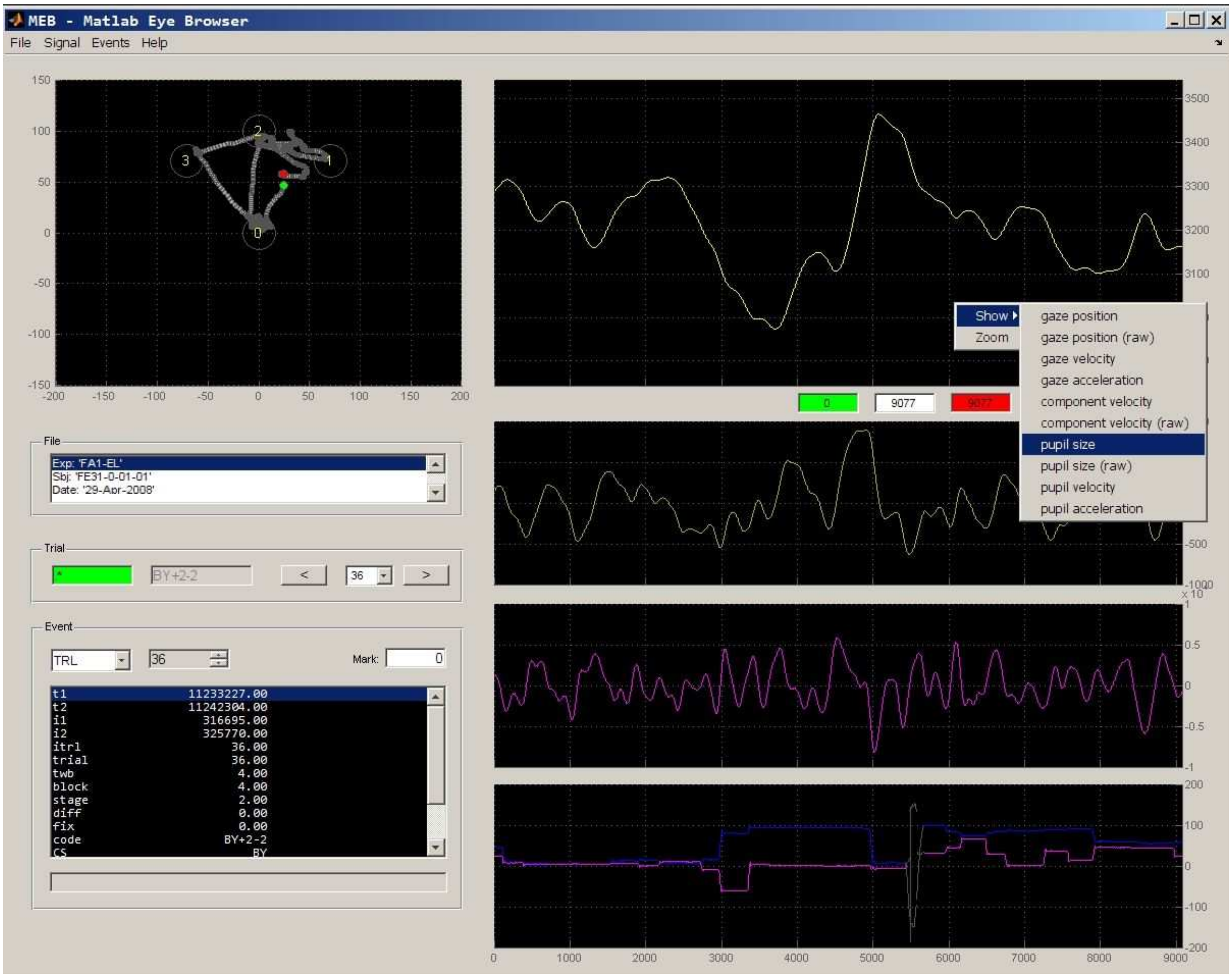
Event: SAC | 318 | Mark: 0

gx1	-60.73
gy1	78.21
gx2	6.66
gy2	95.63
dis	69.61
dir	14.50
amp	5.27
gxm	-4.69
gym	94.72
gxi	-54.64
gyi	80.24
len	70.12
acc	0.00

Amplitude (°)

2722 | 669 | 3391 | Standard

Figure 1. Main screen of the Matlab Eye Browser (MEB). Time plots on the right are selected to show gaze position, gaze velocity, component velocity and gaze acceleration respectively. Vertical start (green) and end (red) marks enclose a sequence of three saccades.



Exp: 'FA1-EL'
Sbj: 'FE31-0-01-01'
Date: '29-Apr-2008'

^ BY+2-2 36

TRL 36 Mark: 0

0 9077 9077

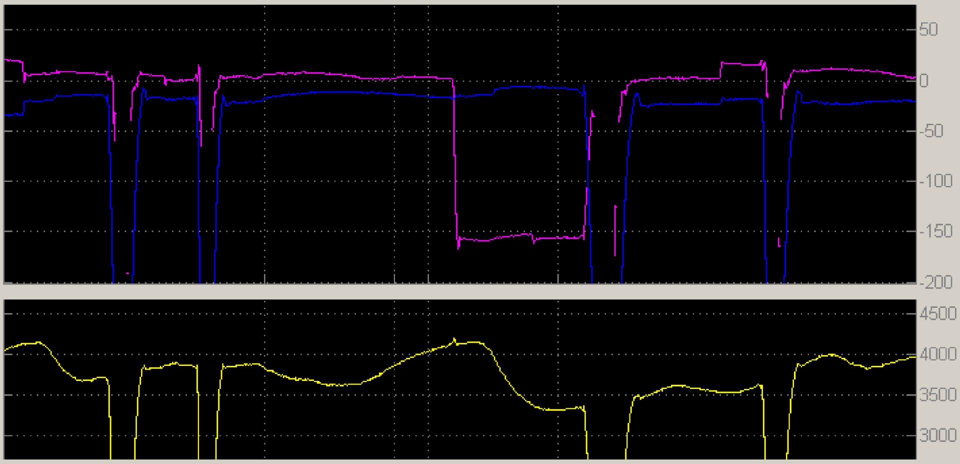
```
t1           11233227.00
t2           11242304.00
i1             316695.00
i2             325770.00
itrl               36.00
trial              36.00
twb                 4.00
block               4.00
stage               2.00
diff                0.00
fix                 0.00
code              BY+2-2
CS                    BY
```

Figure 2. Main screen of the Matlab Eye Browser (MEB) with time plots on the right configured to show pupil size, velocity and acceleration as well as gaze position (from top to bottom). The context menu for the first time plot is accessed by a right mouse click and demonstrates how to select the data source for a graph.

```
% Example of EMA Configuration Script

% --- Definition of Trial Structure
ED.SET.TTIME.event = {'START'; 'CUE'; 'TGT'; 'END'};
  % event labels
ED.SET.TTIME.msg   = {'START'; 'CUE'; 'TGT'; 'END'};
  % message string send during recording
ED.SET.TTIME.add   = [     0 ;    0 ;    0 ;    0 ];
  % numeric constant to add to message tim stamp

% --- Definition of Trial Variables
ED.SET.TVARS.vars = {'TRIAL_VAR_LABELS'};
  % names of trial variables
ED.SET.TVARS.msg  = {'!V TRIAL_VAR_DATA'};
  % start of message that records trial variables
ED.SET.TVARS.dlm  = {'\t'};
  % delimiter character for trial variables
ED.SET.TINFO.code = 'code';
  % name of trial variable that specifies trial code
ED.SET.TINFO.target = 'code';
  % name of trial variable that specifies trial target

% --- Configure Scaling of Gaze Position
ED.SET.GSCALE.status = 1;
  % rescale gaze position during import
ED.SET.GSCALE.xmin = -200; ED.SET.GSCALE.xmax =  200;
ED.SET.GSCALE.ymin = -150; ED.SET.GSCALE.ymax =  150;
  % scale gaze position to 400 x 300 area

% --- Configure Artifact Filter
ED.SET.FART.status = 1;
  % run artifact filter during import using default parameters

% --- Configure Saccade Detection
ED.SET.SACD.status = 1;
  % run saccade detection during import
ED.SET.SACD.minvel = 20;
  % minimum velocity for saccades; use default values otherwise
```

Figure 3. Example EMA configuration script.

(A) Unfiltered Raw Data



(B) Artefact Filter Applied

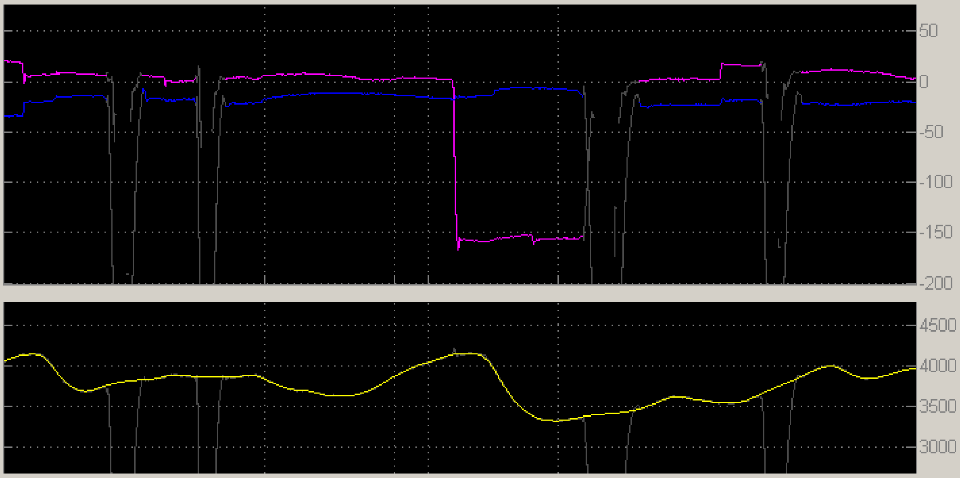
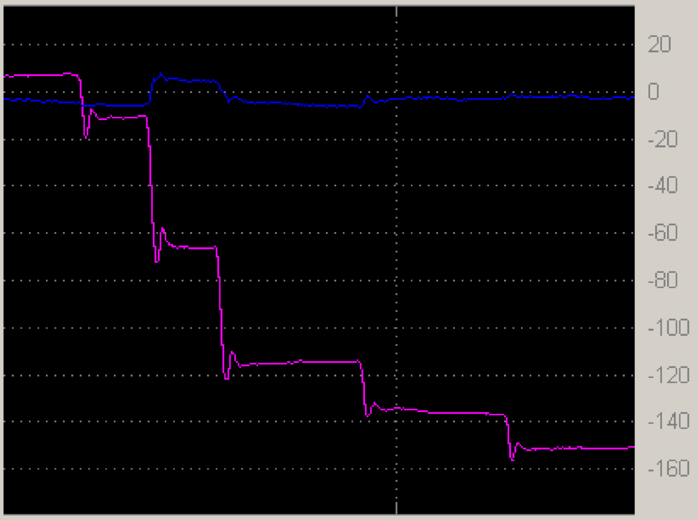


Figure 4. Applying the EMA artifact filter.

(A) Unfiltered Raw Data



(B) Heuristic Filter Applied

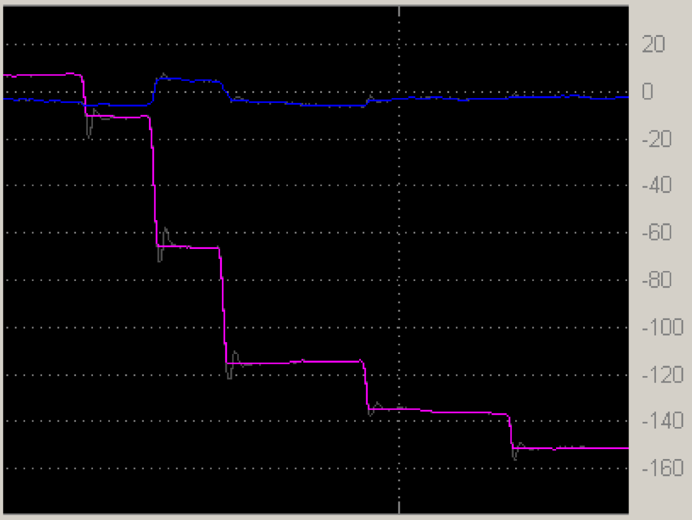


Figure 5. Applying the EMA heuristic filter.

Tables

Table 1. Artifact parameters.

Parameter	Description
`t1, t2`	Start and end time of artifact
`iart`	Number of artifact
`itrl`	Trial number of occurrence
`dur`	Duration of signal loss
`x1, x2`	Horizontal gaze position at start and end of artifact
`y1, y2`	Vertical gaze position at start and end of artifact
`dltxy`	Euclidean distance between start end end position
`dir`	Direction of gaze position change during artifact (°)
`amp`	Amplitude of gaze position change during artifact (°)

Note. Artifact parameters are stored within the Matlab data structure `ED.ART`.

Table 2. Saccade parameters.

Parameter	Description
`t1, t2`	Start and end time of saccade
`isac`	Number of saccade
`itrl`	Trial number of occurrence
`dur`	Duration of saccade
`lagon`	Time from onset of the previous saccade
`logoff`	Time from offset of the previous saccade
`vavg`	Average velocity
`vel`	Peak velocity
`vamp`	Peak velocity at `vamp` percent of saccade amplitude
`vtime`	Peak velocity at `vtime` percent of saccade duration
`acc`	Peak acceleration
`dec`	Peak deceleration
`x1, x2`	Horizontal gaze position at start and end of saccade
`y1, y2`	Vertical gaze position at start and end of saccade
`dis`	Euclidean distance between start end end position
`dir`	Direction of gaze position change [°]
`amp`	Amplitude of gaze position change [°]
`len`	Trajectory length
`cidir`	Initial direction of saccade (at defined percent of amplitude)
`cmx, cmy`	Gaze position at point of maximum curvature (deviation from straight line)
`cmdir`	Direction of saccade at point of maximum deviation
`cmamp`	Amplitude of deviation from straight line
`cmproj`	Maximum curvature at `cmproj` percent of saccade amplitude
`cmtime`	Maximum curvature at `cmtime` percent of saccade duration
`carea`	Area enclosed by straight line and trajectory
`poly2`	quadratic parameter of 2nd order polyniom
`mvl`	Mean vector length
`mad`	Mean angular deviation
`srt_XYZ`	Saccade latency relative to recording message XYZ

`eons, eoff`	position error (distance to defined stimuli) at onset and offset of saccade
`edir`	direction error

Note. The table shows a selected subset of saccade parameters. A more complete reference is given in the program manual. Saccade parameters are stored within the Matlab data structure `ED.SAC`.

Table 3. Fixation parameters.

Parameter	Description
`t1, t2`	Start and end time of fixation
`ifix`	Number of fixation
`itrl`	Trial number of occurrence
`mark`	Manual marker set via MEB
`adjust`	Event manually adjusted (0 = no, 1 = yes)
`x1, y1`	Horizontal and vertical gaze position at start of fixation
`x2, y2`	Horizontal and vertical gaze position at end of fixation
`x, y`	Mean horizontal / vertical gaze position during fixation
`dur`	Fixation dwell time
`lagon`	Time from onset of previous fixation
`logoff`	Time from offset of previous fixation
`mscnt`	Number of microsaccades within fixation
`artcnt`	Number of blinks within fixation
`left`	Type of preceding event (1 = saccade, 2 = artifact)
`right`	Type of following event (1 = saccade, 2 = artifact)
`ps`	average pupil size during fixation

Note. Fixation parameters are stored within the Matlab data structure `ED.FIX`.

Table 4. Fields of top level data structure.

Field	Description
`SET`	Program settings holding parameters for EMA function.
`HDR`	Header storing all meta information.
`SMP`	Sample data including eye position and pupil size as well as velocity and acceleration for all signals.
`MSG`	Messages sent to the eye tracker during the recording session.
`REC`	The recording session might be partitioned into different blocks of contiguous recording.
`TRL`	Trial variables sent to the eye tracker at the beginning of each trial.
`SAC`	Saccade parameters
`FIX`	Fixation parameters
`ART`	Artifact parameters

Note. The EMA_ED data type is the top level data structure hierarchically organizing sub structures that correspond to different aspect of the eye data record.

4 Empirical Study I: Revisiting the Memory-Based Curvature Effect

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Abstract

In three experiments we examined trajectory deviations of vertical saccades induced by the presentation of lateral distractor stimuli. In Experiment 1 the distractor was physically present during saccade preparation and execution. In Experiment 2 and 3 we investigated trajectory deviations caused by a lateral stimulus that was transiently presented about 1500 and 2000ms prior to saccade onset respectively. The memory representations of the lateral distractor stimuli caused saccades to deviate away from their prior location. In Experiment 2, participants were instructed to remember the exact location of the lateral stimulus and a trial ended with a test for the remembered location. In Experiment 3, the lateral stimulus position was irrelevant for the task, and there was no explicit instruction to remember its exact location. Saccades deviated away from the distractor stimulus regardless of task requirements. We interpret our results in favour of automatic stimulus encoding that leads to an interfering memory trace of the distractor. In contrast to prior experiments (Theeuwes, Van der Stigchel, & Olivers, 2006) results in Experiment 3 suggest, that curvature in the retention interval does not depend on the active rehearsal of the distractor's location.

KEYWORDS: Eye Movements; Saccade Trajectories; Interference; Inhibition

Introduction

During a saccadic eye movement the observer redirects the point of gaze towards a target stimulus in the visual periphery. The movement involves high rotational velocities that “catapult” the eyeball to its new position in a jerk-like, ballistic way. The trajectory of the eye movement, however, typically does not follow a straight line but shows some curvature instead. The present article replicates and extends recent findings of Theeuwes, Olivers, & Chizk (1995) that suggested that saccade deviations indicate the retention of positional information in spatial working memory. Before we turn to this memory-based effect, we will give a brief outline of saccadic curvature in general.

Distractor Induced Curvature

Curved saccade trajectories have been shown to be elicited by interference induced by peripheral stimuli competing for selection as the saccade target. In a series of experiments Sheliga, Riggio and Rizzolatti (1994, 1995) demonstrated that saccades deviated away from the point of covert visual attention. If, for example, a human participant is instructed to perform a vertical saccade to the upper hemifield while covertly attending to a location in the upper left quadrant, the trajectory of the saccade will deviate to the right, i. e. away from the attended location (Sheliga et al., 1995, Experiment 2). These results have been interpreted as evidence supporting the *premotor theory* of attention (Rizzolatti, Riggio, Dascola, & Umiltá, 1987; Rizzolatti, Riggio, & Sheliga, 1994). If the locus of covert spatial attention inflicts curvature upon saccade trajectories, the mechanisms of covert and overt attention must share some common physiological substrate. From this perspective the premotor theory assumes that shifts in covert attention essentially consist of programming a saccade to the attended location that is then withheld from being executed. The suppression of the saccade towards the attended location is believed to inflict curvature upon the saccade towards the target.

Doyle and Walker (2001) extended these findings to the case of task irrelevant distractor stimuli that might exogenously attract attention but that don't require any endogenous attention shifts. For example, participants performed vertical saccades whose direction were specified by an up or down-facing central arrow. Together with the central arrow, a lateral distractor stimulus was presented in one quadrant. The distractor carried no task relevant information and participants were instructed to ignore the stimulus. Nonetheless, saccades deviated away from the side of distractor presentation. Deliberate shifts in covert attention thus do not seem to be an essential requirement to evoke saccadic curvature. Rather, it seems that any peripheral stimulus that conflicts with the saccade goal may cause trajectory deviations. An increasing body of eye movement studies have explored further details of this distractor induced curvature effect (for reviews see Van der Stigchel, Meeter and Theeuwes, 2006; Van der Stigchel, 2010).

Selection From Population Codes

Saccade trajectory deviations away from a distractor stimulus have been explained in terms of an interaction between competing motor programs. Selection of a saccade towards the target stimulus is assumed to involve the inhibitory deselection of a saccade towards the competing distractor stimulus (Sheliga et al., 1994, 1995; Tipper, Howard, & Houghton, 2000). Tipper et al. (2000) also suggested that the motor program for a saccade is represented by the activity of populations of neurons coding for the target location in topographically organized motor maps such as the superior colliculus. Within the motor map, presentation of a visual target stimulus evokes a bell shaped activation gradient centered at the position of the target. Peak activity is evoked in cells coding for the direction and amplitude of the saccade to the stimulus, and activity gradually falls off with increasing distance from the locus of peak activity (Sparks & Hartwich-Young, 1989). When two targets are presented side by side their population codes might overlap and summation may lead to a new activity peak at an intermediate location. This interaction of excitatory gradients leads to the so called *global effect* (Ottes, Gisbergen, & Eggermont, 1984) where the averaging saccade lands at the "center of gravity" in between stimuli.

Saccade deviations away from a competing distractor stimulus are observed whenever the task explicitly defines one stimulus as the target and the other as a distractor stimulus that must not attract gaze as in the experiments by Sheliga et al. (1994, 1995) and Doyle and Walker (2001) outlined above (for review see Van der Stigchel et al., 2006). In this situation, top-down inhibitory control process are thought to actively deselect the distractor by inhibiting activity at the distractor site. With overlapping population codes, inhibiting the distractor inflicts inhibition upon some neurons coding for the target location as well, and as a result the locus of peak activity elicited by the target is shifted away from the distractor location (Tipper et al., 2000). The shift in peak activity causes initial deviation of the saccade away from the distractor location while curvature back towards the target at the end of the saccade is assumed to be caused by online control processes possibly involving the cerebellum (Quaia, Lefevre, & Optican, 1999).

The inhibition account of trajectory deviation is supported by reports on the functional relation between trajectory deviation and the latency of the response (McSorley, Haggard, & Walker, 2006, 2009; Walker, McSorley, & Haggard, 2006). If the target and the distractor appear together at unpredictable locations, and thus any selection process cannot start prior to their simultaneous onsets, short latency saccades directed at the target curve towards the distractor location while long latency saccades curve away. Deviation towards the distractor in short latency saccades is attributed to residual excitation at the distractor site that competes with the saccade towards to target when the target selection process has not been completely resolved prior to saccade execution. In contrast, deviation away from the distractor indicates the successful inhibitory deselection of the distractor site. This view is supported by micro-electrode recordings from the superior colliculus and the frontal eye fields in the monkey (McPeek, Han, & Keller, 2003; McPeek, 2006). These experiments revealed that for deviation towards a lateral distractor neural activity at the distractor site is elevated above baseline while activity is suppressed in saccades that deviate away. In sum the inhibition account currently provides the most vivid explanation of saccade deviations (for an alternative explanation see Sheliga et al., 1994). The time course seem to follow a rapid increase in inhibition applied to the distractor site after the distractor evoked some excitatory activity initially.

Spatial Memory and Saccade Trajectories

Theeuwes, Olivers, and Chizk (2005; Theeuwes, Van der Stigchel, & Olivers, 2006) demonstrated that trajectory deviations are not only elicited by competing *visual* stimuli, but also by *remembered* stimulus locations. Their participants performed vertical saccades to the upper or lower hemifield that were instructed by the direction of a central arrow. The arrow was preceded by a 500ms lateral dot (distractor stimulus) and dot and arrow were separated by a 1000ms blank interval. By the time of saccade execution, the saccade thus could only be influenced by a memory of the dot's location. The authors found that the trajectories of vertical saccades deviated away from where the dot had been presented. Much like Rizzolatti et al.'s (1994) premotor theory proposed a direct link between attention and the oculomor system, Theeuwes et al. (2005, 2006) argued in favor of a direct link between spatial working memory and oculomotor control.

In the experiments of Theeuwes et al. (2005, 2006), a trial ended with a memory test for the distractor's location. The task thus required participants to explicitly retain a memory of the distractor up to, during and after the saccade and possibly involved deliberate processes such as endogenous shifts of covert spatial attention and active rehearsal of the distractors's location in the retention interval. Correspondingly, Theeuwes et al. suggested that saccade deviations were triggered by the following processes: Presentation of the lateral distractor initially elicited exogenous activation at the distractors site. With a rather rapid decay of that exogenous activation, the representation of the distractor's location was transformed into sustained endogenous activation when participants engaged in active retention. In the retention interval, programming of the vertical saccade elicited inhibition of the distractor's sustained activation because it conflicted with the saccade to the target stimulus. In support of this view, Theeuwes et al. (2006) subsumed in their discussion that “there was basically no effect […] on saccade deviation” (p. 612) in a control condition in which the memory test was omitted and participants were not instructed to remember the distractor but rather to ignore it. The effect was proposed to be absent because without the need for active maintenance, the short-lived exogenous excitation caused by the distractor would rapidly decay and thus elicit no inhibition when the saccade was programmed. However, careful inspection of the result section in the same article reveals that even though the deviation effect was stronger in the memory condition, “the deviation in the no-memory condition was significantly different from zero” (p. 609). In contrast to their interpretation, these authors thus observed that saccades - although somewhat less pronounced - deviated away from a location that did not contain any physical stimulus and that also did not require any retention. This result is hard to explain from the perspective of deliberate memory processes and rather seems to require a more “automatic” interpretation.

The experiments reported in the present paper will revisit the memory-based curvature effect and we will suggest an alternative interpretation based on automatic stimulus encoding and sustained suppression rather than activation. Experiment 1 was conducted as a reference condition, in which curvature was evoked by a perceptual distractor presented together with the saccade target. Experiment 2 in part replicated the design of the original study by Theeuwes et al. (2005) including a memory test for the distractor location at the end of each trial. Experiment 3 revealed that deviation away from a lateral location can be observed without the requirement to encode or retain a memory for that location with an interval of up to 2000ms between distractor offset and the start of the saccade. We suggest that sustained *inhibition* of the distractor location triggered by distractor onset caused the observed deviation effects rather than sustained excitation that becomes inhibited when the saccade is performed.

General Method

Participants

Participants in all experiments were undergraduate students at the University of Marburg and received either course credit or payment. Participants were informed that the eye movement measurement would require them to sit still for about 30min as well as to avoid frequent blinking. All participants had normal or corrected-to-normal vision.

Apparatus

Testing took place in a sound-attenuated, dimly lit room. Monocular eye movements were recorded using an infra-red video-based eye tracker (Eyelink 2000, SR-Research) that sampled position of pupil and corneal reflection at 1000Hz. Sampling sides (left/right eye) were counterbalanced across participants. The eye tracking column restrained the participants head via chin and forehead rests. It was table-mounted in front of a 22” CRT-monitor (Iiyama, Vision Master Pro514) for computer-controlled stimulus presentation, yielding an eye to screen distance of 78 cm. To prevent environmental distraction from the experimental chamber the screen was framed by a rectangular funnel-shaped aperture that opened up from screen size to a width of 80cm on the participants site. Its dull light gray inner surface was homogeneously illuminated by hidden LED panels yielding an optimal indirect illuminance of the participants eyes.

Stimuli

Visual stimuli were presented on a dark gray (25%) background. Participants performed saccades to two centrally aligned target boxes located 7.5° (9.3° in Experiment 2) above and below fixation. Saccades were elicited either endogenously by a central arrow pointing towards one of the boxes, or exogenously by a white annulus with 1.1° outer and a 0.5° inner radius presented inside one of the boxes.

Procedure

At arrival participants gave written consent to the requirements to sit still and to avoid blinking during sampling intervals, and agreed to the anonymous storage and analysis of their data. Written instructions were presented that exemplified the events and task demands that occurred within a trial. Twelve practice trials were run prior to the actual experiments to ensure that participants had understood the instructions. Demographic data were collected on a post-experimental questionnaire.

Before each experiment, the eye tracker was calibrated using a 13-point grid of calibration targets. If necessary, the calibration procedure was rerun until the subsequent validation procedure confirmed an average calibration error < 0.5°.

Data Analysis

We used the Eye Movement Analysis software package (Koenig & Lachnit, 2010a) for MATLAB (The MathWorks, Inc.) to calculate saccade parameters. Saccades were detected using a multiple-pass, velocity-based algorithm that first identified saccades which exceeded an eye velocity of 60°/s, merges overlapping saccades (Bahill & Stark, 1975) in a second step and set final limits of the response at 15% peak velocity. We used several measures to quantify the shape of saccade trajectories. The *final direction* of a trajectory was computed as the angle between the line joining gaze position at the start and end of the saccade and the line joining saccade start and target position. As a measure of the *initial direction* of the saccade we first computed instantaneous directions for each each single sample within the the first 5 to 25% of the trajectory and for each sample computed the angular difference between instantaneous and final saccade direction. The maximum of these angular differences defined the point of maximum initial deviation from a straight line. Initial direction then was computed as the angular difference between the line joining saccade start and the point of maximum initial deviation and the line joining saccade start and target position. *Curvature* was computed as the area (mm^2) enclosed by the saccade trajectory and a straight line joining start- and endpoint of the movement (Ludwig & Gilchrist, 2002). Figure 1 exemplifies how these response parameters were computed.

Multivariate analyses of variance (MANOVA) were used to analyze the repeated measures data. Degrees of freedom were corrected with the Huynh-Feldt method (Huynh & Feldt, 1976). Effect sizes are reported as generalized eta squared (Bakeman, 2005). Data on error frequencies were subjected to a square root transformation prior to statistical analysis.

Experiment 1: Perceptually Present Distractor

Experiment 1 was conducted as a reference condition for Experiment 2 and 3 and presented a perceptual distractor stimulus competing with the saccade target. Participants performed vertical saccades instructed by a central arrow pointing either upwards or downwards. In the hemifield specified by the arrow, we additionally presented a distractor stimulus in the left or right quadrant and examined how the trajectories of vertical saccades were influenced by the simultaneous distractor presentations. There was no need for participants to deliberately shift covert attention towards the distractor location and any effect of the distractor on saccade trajectories thus should be caused by an exogenous, automatic capture of attention. We investigated the time course of inhibition applied to the distractor site by comparing short and long latency saccades.

Method

Subjects. Twenty undergraduate students participated in the experiment. The data of one participant was excluded because of signal noise and poor calibration. Another three participants were excluded because they performed poorly on the task and made saccades to the distractor instead to the target in more than 50% of the trials. Of the remaining participant 13 were female and three were male. Their age ranged from 20 to 27, *M* = 22.56, *SD* = 1.97.

Procedure. Figure 2a depicts the trial design used in Experiment 1. Participants performed vertical saccades towards one of two target boxes in the upper or lower hemifield. The direction of the saccade was instructed by a central arrow that pointed either up- or downwards. A lateral distractor stimulus was presented together with the central arrow in one of the four quadrants in two thirds of all trials. Unlike the original study by Doyle and Walker (2001), we only presented congruent trials, in which the distractor was presented in the same hemifield (upper versus lower) as the saccade target. For example, an arrow pointing upwards could have been accompanied by either a distractor in the upper-right or upper-left quadrant or no distractor at all. Although the distractor provided information about the saccade target, this information was also provided by the central arrow and participants were instructed to ignore the distractor stimulus. A manual response task was included to motivate participants to perform as instructed. A white annulus was presented within the cued target box 750ms after cue (arrow) onset. At annulus onset a small dot was flashed inside the annulus for 60ms in 50% of the trials. Participants performed a simple go / nogo manual response depending on whether they detected the faint flash. Auditory feedback was provided by a high versus low pitch tone indicating a correct or erroneous response respectively. The flashing dot was detectable only if participants performed a saccade in response to the preceding central arrow and fixated the correct target box prior to annulus onset. Six different trial types resulted from the combination of factors target (up, down) and distractor (none, left, right). The experiment thus consisted of 144 trials with 24 replication per trial type. For half of the subjects, the distractor was separated from the target by a polar angle of 45° (Group *Sep45*, far distractor), for the rest of the subjects separation was 30° (Group *Sep30*, near distractor).

Data Exclusion. For the analysis of saccadic curvature we only used *artifact free* and *correct* trials. A trial was scored as an *artifact* if there was temporary signal loss during the cue interval (arrow) due to eye blinks or other occlusion of the pupil by lashes or lid that masked or could have masked an eye movement. A trial was scored as *correct* if the participant fixated centrally at the onset of the arrow (fixation error < 2.5°) and performed a correct single saccade towards the cued target box (direction error < 15° and amplitude > 4°). With these criteria, on average, we excluded 18.32% of the trials for each participant. Error rates were subjected to a 2 x 3 x 2 ANOVA with factors target (up, down), distractor (none, left, right) and group (*Sep30*, *Sep45*) which revealed main effects for target, $F(1, 14) = 4.842$, $p < .005$, $\eta_G^2 = .053$, as well as distractor, $F(2, 28) = 6.013$, $p < .013$, $\eta_G^2 = .086$, with no interactions. Exclusion rates for trials requiring upward saccades were 13.5%, 26.6% and 24.7% with the distractor absent, or presented to the left or right respectively. In the same order, error rates for saccades to the lower target were 9.1%, 17.3% and 13.1%. Error trials thus more frequently occurred if a distractor was present on either side and if saccades were directed to the upper hemifield.

Results and Discussion

Saccade trajectories. The results from the experiment are shown in Figures 3 and 4. Figure 3 shows average saccade trajectories. Saccades started at central fixation (horizontal and vertical position = 0mm) and were directed towards the target box located 100mm above or below fixation. Participants performed saccades in the distractor absent condition (gray line), with a distractor to the left of the target (dashed line) and with a distractor at the right (solid black line). The leftmost four panels depict saccades observed in Group *Sep30* (near distractor) the rightmost four panels show saccades in Group *Sep45* (far distractor). Within group, left panels show low latency saccades, right panels show long latency saccades. Upper panels depict saccades directed at the target above fixation, bottom panels refer to saccades directed to the lower target. In general, saccades deviated *away* from the distractor. A distractor presented to the left caused rightward deviation, a distractor presented to the right caused leftward deviation. An exception from this overall effect was observed for fast upward saccades in Group *Sep30* (upper left panel). Trajectories of these short latency saccades deviated *towards* the distractor. In contrast, slow upward saccades in the same group again deviated away. Inspection of the upper panels in Group *Sep45* suggest that a somewhat less pronounced increase in deviation away with latency also was present with a far distractor. In both groups an increase in deviation away with latency was absent in saccades directed at the lower target. Inspection of Figure 3 suggests that the observed effects were superimposed on an overall baseline deviation to the right. However, baseline curvature was a highly idiosyncratic characteristic of single subjects and never significantly differed from zero in any of the experiments reported here.

For the statistical analysis of trajectories, we derived response parameters of initial direction, area curvature and final direction as outlined in the methods section. For each dependent variable, the baseline condition (gray lines of Figure 3) was subtracted from both, the distractor right and distractor left conditions. Baseline corrected variables were subjected to a multivariate analysis of variance (MANOVA). The Analysis used the same factorial design as depicted in Figure 3 and included factors target (of saccade: upper versus lower hemifield), distractor (in left versus right visual field; with baseline condition subtracted from both), latency (of saccade: fast versus slow) and group (target-to-distractor separation: 30° in Group *Sep30* vs. 45° in Group *Sep45*). The latency factor was computed based on a median split (for each experimental condition within subject) of saccadic reaction time (relative to the onset of the central arrow that instructed the saccade).

The MANOVA revealed a main effect for distractor, $F(1, 12) = 20.957$, $p < .001$, indicating that trajectories in general had different shapes in the distractor left versus right condition. However, the main effect was modulated by interactions Distractor X Target, $F(1, 12) = 10.116$, $p < .001$, Distractor X Latency, $F(1, 12) = 8.556$, $p < .003$, and Distractor X Target X Latency, $F(1, 12) = 7.356$, $p < .005$. The three-way interaction confirmed the observation that a modulation of the distractor effect by latency occurred in the upper hemifield only. Modulation of the distractor effect by group was absent, however, there was a trend for the three way interaction Distractor X Latency X Group, $F(3, 12) = 2.549$, $p > .105$.

For further investigation, dependent variables were subjected to univariate analyses of variance (ANOVA) using the same factorial design as above. The upper row of Figure 4 shows cell means and standard errors for the *initial direction* of saccades. In general, a distractor presented to the left (dashed line) caused initial rightward deviation (positive values on y-axis) while distractors to the right (solid line) caused leftward deviation (negative values on y-axis), the main effect for distractor, however, failed to reach statistical significance, $F(1, 14) = 3.088$, $p > .101$, $\eta_G^2 = .096$. The Distractor X Latency interaction, however, was significant, $F(1, 14) = 23.079$, $p < .001$, $\eta_G^2 = .068$. As can be seen in the top row of Figure 4 this effect was caused by an overall increase in deviation away with saccadic latency. In both groups, this increase was more pronounced in the upper hemifield, as indicated by a Distractor X Latency X Target interaction, $F(1, 14) = 9.954$, $p < .007$, $\eta_G^2 = .029$. Also, this increase was stronger with less target-to-distractor separation in Group *Sep30*, as indicated by an significant interaction Distractor X Latency X Group, $F(1, 14) = 8.381$, $p < .011$, $\eta_G^2 = .025$. In fact, the initial direction of upward saccades in Group *Sep30* was *towards* the distractor stimulus while only long latency saccades deviated *away*. The four-way interaction Distractor X Latency X Target X Group fell short of statistical significance, $F(1, 14) = 3.568$, $p > .080$, $\eta_G^2 = .001$.

The *area curvature* measure yielded a similar pattern of results (mid row of Figure 4). A main effect of distractor, $F(1, 14) = 12.899$, $p < .003$, $\eta_G^2 = .383$, was modulated by an interaction Distractor X Latency X Group, $F(1, 14) = 5.535$, $p < .034$, $\eta_G^2 = .015$, indicating an increase in curvature away from the distractor with saccadic latency for Group *Sep35*, while such modulation was absent with greater target-to-distractor separation in Group *Sep45*. The two-way interaction Distractor X Target also reached statistical significance, $F(1, 14) = 8.963$, $p < .010$, $\eta_G^2 = .029$; differential curvature was more pronounced in the lower as compared to the upper hemifield. A Distractor X Target X Latency interaction fell short of statistical significance, $F(1, 14) = 3.463$, $p > .083$, $\eta_G^2 = .015$.

The *final direction* of saccades was modulated by the side of distractor presentation as well (bottom row of Figure 4). However, in contrast to the former measures, in general, landing positions were shifted *towards* the site of distractor presentation. The main effect for distractor, $F(1, 14) = 11.698$, $p < .004$, $\eta_G^2 = .252$, was modulated by interactions Distractor X Latency, $F(1, 14) = 15.614$, $p < .001$, $\eta_G^2 = .085$, and Distractor X Latency X Target, $F(1, 14) = 20.651$, $p < .001$, $\eta_G^2 = .048$. Deviation towards the distractor thus decreased with increasing latency. The three way interaction confirmed that this modulation was more pronounced in the upper hemifield.

Inspection of Figure 3 suggests that saccades to the upper hemifield, in general, were of smaller amplitude, $M = 6.39$, $SD = 0.74$, than downward saccades, $M = 7.11$, *SD* = 0.64. A 2 x 3 ANOVA with factors target (up down), and distractor (none, left, right) confirmed a main effect of target, $F(1, 14) = 12.113$, $p < .004$, $\eta_G^2 = .186$.

Saccade latency. The preceding analysis revealed that trajectory deviations were dependent on saccadic latency. However, in the analysis above, factor latency was nested within the other experimental conditions. The latency median split was computed for each experimental condition separately (within subject). In this section we analyze latency differences between experimental conditions. A 2 x 3 ANOVA with factors target (up down), and distractor (none, left, right) revealed main effects of target, $F(1, 14) = 7.611$, $p < .015$, $\eta_G^2 = .043$, and distractor, $F(1, 14) = 7.439$, $p < .009$, $\eta_G^2 = .054$, modulated by a Distractor X Target interaction, $F(1, 14) = 3.760$, $p < .035$, $\eta_G^2 = .011$. Upwards saccades had latencies of 342 (44.8), 305 (57.3) and 304ms (57.4) with the distractor absent, presented to the left, or presented to the right respectively (with standard deviations in parentheses). In the same order, mean latencies for downward saccades were 348 (49.6), 334 (63.2) and 333ms (56.5). We thus observed shorter latencies if the trial required a saccade to the upper hemifield and if a distractor was presented on either side (remember that the distractor was always presented in the same hemifield as specified by the central arrow). The interaction indicated that the decrease in latency for the distractor present condition was more pronounced in the upper hemifield than the lower hemifield.

In summary, Experiment 1 replicated the saccadic curvature effect induced by visual onset stimuli. In agreement with the distractor inhibition account we observed trajectory deviations away from the side of distractor presentation in a task that required participants to suppress a saccade to the onset distractor. With this task structure, compared to baseline curvature, a distractor presented to the left caused target-directed saccades to deviate to the right while a distractor to the right caused leftward deviation.

Trajectory deviations were modulated by the latency and direction of the saccade as well as the target-to-distractor separation. Figure 5 revisits this effect in some more detail using a quartile split for the latency factor. Inspection of the bottom row (collapsed across groups) again shows that the trajectories of downward saccades are not dependent on the time of saccade execution. The top rows representing upward saccades in groups *Sep30* and *Sep45* however reveal a shift from deviation towards the distractor in short latency saccades to deviation away for longer latency saccades. The effects observed for the upper hemifield comply with the reactive inhibition account by Tipper et al. (2000): Increases in curvature away from a distractor site may have been caused by the gradual buildup of inhibition of the distractor-related saccade (increase in curvature away) after the distractor evoked some activity initially (curvature towards the distractor for saccades with shortest latencies). Furthermore, the stronger modulation of curvature in group *Sep30* supports the population coding hypothesis of Tipper et al.: Distractor induced modulation can be expected to be stronger when there is more representational overlap between target- and distractor-related motor programs respectively.

Whereas the effects of saccadic latency on direction and amplitude of trajectory deviations has been previously documented (Walker, McSorley, & Haggard, 2006; McSorley, Haggard, & Walker, 2006, 2009), upper versus lower hemifield differences with respect to this modulation to our knowledge have not been reported in previous studies and we will turn to this topic again in the general discussion. In the following experiments we investigated how trajectory deviations are affected by the contents of spatial memory. The results of Experiment 1 will yield a reference condition in order to evaluate the memory effects in the concluding discussion.

Experiment 2: Memorized Past Distractor

In Experiment 1, trajectory deviations were caused by a lateral distractor stimulus that was physically present at the time of saccade programming and execution. Experiment 2 examined whether the *memory representation* of a distractor had a similar effect on saccade trajectories. As outlined in the introduction, experiments by Theeuwes et al. (2005, 2006) provided evidence in favor of a representational overlap between spatial working memory and the oculomotor system and these authors subsumed, that “remembering a location makes the eye curve away”. Experiment 2 sought to replicate this memory-based trajectory effect. Participants were instructed to attend to a cued spatial location and to retain the location until completing a memory test for the location at the end of the trial. We observed how saccade trajectories were affected by the remembered location in the retention interval.

Method

Subjects. Sixteen students participated in the experiment. The data of one participant was excluded because of signal noise and poor calibration. Of the remaining participants 10 were female and 5 were male. Their age ranged from 20 to 31, *M* = 22.83, *SD* = 3.52.

Procedure. Figure 2b depicts the trial design used in Experiment 2. Participants performed saccades towards one of two target boxes in the upper or lower hemifield. The direction of the saccade was instructed by a central arrow that pointed either up- or downwards (50% of all trials each). At 1500ms prior to the presentation of the arrow, a to be remembered dot was presented in either of the four quadrants, to the upper-right, lower-right, upper-left or lower-left (20% of all trials each) respectively. At the end of the trial a memory test was conducted in which participants had to perform a manual response as to whether a test stimulus was presented at the same exact location as the remembered dot or at a slightly shifted location. In the remaining 20% of the trials the dot stimulus and the memory test were omitted. The combination of factors *saccade target* (up, down) and *distractor* (upper-left, lower-left, upper-right, lower-right, none) resulted in 10 different trial types that were presented in random order within each of 16 subsequent blocks, yielding a total of 160 trials. Figure 2b exemplifies the progression of stimuli within a trial. A 1000ms delay period was inserted between distractor (dot) offset and cue (arrow) onset so that the dot's influence on saccade trajectories would be mediated by spatial memory only.

Data Exclusion. For the analysis of saccadic curvature we only used artifact free, correct trials. A trial was scored as correct if the participant did not break fixation during the presentation of the dot (no saccade > 2°), still fixated centrally at the onset of the arrow (fixation error < 2.5°), and performed a correct saccade (direction error < 15°, amplitude > 4°) towards the cued target box. With these criteria, on average, we excluded 20.04% of the trials for each participant. Square root transformed error rates within experimental conditions were subjected to a 2 x 2 ANOVA with factors target (up, down) and distractor (none, any). A main effect for distractor fell short of statistical significance, $F(1, 14) = 3.693$, $p > .075$, $\eta_G^2 = .019$. Average error rates were 17.3 and 20.7% with the distractor absent and present respectively. Within the distractor present condition a 2 x 2 x 2 ANOVA with factors target (up, down), congruence (same, opposite) and distractor (left, right) revealed no significant effects.

Results and Discussion

Saccade trajectories. The results from the experiment are shown in Figures 6 and 7. Note that the x-axis of Figure 6 shows half the range of Figure 3 and in comparison magnifies the observed effect (the same is true for comparing the y-axes of Figure 4 and 7). Figure 6 shows average trajectories of saccades directed at the target whilst participants retained memory of the dot's location in order to complete the memory test at the end of the trial. Trajectories again represent saccades in the no distractor (gray), left distractor (dashed) and right distractor (solid) condition. The leftmost four panel depict saccades in the congruent condition with saccade target and distractor in the same hemifield. The rightmost four panels depict saccades directed to the opposite hemifield than the previously presented distractor. Within congruence conditions (same, opposite) different panels refer to factor combinations of target (up, down) and latency (fast, slow) as in Figure 3. In general, saccades deviated away from the remembered location. In comparison to Experiment 1 a modulation of curvature by latency was absent. The clearest effect was observed in downward saccades that were preceded by a distractor in the lower hemifield (bottom left panels). In three out of four slow saccade conditions, the trajectories observed without previous dot presentation (gray line, representing the no distractor baseline) did not lie in between the distractor left versus right conditions. This result was not anticipated, however, it did not affect the distractor effect in general. With the exception of slow downward saccades preceded by a distractor in the upper hemifield (bottom right panel) saccade trajectories still differed comparing the left versus right distractor condition.

A 2 x 2 x 2 x 2 MANOVA with factors target (of saccade: up, down), congruence (of distractor and target hemifield: same, opposite), distractor (left, right) and latency (slow, fast), revealed a main effect for distractor, $F(3, 12) = 4.016$, $p < .034$, that was modulated by an interaction Distractor X Target, $F(3, 12) = 6.666$, $p < .007$, confirming that distractor left versus right differences were greater in the lower hemifield. A trend for interactions Distractor X Congruence, $F(3, 12) = 2.962$, $p > .075$, and Distractor X Congruency X Latency, $F(3, 12) = 2.723$, $p > .091$, suggested that this hemifield effect was slightly more pronounced in the congruent condition.

For further analysis, we conducted univariate ANOVAs using the same factorial design as above. Cell means and standard errors for each variable are shown in Figure 7. The top row of Figure 7 depicts the saccades' *initial direction.* Trajectories deviated away from the distractor location, $F(1, 14) = 10.735$, $p < .006$, $\eta_G^2 = .009$. A distractor to the left of the target (dashed line) caused rightward curvature (positive values) while a distractor to the left caused rightward curvature (negative values). The main effect was modulated by a Distractor X Congruence Interaction, $F(1, 14) = 9.347$, $p < .009$, $\eta_G^2 = .015$. Comparing left versus right panels in the top row of Figure 7 indicates that deviation away from the distractor was more pronounced if the distractor was presented in the same hemifield as the saccade target.

The same effects, in general, were confirmed in the analysis of *area curvature.* Again, the distractor effect, $F(1, 14) = 12.148$, $p < .004$, $\eta_G^2 = .064$, was modulated by the Distractor X Congruence interaction, $F(1, 14) = 5.071$, $p < .041$, $\eta_G^2 = .010$, indicating a greater distractor effect in condition same (left panels). However post-hoc single comparisons revealed that there were no differences in trajectories to the upper hemifield at all (c. f. asterisks in Figure 7). The only significant effect with respect to the *final saccade direction* was a three way interaction Distract X Congruence X Latency, $F(1, 14) = 6.368$, $p < .024$, $\eta_G^2 = .008$, while the four way interaction fell just short of statistical significance, $F(1, 14) = 4.472$, $p > .053$, $\eta_G^2 = .005$.

Again, saccades to the lower hemifield were of slightly larger *amplitude*, $M = 8.600$, $SD = 0.683$, than upward saccades, $M = 7.815$, $SD = 1.052$, $F(1, 14) = 4.995$, $p < .042$, $\eta_G^2 = 0.172$.

Latency. Presentation of the distractor stimulus decreased the latency of the subsequent saccade. Average latencies were 406 (74.9), 341 (60.5) and 345ms (53.5) for upwards saccades with the distractor absent, presented in the same, or presented in the opposite hemifield respectively (standard deviations in parentheses). In the same order average latencies in the lower hemifield were 452 (66.8), 361 (60.9) and 363ms (51.8). A 2 x 3 ANOVA confirmed main effects for target, $F(1, 14) = 4.851$, $p < .045$, $\eta_G^2 = 0.052$, and congruence, $F(1, 14) = 52.600$, $p < .001$, $\eta_G^2 = 0.267$, as well as a Target X Congruence interaction, $F(1, 14) = 3.460$, $p < .045$, $\eta_G^2 = 0.011$. Saccades thus were faster if they were preceded by a distractor stimulus in the same hemifield and if they were directed to the upper hemifield.

In summary, Experiment 2 replicated the findings of Theeuwes et al. (2005, 2006) that saccade trajectories are affected by the contents of spatial working memory. When participants performed saccades instructed by the central arrow, there was no lateral distractor stimulus that was actually present at the time of saccade execution. The arrow that instructed the saccade was preceded by a blank screen for 1000ms. Nonetheless, the distractor inflicted curvature upon saccade trajectories which deviated away from the side of prior distractor presentation. A distractor presented to the left caused saccades to curve to the right, a distractor presented to the right caused saccades to curve to the left. As reported in the original study by Theeuwes et al., and again in accord with the population coding hypothesis (Tipper et al., 2000), this differential curvature effect was more pronounced when distractor and saccade target were presented in the same hemifield. Supposedly, motor programs of saccades to the same hemifield feature more representational overlap, and the distractor effect therefore is expected to be stronger.

Although, in general, Experiment 2 replicated the prior evidence for an interference between spatial memory and oculomotor programs, the exact nature of the memory representation causing saccadic curvature remained unclear. On the one hand, it is possible that trajectory deviations were caused by the active rehearsal of the distractor location. Sustained endogenous activity then conflicted with the saccade goal in the retention interval and became inhibited right before the saccade was executed (as suggested by Theeuwes et al., 2006). On the other hand, the effect may have been caused by inhibition applied to the distractor location right after distractor onset. In fact, in Experiment 2 (and in the Theeuwes et al. experiments) participants were instructed not to look at the distractor when it was presented and participants also knew for sure that the distractor location *never* was the target of their next saccade. With this task structure, inhibition of the distractor site could begin to build up right after distractor onset. If this inhibition was sustained until the time of saccade execution (possibly with some passive decay in the retention interval), saccades also would deviate away from the distractor location. This alternative account complies with the observation that in comparison to Experiment 1 any modulation of trajectory deviations by saccade latency were absent. If inhibition was applied to the distractor right after distractor onset (starting from 1853ms before the saccade on average) residual excitatory activation at the distractor site and thus any deviation *towards* the distractor as observed in Experiment 1 was rather unlikely. To further evaluate if active retention and sustained endogenous activation yield a responsible explanation for the memory-based effect observed in Experiment 2, in the next experiment we omitted the memory-test and used a distractor stimulus whose lateral location was irrelevant.

Experiment 3: Irrelevant Past Distractor

We conducted Experiment 3 in order to investigate whether the memory-based curvature effect really relies on deliberate rehearsal processes as suggested by Theeuwes et al. (2005, 2006). In Experiment 3 we used a task that did not require participants to deliberately shift covert attention towards the distractor stimulus or to encode or to memorize its lateral position. There was no such instruction, nor was there any test on the distractor position at the end of the trial. However, the distractor *was* relevant with respect to its vertical position because it specified whether a subsequent saccade was to be directed to the upper or lower hemifield (see Figure 2c). In contrast to Experiment 2, participants knew right after distractor onset whether the subsequent saccade was directed upwards or downwards and movement preparation could have started while the distractor was still visible. On the one hand, we assumed that this design would enhance the immediate inhibition elicited by the onset distractor. On the other hand, there is no obvious reason why activity at the distractor site should have been actively sustained, when the distractor's lateral position was irrelevant and the distractor also was in conflict with the saccade goal. In contrast, there are good reasons believe (Deubel & Schneider, 1996), that any attention exogenously captured by the distractor should subsequently shift away from the distractor location and towards the cued target location. All in all, deliberate retention of the distractor location in this design seems unlikely and saccades thus should not be affected by sustained endogenous activity at the distractor site. In summary, Experiment 3 thus investigated how automatic encoding of the distractors' lateral location influenced saccade deviations. To push boundaries, we adjusted the stimulus timing, so that saccades started about 2000ms after distractor offset.

Method

Subjects. Ten female and six male undergraduate students participated in the experiment. Their age ranged from 20 to 38, *M* = 22.38, *SD* = 4.27. All subjects were included for statistical analysis.

Procedure. The trial design used in Experiment 3 is shown in Figure 2c. As in the previous two experiments in each trial participants performed a saccade towards one of two target boxes located above and below fixation. A trial started with the presentation of a central fixation cross. After 2000ms a lateral distractor stimulus was flashed for 150ms in the upper or lower hemifield but participants were instructed to keep their eyes on the central fixation stimulus. After another 1850ms the fixation cross disappeared, the target stimulus was presented inside the target box, and participants performed the saccade. In two thirds of the trials a single distractor was presented in one quadrant (up-right, up-left, down-right, down-left). The remaining trials simultaneously presented a distractor on each side in the upper or lower hemifield (up-left/right, down-left/right). Because saccade trajectories have been shown to be straight if two distractors are presented at mirrored locations at both the left and right hemifield (McSorley, Haggard, & Walker, 2004), these trials were used as a baseline condition for saccadic curvature. In any case the target always appeared in the same hemifield (upper vs. lower) as the preceding distractor(s). For example, if a distractor was presented in the upper-right quadrant, the target appeared in the box above fixation 1850ms after distractor offset. The combination of factors target position (up, down) and distractor (both, right, left) resulted in six different trial types that were presented with 24 replications each. The experiment thus presented a total of 144 trials.

Participants were instructed to use the information provided by the distractor to prepare for the execution of a fast saccade as soon as the target appeared. From 300 to 440ms after target onset a small dot was flashed inside the target annulus in 50% of the trials. Participants responded with a mouse click whenever they detected this faint flash and received auditory feedback in form of a high (correct) versus low (incorrect) pitch tone after target offset. The manual response task was included to ensure that the participants would attend to the distractor stimulus in order to prepare for a fast saccade.

Unlike the experiment by Theeuwes et al. (2005) and the replication study reported in Experiment 2, the *lateral* position of the distractor in Experiment 3 was completely irrelevant to the participants' task. Any deliberate encoding of either the exact distractor position within quadrant (Theeuwes et al., 2005) or even the actual side (left vs. right) of distractor presentation was dispensable and any effect of the distractor on saccade trajectories thus should be attributable to an automatic encoding of the distractor position that interferes with the saccade program some 2000ms later.

Data Exclusion. Only valid trials were included in the statistical analysis. A trial was scored as valid if the participant kept fixating centrally during distractor and gap intervals (no saccade with amplitude > 2°) and performed a single saccade that started within 500ms from target onset, had an amplitude of at least 4° and was directed at the correct target box with a direction error < 15°. With these criteria we excluded 10.9% of all trials. Error frequencies did not differ between experimental conditions (all *p* > .26).

Results and Discussion

Saccade trajectories. Figures 8 and 9 depict average trajectories and cell means of dependent variables. As in Experiment 1 and 2, both Figures show trajectory deviations observed under factor combinations of target (up, down) and latency (slow, fast). Saccades were performed after a 150ms distractor stimulus and a subsequent 1850ms blank screen. Figure 8 shows that memory for the distractor location influenced saccade trajectories which again deviated away from the side of previous distractor presentation.

Baseline corrected variables were subjected to a 2 X 2 X 2 MANOVA which revealed a main effect for distractor, $F(3, 13) = 8.956$, $p < .002$, indicating that trajectories had different shapes in the distractor left versus right condition. Univariate analyses confirmed a distractor main effect for *initial direction*, $F(1, 15) = 25.153$, $p < .001$, $\eta_G^2 = 0.141$ (top row of Figure 9), and *area curvature*, $F(1, 15) = 18.312$, $p < .001$, $\eta_G^2 = 0.130$, (middle row of Figure 9). Again, trajectories deviated away from side of previous distractor presentation. No other effects were significant.

Amplitude of saccades were subjected to a 2 x 3 ANOVA with factors target (up, down) and distractor (symmetrical, left, right). A main effect of target, $F(1, 15) = 9.221$, $p < .008$, $\eta_G^2 = 0.167$, was modulated by the Target X Distractor interaction, $F(2, 30) = 3.868$, $p < .032$, $\eta_G^2 = 0.08$. Amplitudes of upwards saccades were 7.82 (0.439), 7.161 (0.453) and 7.122 (0.437) in distractor conditions symmetrical, distractor left, and distractor right respectively (standard deviations in parentheses). In the same order cell means in the lower hemifield were 7.526 (0.325), 7.464 (0.369) and 7.419 (0.365). Saccades to the upper hemifield thus were of shorter amplitude than downward saccades. Also, the presence of two distractor stimuli increased amplitude in upward saccades, while there was no articulate distractor effect in the lower hemifield.

Latency. As in the preceding experiment, saccade latencies were shorter in saccades directed to the upper hemifield, $F(1, 15) = 22.331$, $p < .001$, $\eta_G^2 = 0.107$. The effect occurred irrespective of the distractor condition. Average latencies were 166

(18.32), 170 and (20.01) 170ms (18.71) for upwards saccades in distractor conditions symmetrical, left and right respectively (standard deviations in parentheses). In the same order average latencies in the lower hemifield were 181 (22.48), 184 (23.30) and 182ms (20.47).

In summary, Experiment 3 provided clear cut evidence that the memory for a lateral distractor influenced the trajectory of a vertical saccade more than two seconds after the actual distractor had disappeared. In accord with Experiment 1 and 2, we found that saccades deviated away from the distractor location. Furthermore, Experiment 3 revealed the automatic nature of the memory based curvature effect. As opposed to Experiment 2, there was no need for participants to actually encode or retain the exact position of the distractor. Nonetheless, the distractor affected trajectories of saccades that on average started 2026ms after distractor offset. These results are hard to explain by the active retention account offered by Theeuwes et al. (2005, 2006) but are in accord with the idea of sustained distractor inhibition triggered by distractor onset. In support of this view and in accord with Experiment 2, modulation of distractor induced curvature by saccade latency again was absent (in contrast to Experiment 1). If deviation away from the distractor is the result of an inhibition process that started over two seconds before the saccade, the rate of change in inhibition when the saccade is executed can be expected to be low and deviations thus cannot be expected to change with saccade latency.

Summary and Concluding Discussion

Summary of Empirical Findings

In three experiments participants performed vertical saccades to target boxes above and below fixation and we observed the effect of present (Experiment 1) or past

(Experiments 2 and 3) distractor stimuli in the left versus right visual field. In all experiments, saccade trajectories deviated away from the side of distractor presentation. In Experiment 1, the deviation was caused by distractor stimuli that were presented simultaneously with a central arrow instructing saccade direction. The observed modulation of saccade deviations by saccadic latency revealed the time course of distractor inhibition. In the upper hemifield, short latency saccades deviated *towards* the distractor location while saccades with longer latencies deviated *away*. We thus observed an increase in deviation away with latency. Experiment 2 revealed that saccades also deviated away from a *remembered* distractor location. In this experiment, the distractor stimulus had been transiently presented one second prior to saccade execution and participants were instructed to memorize the distractor position. Although Experiment 2 replicated the memory effect reported by Theeuwes et al. (2005, 2006) the results allowed for an alternative interpretation in which saccade deviations were not caused by the active retention of the distractor's location. We conducted Experiment 3 to shed some further light on this alternative explanation. Experiment 3 revealed that the memory-based curvature effect was not restricted to a situation that required a deliberate encoding and memorization of the distractor location. We observed that saccades deviated away from a past distractor that was presented more than 2000ms prior to saccade execution (relative to distractor offset) and whose lateral position was irrelevant to the task.

Spatial Working Memory and Eye Movements

The memory based curvature effect has been interpreted with reference to spatial working memory and rehearsal of the distractor location that causes the eyes to deviate away. The inclusion of an explicit memory test in the studies of Theeuwes et al. (2005,

2006) facilitated such interpretations. Trajectory deviations observed within an instructed retention interval may be caused by actively keeping the distractor position in memory. When the saccade is executed during retention, the sustained endogenous activity at the distractor site is assumed to conflict with the saccade goal and selective inhibition of this conflicting activity is proposed to cause curvature away from the distractor location (Theeuwes et al., 2006). Awh, Armstrong and Moore (2006) suggested the related idea that the sustain of endogenous activity during retention might be accomplished by shifting covert attention towards the retained location in order to keep the memory active. Accordingly, in a recent review about the interactions between working memory and eye movements Theeuwes, Belopolsky and Olivers (2009) proposed that the memory-based curvature effect is caused by such *covert rehearsal*.

Does active rehearsal really provide a consistent and reasonable explanation for the curvature effect observed in Experiment 2 and 3? Results from Experiment 2 seem to be in line with the rehearsal account. Participants were instructed to remember the distractor location and were tested for the remembered location at the end of the trial. Saccades performed during the retention interval might thus have been influenced by rehearsal processes. However, we would like to argue that such an explanation seems far less likely for trajectory deviations observed in Experiment 3. There was no requirement for participants to encode and remember either the exact position of the distractor within quadrant nor whether the distractor was actually presented in the left or right visual field. All participants had to do, was to extract information about the hemifield (up, down) and to prepare a saccade to an upcoming target stimulus in the specified direction. In this situation it is hard to explain why participants should engage in active rehearsal of the distractor location for several reasons. First, encoding of the distractor's lateral location was not required to process the task and, more importantly, also conflicted with the saccade to the target. Secondly when participants prepared for the impending saccade covert attention should have shifted *away* from the distractor location and towards the target location (Deubel and Schneider, 1995). Attention based rehearsal of the distractor location under these circumstances therefore seems highly unlikely. Nonetheless saccades indicated that the spatial position in fact *was* encoded and somehow sustained, because trajectories deviated away from the side of distractor presentation.

From our perspective, the conceptually most parsimonious explanation of past distractor effects evolves from the following lines of argument: The visual onset of the distractor stimulus excites parts of the oculomotor map coding for the distractor location. The requirement not to break fixation during (and some time after) the presentation of the distractor, as well as the participants' knowledge that the presented stimulus *never* is the saccade target elicits a secondary, reactive (Houghton & Tipper, 1994; Houghton, Tipper, Weaver, & Shore, 1996; Tipper et al., 2000) process that selectively inhibits the distractor location in order to suppress an incorrect response. Inhibition applied to the distractor location might continue to increase for some time after distractor offset, but finally will shift into a gradual decay towards baseline. Whenever a saccade is executed with residual inhibition of the distractor site, the saccade deviates away from the distractor location. The concept of any deliberate rehearsal of the distractor position simply is superfluous in order to explain the memory-based effect observed in experiments 2 and 3.

The preceding assumptions would predict that trajectory deviations are stronger for distractors that are physically present (Experiment 1) as compared to past distractors (Experiment 2 and 3) for which the decay of inhibition already has advanced.

Accordingly, we observed that the absolute amount of deviation in the three experiments decreased with the length of the distractor-to-target interval. Mean effect sizes (difference in initial direction left - right; η_G^2 in parentheses) were 14.72 (.373), 5.55 (.216) and 2.53 (.151) in Experiment 1, 2 and 3 respectively (computed for long latency saccades in all experiments and for the congruent condition of Experiment 2), and these effects corresponded to a simultaneous distractor, a 1500ms and a 2000ms distractor-to-target interval respectively.

Theeuwes et al. (2006) reported that memory-based deviations were more pronounced with the inclusion of a memory test at the end of a trial as compared to a control condition without such a test. Their interpretation was that activation elicited by the onset of the distractor would rapidly decay if not taken over by endogenously sustained activation that interferes with the subsequent saccade. In contrast, we suggest that this effect can also be explained by assuming that the initial activation the distractor elicited in the memory test condition was greater than in the control condition because participants simply paid more attention to the distractor stimulus when it was presented. The critical assumption to acknowledge is that a “strong” distractor stimulus might result in more suppression than a “weak” distractor stimulus. Stronger initial inhibition then results in more residual inhibition at the time of saccade execution and deviation, as observed, would be greater in the memory test condition. Again, an actively sustained memory of the distractor location is superfluous in order to explain the observed deviation effects. The Houghton and Tipper (1994) model of selective attention includes a similar mechanism in which the size of an inhibitory rebound effect increases with the amount of initial distractor activation (Houghton et al. 1996)[1]. Koenig and Lachnit (2010b) recently demonstrated that a mechanism that results in more inhibition for strong distractors yielded an accurate quantitative prediction of how trajectory deviations evolve during a spatial learning task that included the cued prediction of competing target locations. If two competing predictions of where a peripheral target might occur were retrieved from associative memory, saccades were directed to the most likely location and deviated away from the competing location (see Koenig and Lachnit, 2010b, for a discussion about the automatic nature of these predictive responses).

In all experiments reported in the present article, we compared curvature in short versus long latency saccades. Trajectory deviations were modulated by saccadic latency in Experiment 1 only. Such modulation was absent, when the distractors preceded saccade onset for 1500ms and 2000ms in Experiment 2 and 3 respectively. The observation is in line with the assumption that inhibition of the distractor sites quickly builds up following distractor onset and then fades into gradual and rather slow decay afterwards. Whereas saccades occurred with average latencies of 319ms after distractor onset in Experiment 1, latencies were 1852 and 2176ms in Experiment 2 and 3 respectively (referring to distractor onset). Clearly, the rate of change in distractor related activity during saccade preparation is expected to be small at long latencies and substantial change in curvature with saccadic latencies thus cannot be expected in Experiment 2 and 3.

Godijn and Theeuwes (2004; Experiment 1) reported that irrelevant distractors caused saccade deviations after 100ms and 400ms, but there were no deviations after an interval of 800ms. With reference to this finding, Theeuwes et al. (2006) argued that exogenous activity rapidly decays if the task does not require memorization of the distractor location and short-lived exogenous activation is taken over by endogenous sustained activation. How could the memory for the distractor location have lasted more than twice as long in our experiments *without* the notion of active rehearsal? When the lateral onset stimulus was presented in the experiments of Godijn and Theeuwes, participants did not know whether this stimulus was the target of the subsequent saccade or a distractor stimulus. The arrow that instructed saccade direction after 100, 400 or 800ms could point towards the lateral onset on some trials. With this design the distractor was likely to receive less inhibition in the first place because participants were unsure whether the saccade towards the stimulus had to be suppressed at all. In contrast, in our experiments participants knew for sure that the lateral onset stimulus always was a distractor, the stimulus thus received more inhibition in the first place, and the stronger initial inhibition was sustained for a longer period of time. Again, the inhibition process right after distractor onset is sufficient to explain the memory effect without the reference to rehearsal and active retention.

Upper Versus Lower Hemifield Differences

In the last section of the discussion, we will shortly turn towards the differences in trajectory deviations observed when comparing upward and downward saccades. To our knowledge Experiment 1 of the present study is the first experiment that has revealed such differences. In Experiment 1 we observed a transition of deviation towards the distractor for short latency saccades to deviation away from the distractor with long latency saccades. This effect of saccadic latency on direction and amplitude of deviation has been reported previously (McSorley et al., 2006, 2009; Walker et al., 2006). However these studies used up to eight different saccade directions and collapsed across direction prior to data analysis. In contrast, separate analysis of upward versus downward saccades in Experiment 1 revealed a three-way interaction indicating that the effect of latency on saccade trajectories was restricted to saccades to the upper hemifield only, while there was no change in curvature with latency for downward saccades. This effect, however, has to be interpreted carefully, since downward saccades were generally of shorter latency than upward saccades. It thus remains unclear whether the observed effect was due to hemifield differences or the fact that short latency saccades, featuring deviation towards the distractor, occurred in the upper hemifield only.

Hemifield differences in latency have been interpreted with respect to a functional differentiation for attention towards stimuli in the upper versus lower hemifield. Whereas the upper hemifield predominantly represents far, i.e. *extrapersonal* space, sensorimotor functions in the lower hemifield have evolved to represent near stimuli in *peripersonal* space (Previc, 1990). Stimuli in extrapersonal space may require fast orienting responses (approaching predator) whereas stimuli in peripersonal space might require rather precise orienting responses (eye hand coordination). Along this line of argument, the only saccades that exhibited a substantial postsaccadic error (shift of landing position towards the distractor) occurred in short-latency saccades directed to the upper hemifield in Experiment 1.

Summary and Conclusion

Our results demonstrate, that the memory trace elicited by a lateral distractor stimulus induced saccade trajectory deviations away from the side of prior distractor presentation. We observed saccade deviations with an interval of about two seconds from distractor offset to saccade onset even if participants did not engage in active rehearsal of the distractor location. In contrast, prior studies have claimed that oculomotor interference elicited by an irrelevant distractor decays within 400-800ms (Godijn and Theeuwes, 2004). There are no prior reports on the relation between saccade deviations and saccadic latency in memory-guided saccades. The present results reveal that, in contrast to simultaneous distractors, such modulation was absent for past distractors. Taken together, our results suggest that memory-based saccade deviations are better explained by sustained inhibition rather than rehearsal and sustained excitation. All in all, we propose that memory-based curvature effects are an automatic feature of stimulus processing rather than the consequence of deliberate memory processes.

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Footnotes

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[1] The Houghton & Tipper (1994) model originally was put forward to explain object based attention and the negative priming effect. While it does assume that distractors are inhibited following stimulus onset, it does not assume that there is any net inhibitory effect (i. e. suppression below baseline) during stimulus presentation. Such suppression below baseline is assumed to start with stimulus offset and the model in its original formulation thus cannot account for inhibition based curvature away with simultaneous distractor stimuli.

Figures

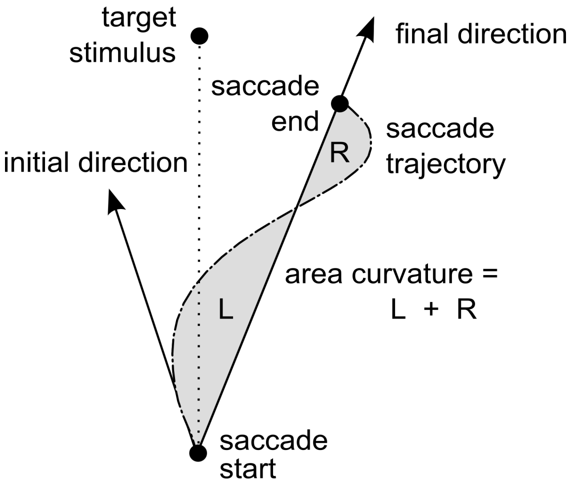

Figure 1. Response parameters derived from a saccade trajectory (dash-dotted line). The f*inal direction* is given by the vector joining start and end point of the saccade. The *initial direction* is given by the point of maximum angular deviation from a straight line within the first 25% of saccade amplitude. Both direction measures are calculated as the angular deviation from the line joining saccade start position and target stimulus position. Leftward and rightward deviations are given negative and positive signs respectively. *Area curvature* is measured by calculating the area enclosed by the trajectory and a straight line (gray shaded areas). Again rightward deviations (area R) have positive sign, leftward deviation (area L) have negative sign. Net area curvature then is computed by summation L + R.

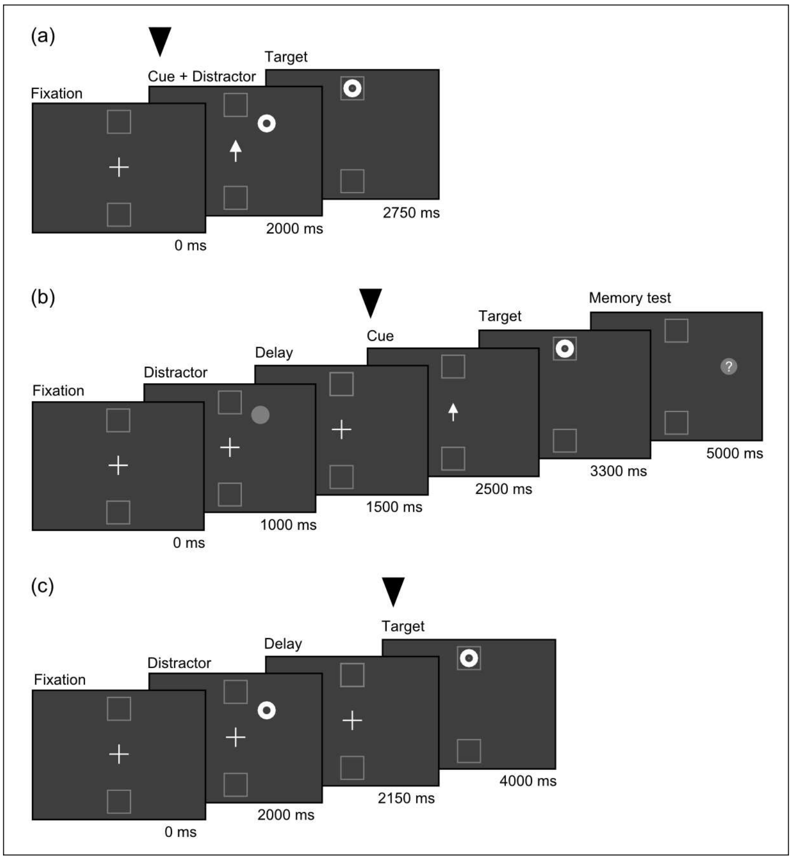

Figure 2. Trial layout of Experiments 1, 2 and 3. **(a)** Experiment 1 examined the effects of a perceptual distractor that is presented together with a central cue specifying the target location and is still present while the saccade is performed. **(b)** Experiment 2 is a replication of Theeuwes et al. (2005) demonstrating how remembering a distractor location influenced saccade trajectories. **(c)** Experiment 3 examined if the memory test at the end of the trial is a necessary condition for inducing saccadic curvature. The time of stimulus presentation is given beneath each frame. Black arrows indicate the time of saccade execution. For further explanations see text.

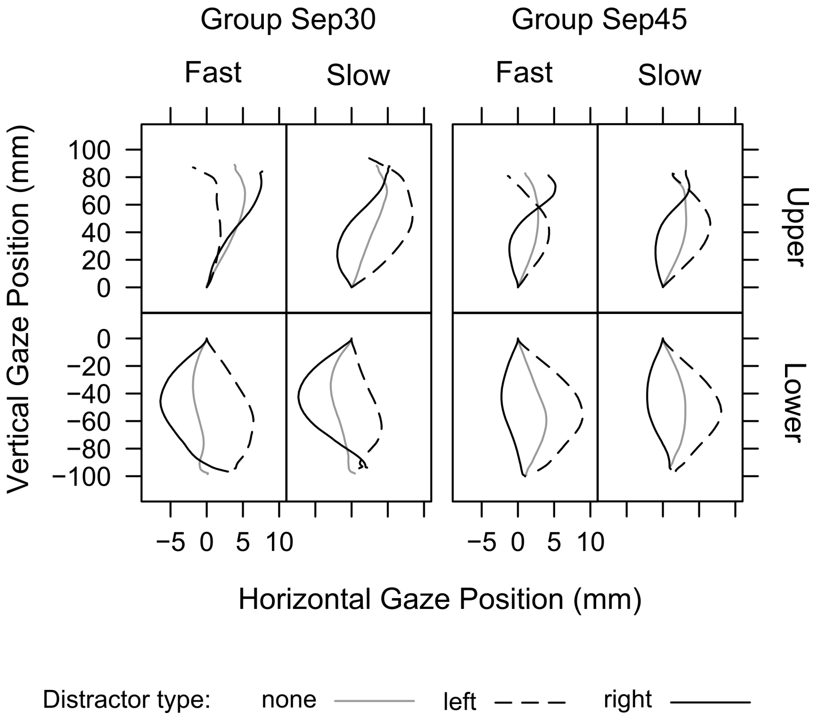

Figure 3. Average saccade trajectories observed in groups *Sep30* and *Sep45* of Experiment 1. Panels *Upper* and *Lower* depict saccades performed to the upper and lower hemifield respectively. Panels *Fast* and *Slow* show average trajectories of short versus long latency saccades (median split of latency within condition within subject). Dashed, solid gray, and solid black trajectories within panel correspond to saccades performed within the distractor left, distractor absent, and distractor right condition respectively.

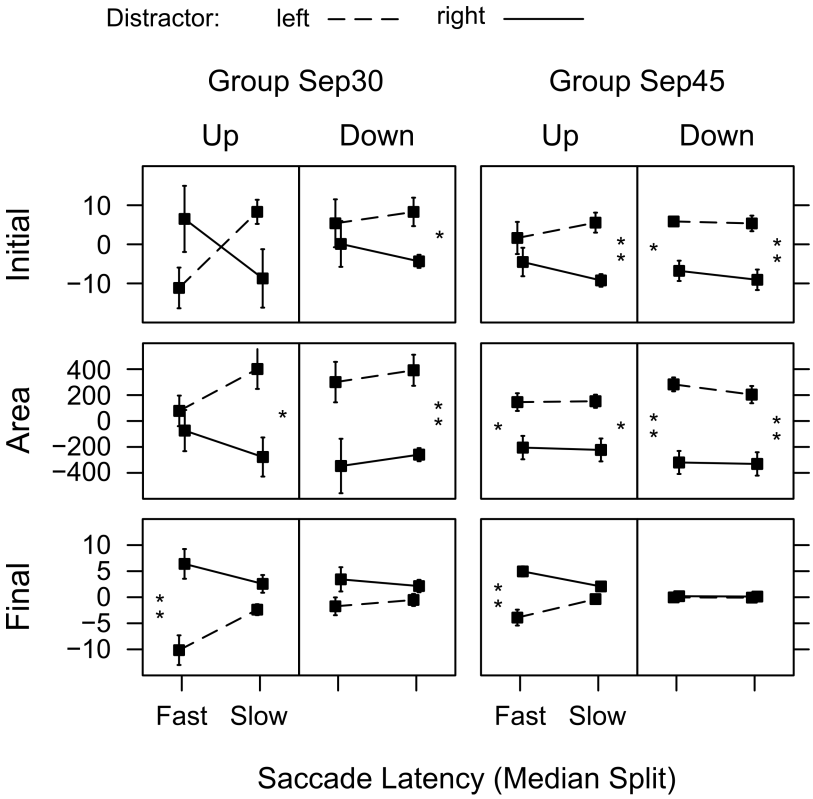

Figure 4. Marginal means of initial direction (upper row; angular deviation in °), area curvature (mid row; area in mm^2) and final direction (bottom row; angular deviation in °). Panels on the left show saccades in Group Sep30, panels on the right refer to Group Sep45. Within group, panels on the left represent upward saccades, right panels depict downward saccades. Each panel contrasts low latency (fast) and high latency (slow) saccades on the horizontal axis. Lines within panel correspond to conditions with the distractor presented in the left (dashed line) versus right (solid line) visual field. Inset asterisks indicate significant t-tests contrasting saccade deviations induced by a left versus right distractor (* p <. 05; ** p < .01; *** p < .001).

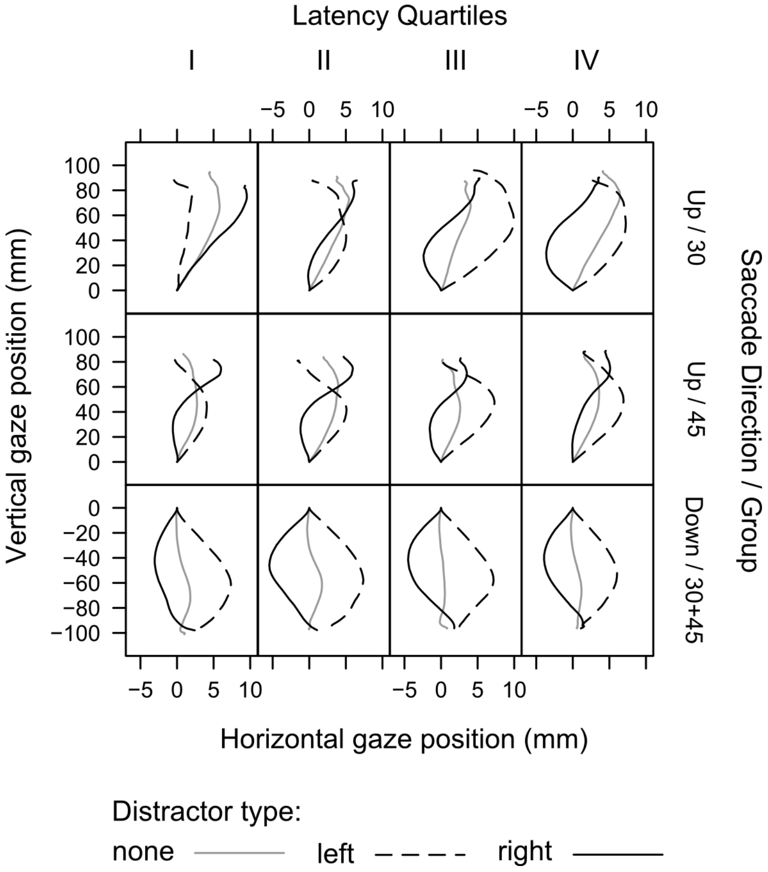

Figure 5. Influence of saccadic latency on trajectory deviations in Experiment 1. Downward saccades of different latencies show no differences in trajectory deviations (bottom row). For upward saccade (top rows), however, short latency responses tend to show less deviation away from the distractor than long latency saccades. In fact short latency saccades clearly deviate towards the target for short latencies especially with small target-to-distractor distance in Group *Sep30* (upper left panel).

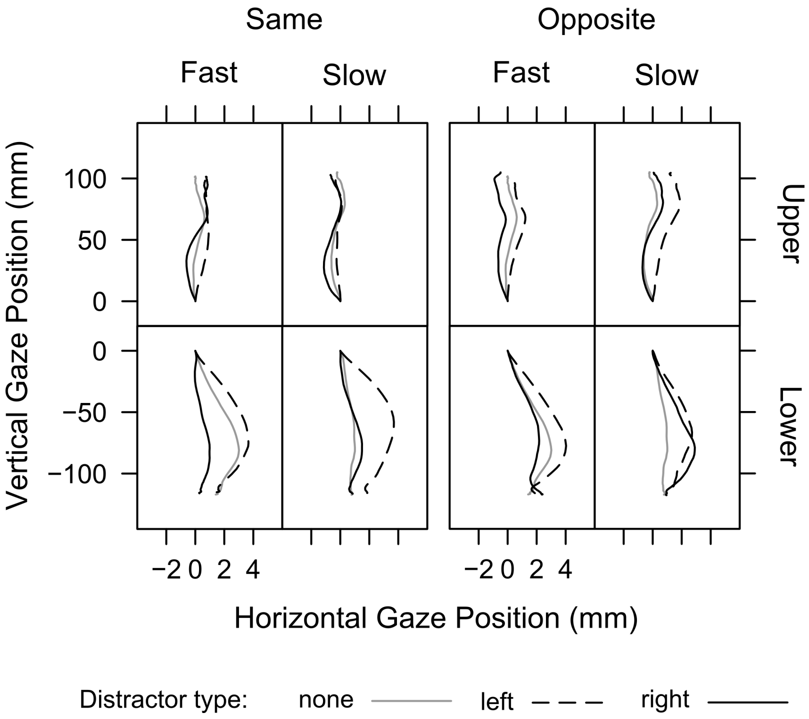

Figure 6. Average trajectories observed in Experiment 2. Panels *Same* depict saccades performed to the same hemifield as the distractor was presented in. Panels *Opposite* depict saccades that were preceded by a distractor in the opposite hemifield as the saccade target. Other conditions as in Figure 3.

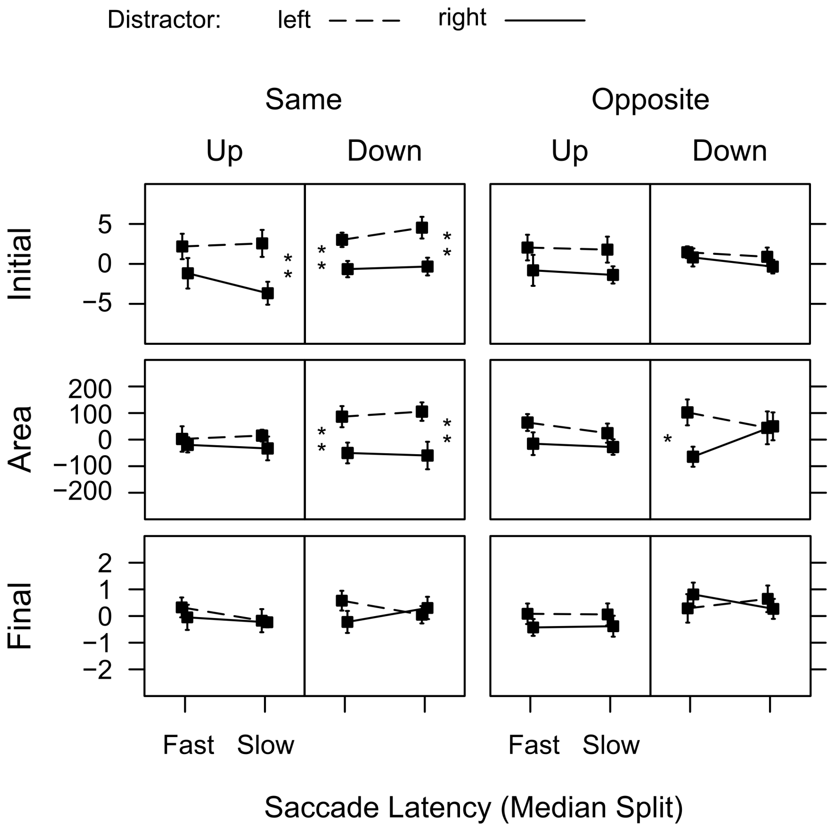

Figure 7. Marginal means of dependent variables in Experiment 2. Panels *Same* represent trials with saccade target and distractor in the same hemifield. Panels *Opposite* depict the reverse case. Other conventions as in Figure 4.

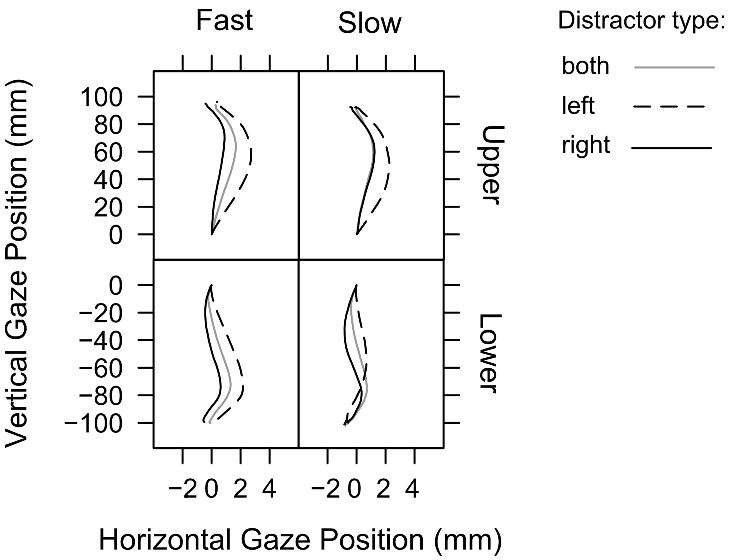

Figure 8. Average trajectories observed in Experiment 3.

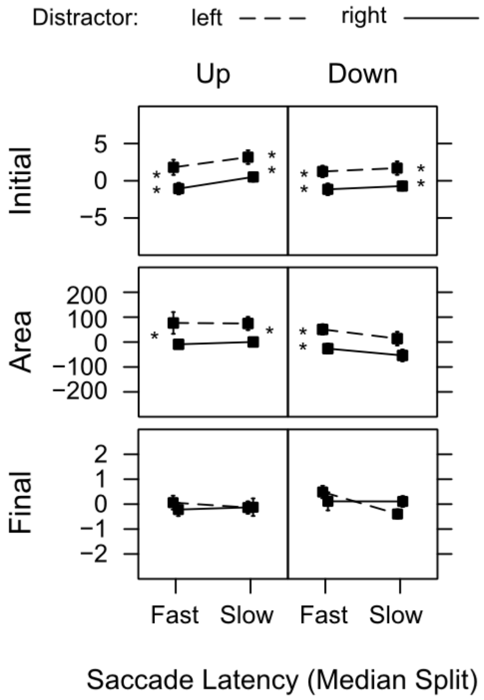

Figure 9. Marginal means of dependent variables in Experiment 3.

5 Empirical Study II: Curved Saccade Trajectories Reveal Conflicting Predictions in Associative Learning.

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Abstract

When a target stimulus in the visual periphery is presented together with a flanking distractor stimulus, the trajectory of the target-directed saccade typically does not follow a straight line but rather shows some curvature instead. Saccades may either curve towards or away from the distractor and direction and amplitude of curvature have been attributed to the excitation versus inhibition of neurons coding for the distractor location within topographically organized motor-maps.

In the present experiment we analyzed curvature in saccades guided by associative memory. Human participants learned to predict which of several central cues predicted which of several peripheral target locations. We measured frequency, latency and curvature of saccades elicited by the cues and directed at the trained locations in anticipation of the targets. We provide simulations of a connectionist network based on the Rescorla-Wagner model learning rule which predicts the acquisition of excitatory associations linking the cues to their trained targets and importantly also predicts how the cues will acquire excitatory or inhibitory associations to competing outcomes. We test different hypotheses on how these concurrent associations might inflict curvature upon saccade trajectories and derive a simple algorithm to yield a quantitative description of how saccadic curvature changes during training. The experimental paradigm may provide a valuable new method for accessing the set of concurrent associations that a cue acquires in the course of classification learning.

KEYWORDS: Associative Learning; Interference; Inhibition; Eye Movements, Saccade Trajectories

Introduction

Associative learning has typically been explored in conditioning experiments that require an organism to predict the presence versus absence of some biologically significant event. In Pavlovian conditioning for example an animal learns to predict the presence versus absence of the same unconditioned stimulus (US / outcome) based on the information provided by the preceding conditioned stimulus (CS / cue). However, many learning situations may require the prediction of multiple competing outcomes. Do the clouds in the sky predict rain or storm or sunshine? Does the facial expression of my opponent imply sympathy or fear or hate? If learning contributes to the acquisition of these classification skills one may ask the question of whether learning in these multiple-outcome situations complies to the same rules of association formation identified in single-outcome conditioning experiments. Particularly, a generalization of formal animal learning theory to the prediction of multiple-outcomes would imply that a cue may not only acquire excitatory associations to its trained outcome but *at the same time* also might acquire conflicting excitatory or inhibitory associations to competing outcomes. This assumption of concurrent weight changes lies at the very heart of the connectionist perspective on classification learning and we will further explore this view by simulations of an associative network based on the Rescorla-Wagner (1972) model learning rule as the “most widely accepted description of associative changes during classical conditioning” (Gluck & Bower, 1988).

In the present experiment we devise an oculomotor classification learning task that required participants to learn which of several arbitrary stimuli (cues) predicted which of several peripheral target locations (outcomes). We use cue-elicited saccadic eye movements that anticipate the correct target location to indicate the acquisition of cue-outcome associations. We explore, how conflicting predictions that the cues acquires during training, inflict curvature upon the trajectories of predictive saccades. Clearly, any adaptive system should try to minimize interference resulting from competing predictions and thus suppress any interfering response tendencies endangering the selection and generation of the “correct” response. In the next sections we will review two different mechanisms, 'distractor inhibition' and 'conditioned inhibition' that have been theoretically advanced in different fields of psychology in order to explain the suppression of such maladaptive, interfering behavior.

Distractor Inhibition

The notion of inhibitory control over cognition and behavior lies at the very heart of many psychological domains. Stimuli have to be ignored to selectively attend to a target stimulus (Tipper, 2001), distracting memories have to be suppressed to selectively retrieve the requested memory (Levy & Anderson, 2002) and competing responses have to be inhibited to generate a response appropriate for the current situation (Monsell, 2003). The common characteristics in these situations is that at least two stimuli, memories or responses compete for selection. One response system for which these mechanisms of selection have been extensively studied both behaviorally and physiologically is the eye movement system in humans and monkeys (Basso & Wurtz, 1998; Deubel & Schneider, 1996; Findlay, 1997; Schall & Thompson, 1999; Schall, 2001). Because we can move our eyes to only one location at a time, stimuli in the visual periphery compete for selection. If a stimulus has been selected and a saccadic eye movement is directed towards that stimulus, the trajectory of the saccade does not follow a straight line, but often shows some curvature instead (Bahill & Stark, 1975 ; Yarbus, 1967; for review see Van der Stigchel, Meeter, & Theeuwes, 2006). Importantly, saccadic curvature seems to be linked to the inhibition of competing distractor stimuli. If for example a participant is instructed to saccade towards a peripheral target and to ignore a distractor stimulus that appears together with the target at a flanking position, the trajectory of the saccade curves away from the distractor location (Doyle & Walker, 2001). Micro-electrode recordings in the monkeys frontal eye field and superior colliculus have shown that for saccades that curve *away* from a distractor stimulus activity at the distractor site is suppressed below the level of activity observed during straight saccades. On the contrary, for saccades that curve *towards* a lateral distractor, activity at the distractor site appears to be elevated (McPeek, Han, & Keller, 2003; McPeek 2006). These curvature effects have been explained by interactions among the neuronal population codes representing the saccade towards the target and distractor respectively (Tipper, Howard, & Jackson, 1997; Tipper, Howard, & Houghton, 2000). If a distractor stimulus is presented with sufficient proximity to the target, activity within the topographic motor-maps coding for both targets might overlap. In the process of target selection, reactive inhibition (Tipper et al., 1997, 2000) of the distractor site then inflicts suppression on some neurons coding for the target position as well. This modification of the motor program for the target-directed saccade is assumed to cause initial curvature away from the distractor because the activity peak of the target-related population code is shifted away from the distractor location. Curvature back towards the actual target position at the end of the saccade has been suggested to be caused by cerebellar control mechanisms (McSorley, Haggard, & Walker, 2004; Quaia, Lefevre, & Optican, 1999).

Behavioral evidence for the existence of such a “reactive” inhibition process comes from studies showing a positive correlation between saccadic latency and curvature away (McSorley, Haggard, & Walker, 2006, 2009; Walker, McSorley, & Haggard, 2006). If the target and distractor stimulus appear at unpredictable locations, and thus any selection process cannot start prior to their simultaneous onsets, short latency saccades directed at the target curve *towards* the distractor location while long latency saccades curve *away*. This effect is in line with the hypothesis that inhibition of the distractor site gradually builds up after the distractor evoked some activity initially.

The present experiment is concerned with the acquisition of associative memory, and competitive selection processes have been reported to exert inhibitory control during memory retrieval as well. Here inhibitory processes are “recruited specifically to support retrieval in the face of distraction from interfering representations” (Levy and Anderson, 2002). The effect of *retrieval induced forgetting* (Anderson, Bjork, & Bjork, 1994) for example shows that after training the retrieval of “Tomato” given the cue “Red”, subsequent retrieval of “Radish” given the cue “Food” is degraded (Anderson and Spellman, 1995). The effect has been explained, by assuming that cue “Red” triggers a retrieval process in which the red items “Tomato” and “Radish” compete for selection. In this process “Radish” repeatedly became inhibited because “Tomato” had to be retrieved during training and thus the threshold for selecting “Radish” was raised when it eventually was to be retrieved during the test stage.

As exemplified, a range of different empirical findings point to the existence of selection mechanisms that includes inhibitory control in order to minimize interference from distracting stimuli, memories or responses. The inhibitory processes here are triggered by the initial *excitation* of two competing alternatives. A somewhat different inhibitory mechanism that enables an organism to selectively suppress an interfering response has been advanced in the field of associative learning theory.

Conditioned Inhibition

Associative learning is the process by which organisms come to anticipate potentially significant events in order to adopt to their environment. For example, the ability to learn which food causes nausea and which food secures nutrition, clearly has some adaptive value. Here too, some learning situations call for inhibitory control to assure correct predictions and thus to guarantee adequate responding. In an animals habitat fruits may be generally edible if they have a red color, however one special small red fruit may be poisonous and the feature 'small' thus should come to inhibit any consummatory response elicited by 'red'. The discrimination problem at hand has been investigated in classical conditioning experiments since the days of Pavlov (1927) and has been known as the *standard paradigm of conditioned inhibition* or *feature-negative training* A+, AX-. If CS A is reinforced when presented alone, but not reinforced when presented in compound with a second CS X, the suppression of conditioned responding in AX trials is assumed to rely on a learned inhibitory association that counteracts the conditioned excitation elicited by A.

Two empirical procedures have been established as standard tests for conditioned inhibition (Rescorla, 1969; Savastano, Cole, Barnet, & Miller, 1999). In the *summation test*, after training A+, T+, AX-, the supposed conditioned inhibitor X must decrease conditioned responding to the separately trained excitatory test cue T (TX?), and this decrease should be greater than compounding T with an untrained, novel stimulus N (NX?). The *retardation test* for conditioned inhibition builds on the assumption that if the trained conditioned inhibitor is subsequently reinforced, the acquisition of conditioned responding should be retarded. Thus training X+ should proceed slower than training a novel, associatively neutral stimulus N+. With many empirical demonstrations of conditioned inhibition as measured by this two-test strategy, the

Rescorla-Wagner model, has been quite influential in assuming that learning may involve the acquisition of inhibitory associations (for alternative accounts see Konorski, 1967; Miller & Matzel, 1988; Pearce & Hall, 1980).

We have discussed two different sources of inhibitory control that act in order to suppress maladaptive behavior. *Distractor inhibition* denotes a process which actively suppresses the internal representation of an “incorrect” alternative whenever this activation interferes with the selection and production of some other, “correct” response. *Conditioned inhibition* on the other hand denotes the process in which a predictive cue, via a learned inhibitory association, acquires the potential to suppress “incorrect” predictions in the first place. In the next section we provide simulations of an associative network based on the RW-model learning rule that is trained with an extended, multiple-outcome versions of the standard paradigm of conditioned inhibition, A+1, AC+1, AX+2, X+2. With this training some cues are predicted to acquire an excitatory association to the trained, correct outcome while competing outcomes are predicted to be suppressed by learned inhibitory associations. On the contrary, other cues are predicted to hold concurrent excitatory associations to two competing outcomes. We will use the predicted changes in associative memory to derive hypothesis on how these changes might affect the curvature of predictive saccades indicating the selection of one outcome over the other.

Model Simulations

Discrimination Problem: Multiple-Outcome Conditioned Inhibition Training

The simulation is based on the same trial sequence that our human participants were trained with in the predictive saccade task (see Figure 3 for a short description of the task; the method section will give further details). Table 1 depicts the discrimination problem. Cue A was trained to predict an upper right saccade target while cues AX and X predicted an upper mid target, A+R, AX+M, X+M (were letters A and X denote different cues and M and R denote the right and mid target location respectively). An equivalent discrimination B+L, BY+M, Y+M, was simultaneously trained to differentiate between the mid and left position. Filler trials AC+R, BC+L were included to prevent participants from learning the abstract rule that a compound stimulus always predicted the target to appear at the mid position[1]. The trial sequence consisted of 6 blocks with 4 replications of trained trial types per block. Training started with establishing the left versus right discrimination in Block 1, additional training of the mid position started with Block 2. Cues A, B, AC, BC and AX, BY, X, Y thus were presented with 24 and 20 replications respectively, yielding a total of 176 training trials.

Table 1 about here

Formally the discrimination problem can be decomposed into two *feature-positive* discriminations A-, AX+ and B-, BY+ for the mid outcome and two simultaneous *feature-negative* discriminations A+, AX- and B+, BY- for the right and left outcome respectively.

One critical aspect with this training schedule is for participants to learn that compound cues AX and BY predict the mid position and that any response tendencies towards the lateral targets R and L elicited by stimulus elements A and B have to be suppressed to generate the correct response. As model simulations in the next section will reveal, with this training schedule, stimulus elements X and Y are predicted to acquire inhibitory associations to the right and left outcome respectively.

Acquisition of Cue-Outcome Associations by a Connectionist Network

Figure 1a depicts a simple connectionist network model capable of representing the discrimination problem at hand. The input layer consists of nodes coding for the trained stimulus elements, the output layer represents the target locations to be predicted. The layers are completely interconnected by associative links that change their strength (or weights) during training. This connectionist perspective on memory and information processing being "distributed", i. e. encoded in a set of multiple associative links acquired during learning, has provided a viable perspective on memory and human classification learning (Anderson, 1983; Gluck & Bower, 1988; Hinton, 1981; McClelland & Rumelhart, 1985; Shanks, 1991). In simple network models that do not assume any hidden layers (e. g. Shanks, 1991) the squared prediction error within the outcome layer is minimized by updating the weights according to the least mean squares rule of Widrow and Hoff (1960) which if formally equivalent to the Rescorla-Wagner (RW-) model learnig rule shown in Figure 1b, where α and β are learning rate parameters determined by the CS and US respectively, λ specifies the strength of the US teaching signal and ΣV is the summed associative strength of all CS currently present to predict the US. The two main characteristics of this error-correction learning rule are, (1) that the outcome has to be "surprising" to elicit any changes in associative strength and (2) that cues compete in generating the correct prediction. Learning is driven by the error term $[\lambda - \Sigma V]$ where λ has some fixed positive value $0 < \lambda \leq 1$ for trials in which the US is presented and λ is zero for trials in which the US does not occur. For simple pairings of CS A with a US in a classical conditioning experiment the associative strength V_A connecting CS A with the US thus will increase as long as $V_A < \lambda$ and will approach $V_A = \lambda$ asymptotically. If training A+ is accompanied by non-reinforced trials AX-, the error term in these AX-trials, $[0 - (V_A + V_X)]$, will be negative to the extent that CS A has been established as a conditioned exciter, $V_A > 0$, and this will gradually render CS X a conditioned inhibitor that exhibits a negative associative link to the US. In the model, inhibitory control thus consists of a learned association that inflicts suppression on the activity of the US representation.

Figure 1 about here

The generalization of the RW-model to the prediction of multiple-outcomes shown in Figure 1 is straightforward. A cue here exhibits associations to *all* outcome units while weights are adjusted using the very same learning rule. Importantly, with this type of model, after a learning trial, a cue might not only increase its associative strength to the outcome presented, but *at the same time* decrease the associative strength to an outcome that was predicted but did not occur. Figure 1c exemplifies this mechanism for weight changes in AX+M trials affecting element X, which will increase its association to the presented M outcome as long as $V_{A\text{-}M} + V_{X\text{-}M} < 1$ and at the same time will decrease its association to outcome R to the extent that $V_{A\text{-}R} + V_{X\text{-}R} > 0$. Figure 1d depicts a computer-simulation of weight changes during training as predicted by applying the RW-model learning rule to the network shown in Figure 1a. The panels show the net associative input that the outcome units will receive when each of the cues is presented (trials B, BC, BY and Y are not shown, because they exhibit an exactly reverse effect).

The simulation reveals that cue X will be established as a conditioned exciter of outcome M with $V_{X\text{-}M} > 0$ and at the same time as a conditioned inhibitor for outcome R with $V_{X\text{-}R} < 0$. In contrast, cue A will become a conditioned exciter for outcome R and initially also gain some excitatory strength for outcome M due to learning in AX+M trials. However, the interfering A-M-association is predicted to extinguish in the course of training due to decrements in A+R trials. A similar course of initial acquisition and subsequent extinction of an interfering association is predicted for cue AC. For the compound cue AX, target selection even asymptotically is challenged by a considerable amount of excitation of the right outcome, interfering with the correct prediction of outcome M. The interference is caused by A's prediction of outcome R that only partially is counteracted by the acquired inhibitory X-R-association. Inhibition of outcome R in AX trials after training A+R, AX+M, X+M is incomplete because cue X not only gains inhibitory X-R-strength in AX+M trials but also looses some of this conditioned inhibition in X+M trials.

In sum, associative learning as predicted by the RW-model learning rule will leave cues A, AC and AX ambiguous to some extent because they associatively retrieve competing predictions. Cue X on the other hand will unequivocally predict the correct outcome M, because outcome R is effectively suppressed by an acquired inhibitory X-R-association.

Hypotheses: The Impact of Associative Memory on Saccade Trajectories

For the time being we presume that the least mean squares network based on the RW-model learning rule provides the best educated guess about how changes in associative memory might evolve during learning. We now turn to the question, of how the acquired associative status of a cue might affect the curvature of saccades elicited by that cue. Figure 2 illustrates four contradictory hypotheses about how learned associations might influence saccade trajectories at the end of training. A more detailed analysis concerning the course of training will be provided in the discussion to this article.

Figure 2 about here

H0: No curvature. Because the present experiment to our knowledge is the first to examine the associative modulation of saccadic curvature, we would like to explicate the null hypothesis. It refers to the possibility that the representational substrate coding for saccade targets does not receive any *direct* input from the associations learned during training. Conflicts in associative memory might be resolved at higher-order processing stages in a *one-winner-takes-all* fashion *before* the result of that selection process is projected to oculomotor centers. Any information about concurrent associations thus would already be lost at the time of saccade execution and trajectories, unaffected by any outcome other than the predicted target, should exhibit no curvature at all.

H1: Conditioned inhibition. Saccadic curvature might correspond to the associative activation of outcome units. If a learned inhibitory association inflicts suppression upon the receiving outcome unit, correct vertical saccades elicited by X should curve to the left, i. e. away from the inhibited right target. On the contrary, vertical saccades elicited by AX should deviate towards the interfering right location to the extent that A's prediction of the right target is not counteracted by the inhibitory X-R-association.

H2: Distractor inhibition. Curvature of associatively-guided saccades might be caused by competitive interactions between predicted outcomes in much the same way that visually-guided saccades are affected by multiple stimuli in the visual periphery competing for selection. If saccadic curvature is caused by a selection process that is triggered whenever two outcomes are associatively activated above baseline, correct vertical saccades elicited by cue AX should curve to the left that is away from the position that initially was activated by the excitatory A-R-association and subsequently became inhibited in the process of active target selection. From this perspective, curvature indicative of such a target selection process should be absent in saccades elicited in X-alone trials, because with outcome R already being suppressed by the inhibitory X-R-association there was no conflict between predictions in the first place and thus no need for triggering any active distractor suppression.

H3: Any inhibition. Our final hypothesis refers to the possibility that saccade trajectories may deviate away from a location regardless of the source of inhibitory control. Saccade trajectories might be affected by acquired feed-forward associative weights as well as any form of inhibition resulting from active distractor suppression. Saccades then should exhibit leftward curvature in both, AX- *and* X-trials.

Interim Summary

We used a simple connectionist network based on the RW-model learning rule to predict changes in associative memory when participants learn to associate multiple different cues with multiple different outcomes. We derived contradictory hypothesis on how the predicted associative status of the cues might affect the trajectory of saccades triggered by these cues. In summary, the current experiment aims at answering the following questions:

(1) Is saccadic curvature affected by concurrent expectancies as acquired during associative learning?

(2) Is curvature sensitive to changes in associative memory as they gradually evolve during training?

(3) Is curvature caused by competitive interactions between conflicting predictions and/or by learned inhibitory associations?

Method

Participants

Twenty-three undergraduate students of the University of Marburg participated in the experiment and received either course credit or payment. All participants had normal or corrected-to-normal vision. The data of three participants were excluded from further analysis because of signal noise or calibration failures. Two participants were excluded, because they performed poorly on the learning task, leaving to few correct saccades that could be analyzed for curvature effects. Of the remaining participants 12 were female and 6 were male. Their age ranged from 20 to 28, *M* = 22.7, *SD* = 2.44.

Apparatus

Testing took place in a sound-attenuated, dimmed room. Monocular eye movements were recorded using an infrared video-based eye tracker (Eyelink 2000, SR-Research) that sampled position of pupil and corneal reflection at 1000Hz. Sampling sides (left/right eye) were counterbalanced across participants. The eye tracking column restrained the participants head via chin and forehead rests. It was table-mounted in front of a 22” CRT-monitor (Iiyama, Vision Master Pro514) for computer-controlled stimulus presentation, yielding an eye to screen distance of 78 cm. To prevent environmental distraction from the experimental chamber the screen was framed by a rectangular funnel-shaped aperture that opened up from screen size to a horizontal diameter of 80cm on the participants site. Its dull light-gray inner surface was homogeneously illuminated by hidden LED panels yielding an optimal indirect illuminance of the participants eye.

Stimuli

Visual stimuli were presented on a dark gray (25%) background. In the upper hemifield three 1.5° x 1.5° target boxes were shown at the right (45°), straight up from fixation (90°) and to the left (135°). All boxes had the same distance of 7.3° to the central fixation point. Participants performed saccades to a target stimulus that could appear inside one of the three target boxes. The target stimulus was a white annulus with 1.1° outer and a 0.5° inner radius. At target onset a high acuity feature was flashed inside the annulus for 30ms. It could consist of either a single vertical line or a horizontal and vertical line forming a cross. The shape of this high acuity feature could only be identified if participants already fixated the correct target box prior to target onset.

As central cues we used rectangular pictograms of different fruits measuring 1.6° x 1.6° of visual angle. In compound trials AX, BY etc. two different fruits were presented side by side. The position of an element in the compound stimulus was counterbalanced across trials within subject so that nominal AX-trials consisted in fact of an equal number of AX and XA presentations. The fruits used were grapes, strawberry, lemon, peach, pineapple and kiwi, as depicted at the right side of Figure 3.

Procedure

At arrival participants gave written consent to the requirements to try to sit still and to avoid blinking during sampling intervals, as well as the anonymous storage and analysis of their data. Written instructions were presented that exemplified the events and task demands that occurred within a trial. Sixteen practice trials were run prior to the actual experiment to assure that participants had understood the instructions. Demographic data were collected on a post-experimental questionnaire.

The eye tracker was calibrated using a 13-point grid of calibration targets. If necessary the calibration procedure was rerun until the subsequent validation procedure confirmed an average calibration error < 0.5°.

Figure 3 about here

The sequence of events within a trial is depicted in Figure 3. A trial started with the presentation of a central fixation cross for 2000ms that instructed the participant to stop blinking and pay attention. The pictogram of a fruit was then presented at the center of the screen for 2000ms followed by the peripheral target stimulus appearing in one of the three target boxes. Participants were instructed to learn which fruit predicted which target position and to perform an anticipatory saccade to the cued target box. If participants correctly anticipated the target position they could detect the faint flash of a vertical line or a cross within the target annulus for 30ms. The vertical line instructed them to press the mouse button once while they made a double-click if they detected the cross. Auditory feedback was given in form of a high versus low pitch tone that indicated a correct or erroneous manual response respectively. The high acuity feature at target onset was detectable only if participants fixated the correct target box prior to target onset. Practice trials prior to the experiment were identical to trials in the experiment besides using an arrow as the central cue pointing to one of the three target boxes.

The trial sequence was the same used for the model simulations and is depicted in Table 1. Trials labeled AC and BC in fact consisted of an equal number of AC and AD as well as BC and BD presentations. Because model predictions were equivalent for these trial types and analysis revealed no differential responding, the results section will report average responses using the labels AC and BC. We used different pseudo-random trial sequences for each participant. The sequence of trials was randomly shuffled within blocks, restricting the same cue or outcome to be presented a maximum number of three times in a row.

Data Analysis

We used custom software written in Matlab (Koenig & Lachnit, 2010a) for the parametrization of saccades. Saccades were detected using a velocity based algorithm. An eye movement qualified as a saccade if eye velocity exceeded 60°/s. Start and endpoints were set at 15% peak velocity. *Saccadic curvature* was computed as the area (mm^2) enclosed by the saccade trajectory and a straight line joining start- and endpoint of the movement divided by the squared saccadic amplitude (mm^2) multiplied by 100. Curvature was assigned negative values, if the deviation from a straight line was clockwise (right-ward curvature for saccades into the upper hemifield), positive values were assigned to denote counter-clockwise deviations (leftward curvature).

Repeated measures analysis of variance (ANOVA) was used to analyze the data. Degrees of freedom were corrected with the Hunh-Feldt method (Huynh & Feldt, 1976). Frequency data were subjected to acrsine transformation (Rao, 1960). Unless stated otherwise ANOVAs include the factor cue with levels A, AC, AX, X, B, BC, BY and Y, as well factor block (2, 3, 4, 5, 6). Analysis concerning A, AC B and BC only will also include the first block of training.

Data Exclusion

Some trials contained temporary signal loss during the cue interval due to eye blinks or partial occlusion of the pupil by lashes or lid. Because some of these signal losses masked or could have masked an eye movement, on average we exclude 5.5% of the trials in each record. For the analysis of saccadic curvature we only used correct trials. A trial was scored as correct if the participant made an anticipatory saccade from fixation to the correct target location (direction error < 20°) that had a minimum amplitude of 4°.

Results

Exploratory Analysis

In the current experiment the presentation of the central cue elicited a sequence of different types of saccades that eventually directed gaze to the anticipated target location. Figure 4 depicts endpoints of saccades occurring in the cue interval. Three major subsets of saccades can be identified from the figure: First, saccade endpoints cluster around the trained target locations, i. e. participants performed saccades that directed their gaze towards the correct target box in anticipation of the target. They learned to look right, whenever cue A was presented while they looked to the left whenever cue B was presented et cetera. Secondly, saccade endpoints cluster around the central cue, because of fixational eye movements. As can be seen from Figure 4 these fixational saccades were more frequent in compound trials AX, BY, BC and AC because the cue to be encoded is perceptually and/or associatively more complex than in trials presenting the elements A, B, X or Y. Thirdly, some saccades were directed towards the wrong target box in anticipation of the target. Cues trained with a lateral position, A, AC, B and BC, sometimes elicited saccades that went to either of the competing two positions. Erroneous saccades elicited by compound cues AX and BY on the other hand selectively led to the lateral position that was trained with A and B respectively. For cues X and Y Figure 4 shows an almost complete absence of any erroneous anticipatory saccades.

Figure 4 about here

To explore how this oculomotor behavior evolves during the cue interval, Figure 5 plots saccadic amplitude versus saccadic latency relative to cue onset for each level of the cue factor. The Figure shows that higher-amplitude saccades (target eccentricity was 7.3°) had different latencies when comparing element and compound cues. Saccades elicited by the compound stimuli showed longer latencies with greater variance than saccades elicited by the elements. In the following sections we will present detailed analysis of how the frequency, latency and curvature of these cue specific saccades evolved during training.

Figure 5 about here

Frequency of Correct Saccades

Acquisition curves in Figure 6 show the increase in frequency of anticipatory saccades that direct gaze to the correct target location as a function of training. The bottom-left panel *A2000* depicts the relative frequency of trials in which the last saccade prior to target onset (i. e. 2000ms after cue onset) ended at the correct target location (within a radius of 38.25mm around the correct target; neighboring target position were 76.5mm apart). As can bee seen, participants needed only very few trials to learn the discrimination so that eye position anticipated the correct target location in over 90% of the trials even in the first block of training. However this high percentage of correct anticipation was the result of *all* saccades occurring within one trial, i. e. the first saccade might have gone to a wrong position and subsequent saccades then corrected this first position to the final anticipatory position. The remaining panels of Figure 6 show the percentage of trials in which the *first* single saccade in a trial with an amplitude > 4° starting at the central cue position was directed at the correct target location with a direction error < 20° . This population of correct saccades will be analyzed for curvature effects later on. Panels *F500*, *F750*, *F1000* and *F2000* show the percentage of trials in which such correct anticipatory saccades occurred with a latency of less than 500, 750, 1000 and 2000ms respectively. Table 2 depicts ANOVA results for the arcsine-squareroot transformed (Rao, 1960) data. Main effects of cue and block were significant for all cumulative intervals, only in interval *F500* main effects were modulated by a significant Cue X Block interaction, $F(28, 476) = 1.579$, $\eta_p^2 = .085$, $p < .05$. The increasing effect of factor cue for earlier intervals was confirmed by an overall ANOVA, yielding a significant interaction Cue X Interval, $F(21, 357) = 12.221$, $\eta_p^2 = .418$, $p < .001$. Post hoc tests revealed that collapsing across block, elements X and Y trained with the upper mid position elicited correct saccades with a higher frequency than any of the remaining cues at all intervals (all $p < .05$). Also, regarding saccades that started within the first 1000ms after cue onset (F500, F750 and F1000 in Figure 6), elements A and B trained with the right and left position respectively, elicited correct saccades with a higher frequency than any trained compound (all $p < .05$). In sum correct anticipatory saccades were most frequent with cues X and Y and correct saccades elicited by elements A, B, X and Y were more frequent in early intervals after cue onset as compared to correct saccades elicited by compounds, AC, BC, AX and AY.

Table 2 and Figure 6 about here

Latency of Correct Saccades

As evident from Figure 5 saccades elicited by compounds had longer and more variable latencies than saccades elicited by the elements. Also Figure 6 shows that correct saccades are more frequent for elements than for compounds at early intervals after cue onset. Figure 7 depicts the decrease in the latency of correct saccades with training. An ANOVA revealed significant main effects for cue, $F(7,119) = 50.357$, $p < .001$, $\eta_p^2 = .748$, and block, $F(4,68) = 8.733$, $p < .001$, $\eta_p^2 = .339$. Post hoc tests showed that collapsed across blocks, correct saccades elicited by the trained elements were of shorter latency than saccades elicited by the compounds (all $p < .001$). Separate contrast analysis for each cue revealed a significant linear trend for all cues (all $p < .05$) except for elements B ($p > .391$).

Figure 7 about here

Inspection of Figure 7 also shows that at the end of training in Block 6, within the same type of cue, i. e. within elements and within compounds, there was a tendency for saccades directed to the mid position to be of shorter latency than saccades directed at the lateral positions (X and Y compared to A and B as well as AX and BY compared to AC and BC). To further examine this difference we conducted a 2 X 2 X 2 ANOVA for Block 6 with factors type (of cue: elements A, B, X, Y versus compounds AC, BC, AX, BY), saccade direction (vertical AX, BY, X, Y versus oblique A, AC, B, BC) and side (of discrimination: right A+R, AC+R, AX+M, X+M versus left B+L, BC+L, BY+M, Y+M) which revealed a significant main effects for saccade direction, $F(1,17) = 25.634$, $p < .001$, $\eta_p^2 = .601$, confirming that latencies were significantly longer for oblique saccades than for straight-up saccades.

Curvature of Correct Saccades

Figure 8a shows curvature of correct saccades elicited by the trained cues in successive blocks of training. An ANOVA revealed a significant main effect for cue, $F(7,119) = 36.240$, $p < .001$, $\eta_p^2 = .681$, that was modulated by a Cue x Block interaction, $F(28,476) = 1.921$, $p < .016$, $\eta_p^2 = .102$. Collapsing across blocks, post hoc tests revealed that the curvature of oblique saccades elicited by cues A, AC, B and BC was of higher absolute amplitude than curvature of vertical saccades elicited by cues AX, BY, X and Y (all $p < .05$). Oblique saccades curved away from the mid position: Saccades directed to the left target position in trials B and BC curved to the left, while saccades directed to the right target position in A and AC trials curved to the right. This apparent baseline curvature then further increased with training. One-sample t-tests revealed that curvature of oblique saccades elicited by A, AC, B and BD was different from zero in every single block of training (all $p < .05$) wheres the same was true for vertical saccades only with AX-sacaddes in blocks 5 and 6 as well as BY-saccades in Block 2.

Figure 8 about here

The main focus of our experiment was to detect any associatively induced differential curvature in anticipatory saccades directed to the mid target position: In BY and/or Y trials the left target position should become inhibited and upwards saccades thus should curve to the right while in AX and/or X trials the right position should become inhibited and saccades should curve to the left. Testing for reverse curvature effects in X versus Y and AX versus BY saccades within subject thus provides a sensitive test to this hypothesis. We conducted a 2 x 2 x 5 ANOVA with factors type (of cue: elements X, Y versus compounds AX, BY), site (of inhibition: left for BY, Y versus right for AX, X) and block (2-6) that revealed a significant three-way interaction Type X Site X Block, $F(4, 68) = 4{,}236$, $p < .005$, $\eta_p^2 = .199$. As can be seen from Figure 8a this interaction is caused by the presence of opposed trends for trials AX vs. BY while a change in curvature is absent in X and Y trials. Separate analysis confirmed a significant interaction Cue X Block comparing AX and BY, $F(4,60) = 4.296$, $p < .004$, $\eta_p^2 = .201$ while any differential curvature was absent comparing X and Y, $F < 1$. The block-wise comparison of curvature in AX versus BY trials revealed a significant difference in curvature for blocks 2, 5 and 6 (all $p < .05$) while X and Y trials did not differ in any block of training. Figure 9 exemplifies this interaction by comparing average saccade trajectories for X versus Y and AX versus BY in the first and last block of training.

Table 3 about here

In Figure 8b we computed saccadic curvature to indicate average curvature away from the mid position for trials A/B and AC/BC, as well as curvature away from the respective lateral positions for trials AX/BY and X/Y. To compensate for baseline differences in oblique versus vertical saccades values were centered within panel by subtracting the respective means. Table 3 depict the results of a contrast analysis testing for linear and quadratic trends in these data. While the gradual increase in curvature away followed a linear trend in trials AX/BY, a quadratic trend best described changes in A/B and AC/AD trials. Curvature away here initially increased and decreased again towards the end of training. Curvature in trials X/Y did not change during training.

Curvature changes during training were superimposed on some subject- and direction-specific baseline curvature that we did not measure in this experiment. As Figure 9 reveals, the absolute amplitude of rightward-curvature in saccades elicited by

AX and BY was less than curvature to the left. Because with the current design, X and Y as well as AX and BY within-subject provided a baseline for each other, this asymmetry in absolute curvature is irrelevant for our current concern. Importantly however, the curvature *acquired* by AX and BY during training (max - min) was about the same size. The same was true for oblique saccades. Despite the fact that the *absolute* amplitude of unsigned curvature for oblique saccades broadly exceeded curvature of vertical saccades, and right-ward curvature in A- and AC-saccades was greater than leftward-curvature in B- and BC-saccades (cf. Figure 8a), changes in saccadic curvature with training again took place in about the same range.

In sum, our analysis revealed a linear increase in curvature away from the lateral positions in AX- and BY- saccades, while significant changes were absent in saccades elicited in X and Y. Progression of curvature in oblique saccades followed a quadratic trend with initial increase and subsequent decrease in curvature away.

Figure 9 about here

Discussion

Summary of Main Empirical Results

In the present experiment we explored how the trajectories of saccadic eye movements were affected by associative learning and memory. Human participants learned to perform saccadic choice responses based on the presentation of arbitrary central cues that were trained to predict the appearance of a peripheral target stimulus at one out of three possible locations, right (R), mid (M) or left (L), in the upper hemifield. As dependent variables indicative of associative learning we analyzed frequency, latency and curvature of saccades elicited by the central cues that were directed at the trained locations in anticipation of the target. We trained two concurrent discrimination problems A+R, AC+R, AX+M, X+M and B+L, BC+L, BY+M, Y+M, derived from the standard paradigm of conditioned inhibition (Pavlov, 1927; Rescorla, 1969). We analyzed, how the associative status of a cue acquired during training affected saccade trajectories.

In the course of training associative learning led to an increase in the frequency of correct predictive saccades while at the same time saccadic latency decreased. Importantly, saccadic curvature was affected by the gradual acquisition of associative memory as well. In the first block of training the trajectories of vertical saccades elicited by AX and BY exhibited significant differences in curvature. Saccades elicited by AX curved to the right, i. e. towards the position trained with A, while saccades elicited by BY curved to the left, i. e. towards the position trained with B. This differential curvature effect then reversed in the course of training. In the last two blocks of training, cues AX and BY again elicited saccades differing in curvature, this time however, saccades had acquired curvature *away* from the locus of associative interference. Vertical saccades elicited by AX acquired curvature away from the interfering right position trained with element A, while vertical saccades elicited by BY acquired curvature away from the left position trained with B. Trajectories elicited by cues X and Y on the other hand, did not differ at any block during training.

Curvature in oblique saccades elicited by A, AC, B and BC was more pronounced than curvature of vertical saccades. However, besides these baseline differences, curvature of oblique saccades also changed during training. While there was a linear increase in curvature away from the lateral positions in AX- and BY-elicited saccades, changes in curvature followed a quadratic trend in saccades elicited by A, AC, B and BC. Curvature away from the mid position increased in the first half of training and decreased again towards the end.

Changes in Frequency and Latency of Correct Saccades

Our experiment was designed primarily to explore the curvature of predictive saccades. Before we turn to the discussion of the observed curvature effects we start with analyzing the acquisition of these correct anticipatory saccades. In the course of training all cues gradually acquired the potential to elicit correct anticipatory responding. The frequency of correct predictive saccades increased (cf. Figure 6) and their latency decreased (cf. Figure 7). However, as evident from Figure 6, trained cues exhibited differential acquisition rates. Cues X and Y were learned fastest, cues A and B were learned second best and learning was worst for the remaining cues. The model simulation in Figure 1d reveals that these differences in conditioned responding are predicted by the least mean squares network, if the strength of the conditioned choice response is assumed to be some positive function that contrast the prediction of the correct outcome with the prediction of the competing outcome(s), $F(V_{X\text{-}M} - V_{X\text{-}R}) > F(V_{A\text{-}R} - V_{A\text{-}M}) > F(V_{AX\text{-}M} - V_{AX\text{-}R}) \approx F(V_{AC\text{-}R} - V_{AC\text{-}M} - V_{AC\text{-}L})$. Interference between contradictory associations as predicted by the RW-model learning rule thus yields one plausible explanation of the observed differences in correct responding.

As evident from Figure 7, cues elicited saccades with different latencies. At the end of training, differential latencies of correct saccades were X/Y < A/B < AX/BY < AC/BD. If conflict resolution triggered by concurrent excitatory associations is a time consuming process (McSorley et al, 2006, 2009; Walker et al., 2006), these differences in saccadic latency too are in line with the predictions of the least mean squares network. Slower responding to AC/BC as compared to AX/BY may be caused by AC/BD exhibiting *two* conflicting associations (cf. Figure 1d). However, in our experiment the amount of associative interference elicited by a cue was confounded with its perceptual complexity. Compound cues consisting of two stimulus elements were predicted to induce a greater amount of associative interference than single elements. For example, slower correct responding to compound cue AX as compared to the single element X could be caused (1) by AX retrieving an interfering prediction while cue X is unambiguous *or* (2) because cue AX demands more time of perceptual encoding. Also, faster responding to cue X as compared to cue A could be caused (1) by less associative interference elicited by cue X *or* (2) by oblique predictive saccades being slower than vertical saccades. In sum, the observed differences in acquisition rates and latency are in line with the predictions of the associative network but must be interpreted with caution due to missing control conditions.

Associative Interference Causes Saccades to Curve Away

We observed changes in saccadic curvature for all trained cues that induced some kind of associative interference. By contrast the only cues that did not elicit any changes in curvature at all were the unambiguous cues X and Y that were predicted to acquire no conflicting excitatory associations. As depicted in Figure 2, both observations are in line with a distractor inhibition account of saccadic curvature.

In the course of training cues AX and BY acquired curvature away from the right and left target position respectively. Saccades thus acquired curvature away from the position predicted to receive interfering excitatory input that competes with the generation of the correct response and thereby should become inhibited in the process of active target selection subsequently. Also, vertical saccades elicited by AX and BY changed the direction of curvature during training. They initially deviated towards the interfering lateral outcomes and then gradually acquired deviation away from these locations. Early rightward-deviation in AX-trials can be explained by the prediction of outcome M yet being weak as compared to the prediction of outcome R. Model simulations in Figure 1d reveal that the summed AX-M associations are predicted to exceed the summed AX-R associations not until Block 3. Curvature towards the lateral location thus could result from the associative activation of a lateral distractor while the prediction of the correct mid target is yet to weak to effectively suppress distractor activity. The observed effect complies with results published by Walker et al. (2006) showing that saccades deviate away from a lateral distractor if the target location is known in advance and may deviate towards the same distractor without prior knowledge of the target location.

The absence of curvature in X and Y trials also is in line with the distractor inhibition account. If curvature away indicates reactive inhibition (Tipper et al., 1997) of an interfering association, curvature should be absent in saccades elicited in X- and Y-alone trials which unequivocally predict the trained outcome M with competing excitatory associations being absent. As mentioned earlier, the unambiguous cues X and Y in fact were the only cues that did not acquire any differential curvature at all.

If curvature is induced by associative interference, model simulations in Figure 1d make another quite interesting prediction. The interfering activation of outcome M in trials A/B and AC/BD is predicted to exhibit a biphasic progression where interference initially builds up to some point and then gradually extinguishes in the course of further training. This exactly accords to the pattern of results we observed for changes in saccadic curvature in trials A, AC, B and BC. Oblique saccades elicited by these cues initially acquired curvature away from the interfering mid position and this curvature away then declined towards the end of training.

Figure 10 about here

In Figure 10 we explore a simple, tentative algorithm that computes the amount of curvature away based on the cues concurrent associations as predicted by the model simulation in Figure 1d. Figure 10a exemplifies the mechanism for cue AX, which after training retrieves the prediction of the correct mid target via association w_1 but also activates the interfering right target with association w_2, where $w_1 > w_2 > 0$. We assume the most simple input-activation function where activation of the outcomes equals their associative input, $a_M = w_1$, $a_R = w_2$. Simultaneous activation of the competing outcomes then triggers a secondary selection process in which the distracting outcome R receives inhibitory input from the correct mid outcome, $(a_R - a_M) = (w_2 - w_1)$. Because the process should selectively suppress *competing* outcomes, the amount of inhibition received by outcome R is gated by its own state of activation. Competing associations w_2 are set to zero where $w_2 < 0$ as is the case for cue X, which holds a concurrent negative association that should not induce any conflict at all. The upper panels of Figure 10b depict progression of distractor suppression as predicted by this mechanism, the lower panels depict changes in saccadic curvature observed in our experiment. Changes in the associative status of the cues as predicted by the RW-model learning rule nicely fit the observed changes in saccadic curvature. Cue X is predicted to exhibit no curvature at all. Cue AX is predicted to elicited saccades that gradually acquire curvature away until predictions AX-M and AX-R reach their asymptotic thresholds. Cues A and AC are predicted to exhibit biphasic progression of suppression because of biphasic changes in the interfering A-M association.

It appears interesting to note that our models correct description of curvature changes as a function of associative interference critically relies on an associative network actually *producing such interference*. The residual activation of outcome R in trials AX for example (cf. Figure 1d), is caused by the network being incapable of reducing interference any further. From a theoretical perspective, interference could be readily reduced by additional model assumptions such as unique cues (Wagner & Brandon, 2001; Wagner, 2003; Whitlow & Wagner, 1972), stimulus configurations (Pearce, 1987, 1994, 2002) or hidden-layer networks using the backpropagation learning algorithm (Rumelhart, Hinton, & Williams, 1986). These models would, at least asymptotically, predict no interference at all. Because in the current experiment changes in frequency, latency and curvature did not reach a clear-cut steady state at the end of training it might well be that training even after 176 trials left the cues with pre-asymptotic associative strength and curvature away in AX trials would regress with further training. Future experiments should employ extensive over-training schedules to further evaluate this topic.

Conditioned Inhibition Does Not Affect Saccade Trajectories

The RW-model assumes that inhibitory control over conditioned behavior is implemented by the acquisition of inhibitory associations. This view, in one or another way, has been incorporated into many subsequent models of associative learning

(Harris, 2008; McLaren & Mackintosh, 2000, 2002; Pearce, 1994, 2002; Wagner, 1981, 2003; but see Miller & Matzel, 1988; Stout & Miller, 2007). After feature-negative training A+, AX-, the suppression of conditioned responding to AX is assumed to rely on a learned inhibitory association which, from the perspective of an elemental representation of stimulus features, connects stimulus element X with the trained US. The least mean squares network in Figure 1 based on the RW-model learning rule thus predicts that with training the two-outcome discrimination A+1, AX+2, cue X should be established as a conditioned exciter for outcome 2 and at the same time as a conditioned inhibitor for outcome 1. For the current experiment we derived the hypothesis that with differential training A+R, AX+M, B+L, BY+M, stimulus elements X and Y should acquire inhibitory associations acting upon outcomes R and L respectively. Saccades elicited by X and Y hence were predicted to curve away from the inhibited locations (cf. H1 of Figure 2). Contrary to this prediction, we observed no differential curvature in X- and Y-elicited saccades at any stage of training. If an inhibitory association X-R *was* acquired, inhibition of outcome R in trials X+M must have been of some different "quality" than distractor inhibition of the same outcome in trials AX+M which *did* cause curvature away. One possible explanation of this difference derives from a perspective on inhibitory learning offered long ago by Konorski (1948). Inhibitory associations might not modulate the activation of the outcome unit directly, but rather raise the outcome unit's threshold for excitatory activation. The inhibitory X-R weight would decrease any excitatory input to outcome R, but with this input being absent in X-alone trials, outcome R's activity would not be modulated at all. Some learning theories however do hold the assumption that inhibitory associations suppress the receiving unit's activation below baseline whenever it receives net inhibitory input (e. g. McLaren & Mackintosh, 2000, 2002). From this perspective, it is rather hard to explain, why excitatory associations do affect curvature, while inhibitory associations do not.

Using more traditional post-acquisition tests for conditioned inhibition previous studies with human participants have revealed that training including the A+, AX-, X-discrimination does establish cue X as a conditioned inhibitor for the trained outcome (Karazinov & Boakes, 2004; Williams, 1995). These studies however used scenarios in which participants predicted the presence versus absence of one single outcome. Conditioned inhibition in a multiple-category learning task was examined by O'Boyle and Bouton (1996) who trained human participants to predict which of several culprits (outcomes 1 and 2) committed a hypothetical crime based on the cues (A and X) the burglar left behind at the crime scene. After training A+1, AX+2, they instructed their participants to “use a scale of -10 to +10 to rate how likely it is that a given suspect committed a crime given a particular clue” and found indeed that participants rated cue X to be of negative predictive strength for the first outcome. The authors drew the conclusion that “inhibition can arise in a two-outcome situation”, assuming that the participants negative rating reflected *conditioned* inhibition. However, in the light of the present findings, one might want to consider the general validity of such post-acquisition tests. An alternative interpretation would assume that after asking how likely it is that outcome 1 was caused by cue X, negative ratings for X indicate reactive inhibition triggered by a test question which introduced outcome 1 as a potent distractor interfering with the correct X-2 prediction. Negative ratings thus could reflect an excitatory X-2-association just as well as an inhibitory X-1-association. Future experiments using the predictive saccade task should use post acquisition test to further examine the relation between saccadic curvature during training and more traditional tests for conditioned inhibition.

The RW-model assumes that inhibitory learning takes place, whenever the learning rule's error term is negative (cf. Figure 1b-c). Associative weights decrease if the predicted outcome exceeds the actual outcome, and conditioned inhibition naturally evolves from this assumption of negative weight changes. If negative associative weights X-R and Y-L *were* acquired during training, saccadic curvature was unaffected by these associations. Although our data hence did not yield any direct evidence for inhibitory learning with cues X and Y we did find evidence for inhibitory learning in trials A, AC, B and BC. These cues were predicted to acquire some excitatory associations to the incorrect mid outcome at the outset of training (Figure 1d). The acquisition of these interfering associations as predicted by the least mean squares network was accompanied by the acquisition of curvature away from the mid target location in our experiment. Importantly the model predicted the extinction of this associative interference due to inhibitory learning with further training and again saccadic curvature did correspond to these predicted changes in that curvature away regressed towards the end of training.

When participants learn to classify different cues as predicting different outcomes, our data hence seams to support the view that learning involves the simultaneous adjustment of concurrent associations. Learning in trials A+R did not only include the strengthening of the A-R associations but at the same time the strengthening and subsequent weakening of the A-M association in the first and second half of training respectively. This assumption of concurrent weight changes lies at the very heart of connectionism but has to our knowledge never been put to a direct empirical test. The present experiment provides some interesting behavioral data that support the connectionist perspective.

Related Findings From Non-Associative Paradigms

Some previous studies have examined how saccadic curvature is affected by memory and expectancy using experimental paradigms not related to associative learning processes. In an experiment by Van der Stigchel & Theeuwes (2006, Experiment 2) human participants were presented with central cues consisting of one short and one long line fragment pointing to different peripheral locations. The long line segment specified the position of an upcoming saccade target, the short line segment pointed towards a flanking position where a distractor stimulus could be expected to appear together with the saccade target in 80% of the trials. Saccades curved away from the cued distractor and importantly did so also in the 20% of the trials in which the distractor was anticipated but did not occur. These authors drew the conclusion that "the mere expectation that a distractor will appear at a specific location is enough to generate saccade deviations away from the location of the expected distractor". The results from our saccade learning task conform with this view. In our experiment the cues informing the participant where a target will occur, did not consist of two line segments but had acquired two conflicting associations predicting the target to occur at different locations and saccades curved away from the position predicted by the weaker association.

The mechanism of distractor inhibition we proposed in Figure 10 depends on both, associative activation of the target as well as the distractor. U-shaped changes in curvature away from the mid position in trials A, AC, B and BC are caused by initial acquisition and subsequent extinction of interfering associations predicting the mid target. The continuous growth of curvature away from the lateral positions for cues AX and BY on the other hand is caused by the gradual acquisition of associations predicting the mid target, while interfering associations predicting the incorrect lateral targets stay about the same during training (cf. Figure 1d). McSorley and McCloy (2009) reported a similar result in an experiment that varied the strength of evidence for a saccade target. Human participants performed saccades towards one out of two possible targets as cued by the dominant movement direction in a central moving dot pattern. The authors systematically varied the amount of dots moving in the same direction and thereby manipulated the amount of evidence for one stimulus becoming the saccade target. Results revealed that saccadic curvature away from the non-target stimulus increased with the amount of coherence specifying the target. Analogously, in our experiment, curvature away in trials AX and BY increased with the associative strength with which the cues predicted their trained outcomes.

Theeuwes, Olivers and Chizk (2005) reported that saccades deviate away from locations that are kept in working memory at the time of saccade execution. Human participants fixated centrally, while a dot was presented laterally in the upper or lower hemifield for 500ms. After a blank interval of 1000ms a central arrow instructed the participant to perform a vertical saccade to the upper or lower hemifield. In one condition the position of the dot had to be remembered and was queried in a memory test after the saccade at the the end of the trial. With this instruction, saccades deviated away from the to be remembered location. In a subsequent experiment Koenig & Lachnit (2010b) demonstrated that active remembering of the distractor location is not required to inflict curvature upon saccade trajectories. With an interval of 1850ms from the offset of the distractor to the onset of the target a distractor exhibits enough residual activity to cause curvature away without any explicit instruction to remember the target location. In the present experiment the memory of a distractor was *associatively* elicited by the presentation of a trained cue and saccades curved away from this locus of associative interference. The results are in accord with the view that the memory of the outcome retrieved associatively equals its decaying memory trace (e. g. Wagner, 1981).

The Automaticity of Cued Saccadic Choice Responses

The attention literature traditionally has emphasized a distinction between the exogenous versus endogenous control over attention and eye movements (Jonides, 1981; Müller & Rabbitt, 1989; Posner, 1980). This perspective is supported by empirical findings pointing to the involvement of different brain areas (for review see Corbetta & Shulman, 2002). With respect to eye movements, on a functional level, exogenous, visually-guided saccades, elicited by the sudden onset of a peripheral stimulus are believed to be rather reflexive and automatic in nature, whereas endogenous saccades instructed by symbolic, “cognitive” central cues are assumed to be programmed and executed in a less mechanistic way and instead to rely on “voluntary control”, “higher-cognitive processes” and “executive function”.

Contrary to this preconception of voluntary control, we would like to point to some degree of automaticity in the way the arbitrary “cognitive” cues in our experiment elicited the participants expectations and acquired control over oculomotor behavior during training. One aspect of our data that suggests such automaticity corresponds to the time at which saccades were elicited. We used a cue-to-target interval of 2000ms that participants could have used to decide on where the target would appear. This duration proved sufficient for participants to anticipate the correct position prior to target onset in over 90% of all trials from the very beginning of training if final eye position was reached by the sequence of *all* saccades occurring in the cue interval (Figure 6, Panel A2000). After all, we did not instruct participants to perform one single saccade and there was enough time making the correct prediction using multiple saccades. Analysis of the *first* single saccade in a trial revealed that this initial choice response was more likely to reach an incorrect location. For example the first saccade elicited by cues AX and BY were correct in about 60% of the trials at the start and about

80% of the trials at the end of training (Figure 6, Panel F2000). The cues in our experiment thus “automatically” elicited saccades at a time when the conflict between concurrent predictions was not yet resolved. Also, the observed changes in curvature during training suggest that predictive saccades were not the result of some “deliberate” reflection on the training schedule. For example, in our experiment cue A was *always* followed by a single saccade target on the right and AX was *always* followed by a single target at the mid position. Participants did not encounter one exception to this rule, nor was there any trial that actually would have presented two competing peripheral targets. A participant deliberately reflecting on the task structure thus should have disengaged from the possibility that something would *ever* appear to the right in AX trials. If “our eyes deviate away from a location where a distractor is expected to appear” (Van der Stigchel & Theeuwes, 2006) and “expectancy” is meant to be what the participant actually anticipates to happen, the amount of curvature should have decreased in the course of training. On the contrary, we observed that curvature away from the lateral positions in AX- and BY-trials was at its maximum at the end of training.

In sum, our results do not require to resort to any involvement of voluntary control to explain changes in frequency, latency and curvature of endogenous saccades during training. On the contrary, the present experiment suggests that the trained cues acquired associations to the trained targets driven by an error-correcting learning rule and that this set of concurrent associations had direct access to oculomotor centers affecting saccade trajectories. If “higher order cognitive processes” *were* responsible for the deliberate generation of expectations and the subsequent selection among competing alternatives, these higher order process were (a) in accord with a simple associative network derived from animal learning theory and (b) tightly coupled to selection processes at low-level oculomotor centers with respect to time and spatial scales. The point of automatic processing of central cues has recently been made with respect to central arrows directing attention in cueing paradigms (Hommel, Pratt, Colzato, & Godijn, 2001; Pratt & Hommel, 2003; Ristic & Kingstone, 2006). These studies show that a central arrow reflexively directs attention to the indicated target location even if the participants knows that this arrow has no predictive value. Hommel et al. (2001) have suggested that this reflexive orienting is triggered because the spatial relevance of the arrow symbol derives from an “overlearned representation” acquired during frequent encounters with its conventional spatial meaning. Orienting towards the attentional focus of one's communicative partner provides another example of such automatic processes (Langton & Bruce, 2000). Here the eliciting cues consist of the other persons face or gesture. Associative learning as described in the current experiment might provide the mechanism responsible for the acquisition of the spatial meaning of such cues.

Summary and Conclusion

We presented evidence that saccadic curvature is affected by competing predictions retrieved from associative memory. Amplitude and direction of curvature are sensitive to changes in associative memory as they evolve during training. Saccades acquire curvature to the extent that a cue induces associative interference and curve away from a position that is predicted by a concurrent excitatory association.

Saccades did not curve away from locations that were predicted to receive input from an acquired feed-forward inhibitory association. A conditioned inhibitor may not modulate the outcome units activity directly but rather raise its threshold for excitatory activation.

A least mean squares associative network based on the RW-model learning rule correctly predicted changes in saccadic curvature during training. The amount of distractor suppression indicated by curvature away appears to be some positive function of the amount of associative interference predicted by the network. Conditional saccadic choice responses may provide a valuable method to access the associative status a cue has acquired during learning with multiple outcomes. Saccades are directed at the location predicted by the strongest associations and the curvature of *the same response* yields insights into the associative activation of competing outcomes. Information about these concurrent associations within the same response is not easily obtained with other methods.

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Footnotes

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1 The experiment actually included trials AC+R, AD+R and BC+L, BD+L. Model simulations yield equivalent results for these trial types. For the sake of clarity trials AC hence denote average responding to AC and AD, trials BC denote average responding to BC and BD.

Figures

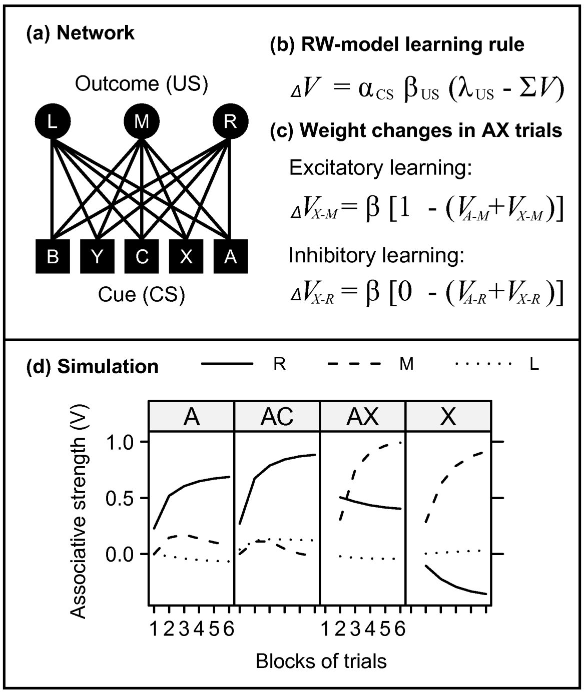

Figure 1. Least-mean-squares network based on the RW-model. **(a)** The network architecture consists of an input layer that represents stimulus elements completely interconnected to an output layer that represents the trained target positions. When a cue is presented, activity is propagated to the output layer via learned associations. **(b)** Learning rule used for updating the weights. **(c)** Example of concurrent changes in associations X-M and X-R in AX-trials **(d)** Predicted changes in associative strength with training schedule as depicted in Table 2.

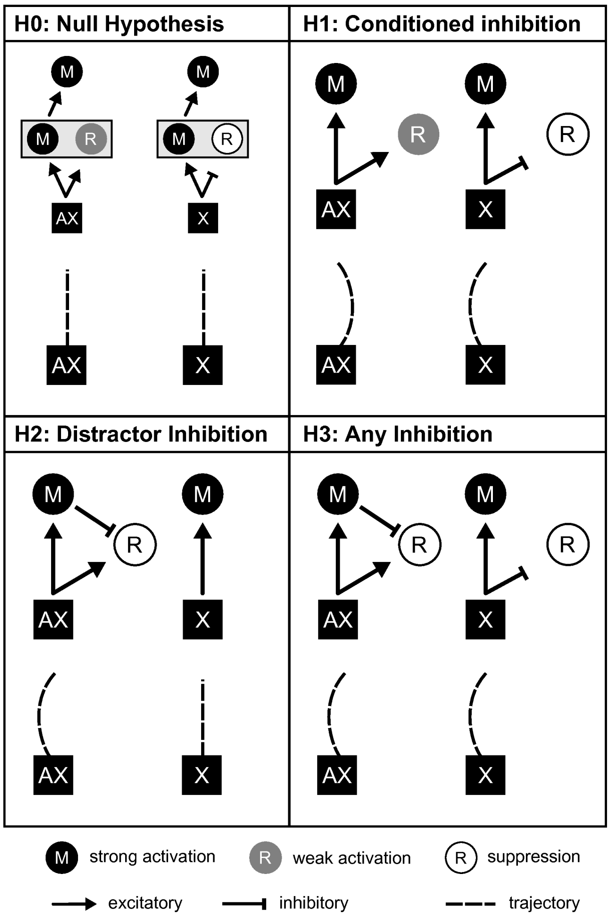

Figure 2. Different Hypotheses on how the acquired associative status of a cue might affect saccadic curvature. For further explanations see text.

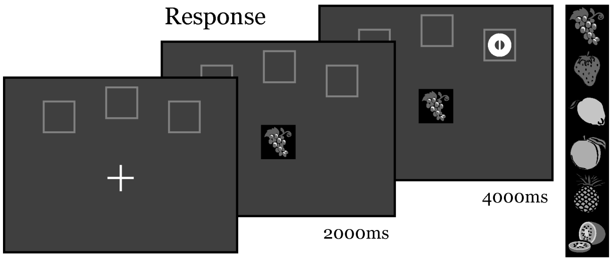

Figure 3. Example of a trial in the oculomotor learning experiment. The small picture of a fruit (cue; CS) was presented at fixation and was followed by the appearance of a target stimulus (outcome; US) at one out of three possible locations in the upper hemifield (right location in the depicted example). Different trials presented different single fruits or combination of fruits (depicted at the right side of the figure) and in the course of training participants learned which fruit predicted which target location. Saccades, elicited after cue onset that anticipated the correct target location were supposed to indicate the acquisition of cue-target associations in memory.

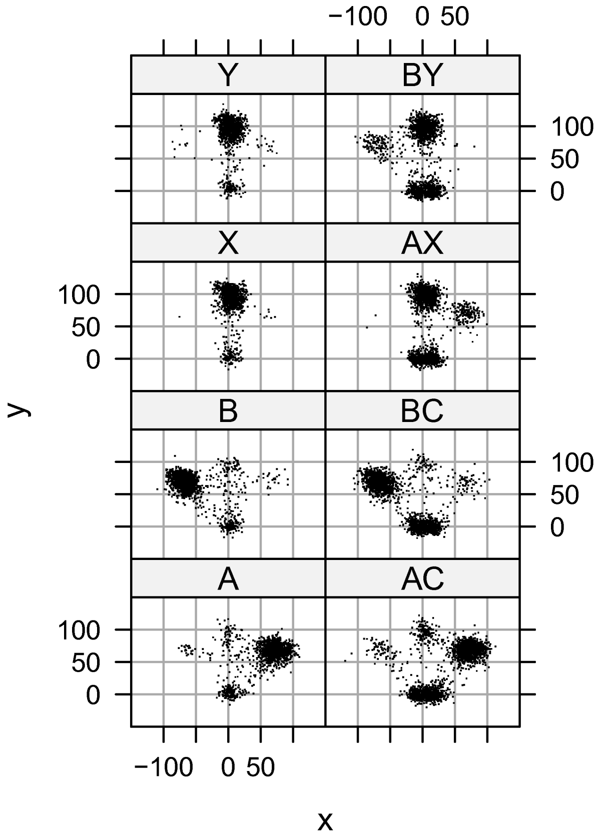

Figure 4. Endpoint scatter of saccades occurring in the cue interval.

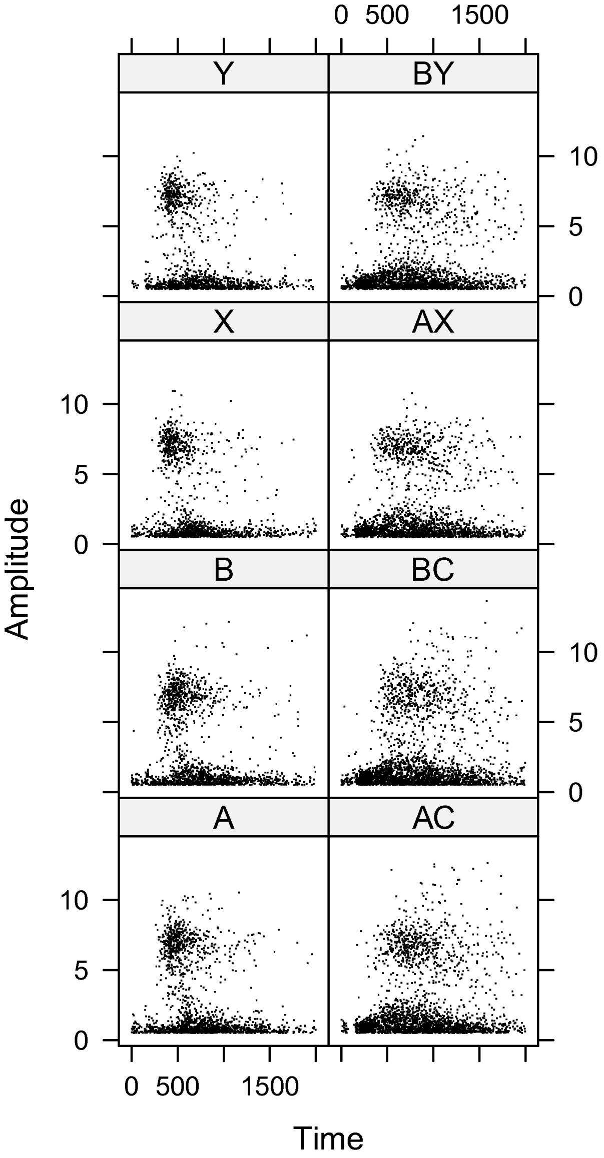

Figure 5. Evolution of oculomotor responses in the cue interval. Trial time zero denotes the onset of the central cue. Trial time 2000 denotes onset of the peripheral target stimulus.

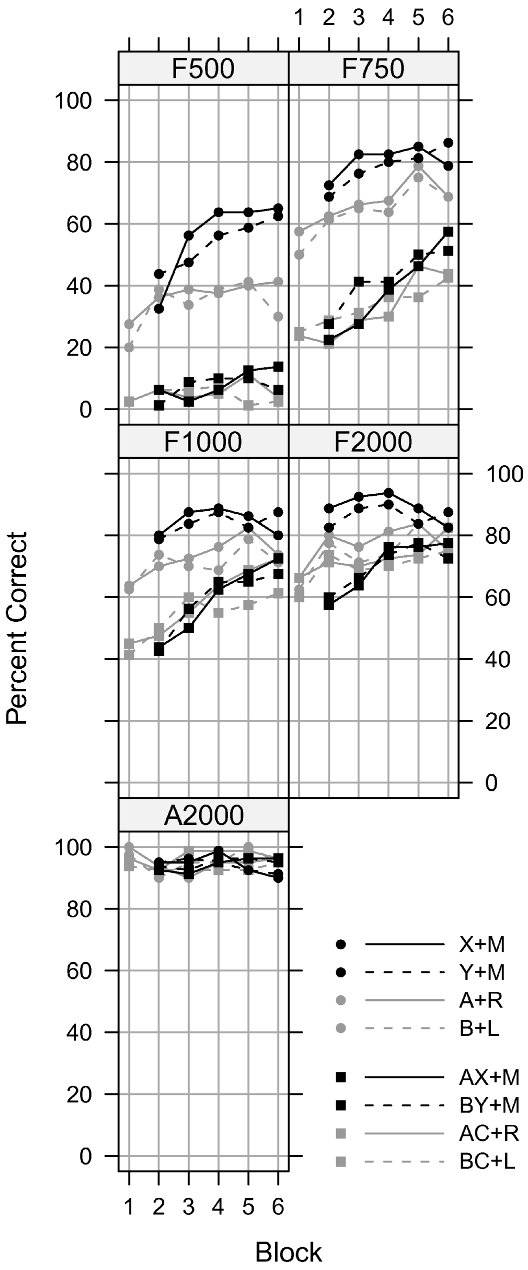

Figure 6. Frequency of correct anticipatory responses as a function of training. Panels F500 through F2000 correspond to the *first* correct saccade within 500 through 2000ms from cue onset. Panel A2000 refers to correct positions reached by the sequence of *all* saccades prior to target-onset.

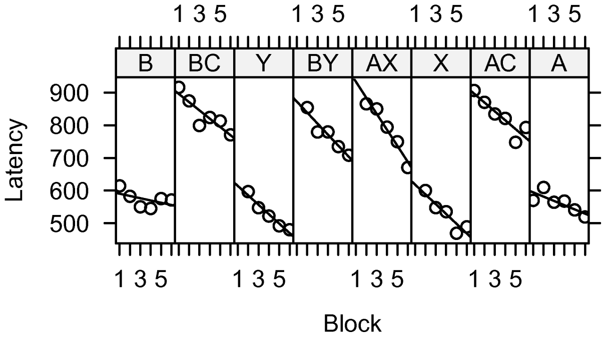

Figure 7. Decrease in latency of predictive saccades with training.

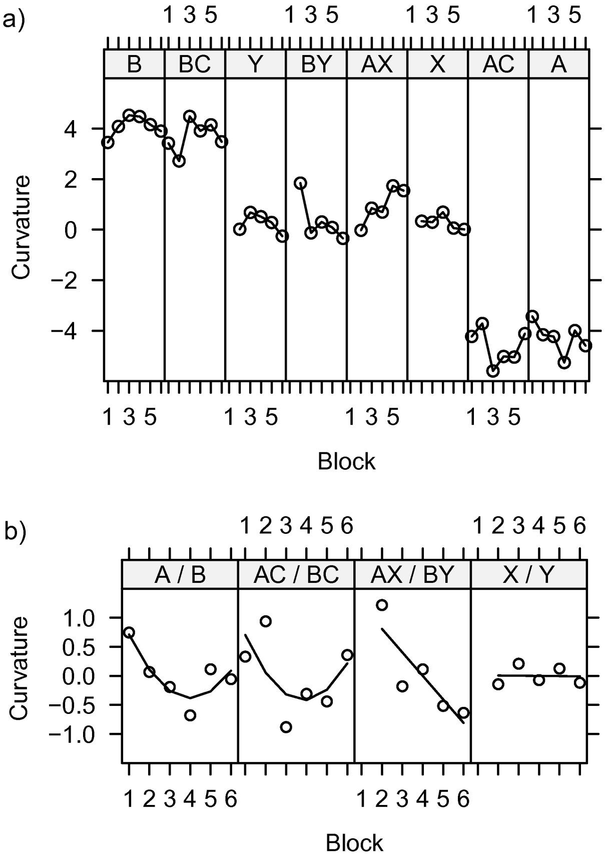

Figure 8. Changes in saccadic curvature with training. **(a)** Curvature of saccades elicited by trained cues. Saccades that curve to the left are represented by positive values, rightward-curvature is indicated by negative values. **(b)** Curvature averaged for equivalent trial types. Values are scaled to indicate curvature away from the mid position for trials A / B and AC / BC as well as curvature away from the respective lateral positions for trials AX / BY and X / Y. Values are centered within panel by subtracting their means. Solid lines indicate linear or quadratic trends.

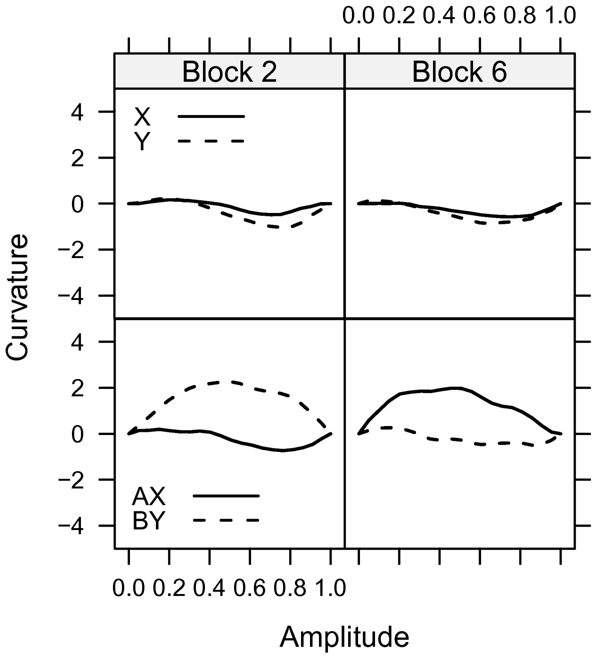

Figure 9. Average saccade trajectories in the first and last block of training for cues X, Y, AX and BY. The deviation (mm) of the saccade trajectory from a straight line is plotted against the fraction of saccade amplitude.

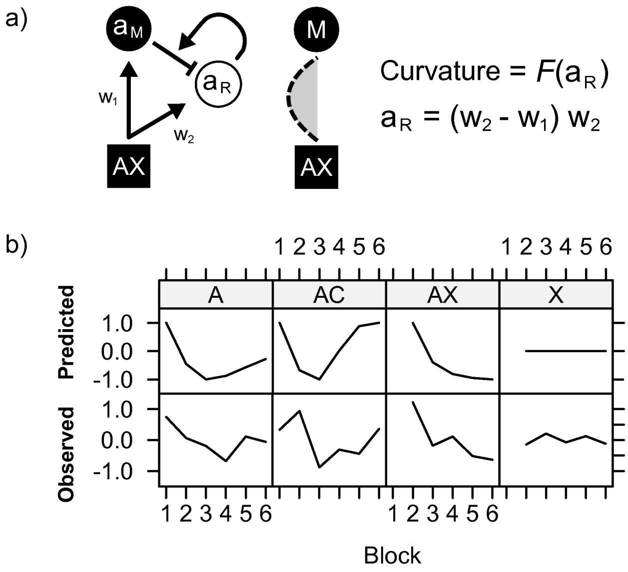

Figure 10. (a) Distractor suppression mechanism that computes the amount of inhibition applied to an interfering prediction. **(b)** Predicted and observed saccadic curvature. Observed values correspond to curvature shown in Figure 8b. Predicted curvature was calculated from associative interference as depicted in Figure 1d and scaled to the range of -1 / +1.

Tables

Table 1. Differential conditioning procedure.

			Target Position		
Stage	Block	Trial	L	M	R
1	1	1-16	B, BC	-	A, AC
2	2-6	17-176	B, BC	BY, Y, X, AX	A, AC

Note. The table depicts which cue (A, AX, X, etc.) is paired with which outcome (L, M, R). Each cue was presented twice per block. Trials were presented in random order restricting the occurrence of successive trials with the same target location to a maximum number of three. Each participant was presented with a different pseudo-random sequence.

Table 2. Analysis of Variance

Interval	Source	df_{Treat}	df_{Error}	F	η_p^2	p
F2000	Cue	7	119	7.637	.310	.001***
	Block	4	68	2.514	.129	.05*
F1000	Cue	7	119	12.998	.433	.001***
	Block	4	68	6.896	.289	.001***
F750	Cue	7	119	33.358	.662	.001***
	Block	4	68	11.199	.397	.001***
F500	Cue	7	119	45.043	.726	.001***
	Block	4	68	4.279	.201	.006**

Note. Results correspond to acquisition of correct anticipatory saccades as depicted in Figure 6.

Table 3. Contrast Analysis.

Cue	Contrast	df_{Treat}	df_{Error}	F	η_p^2	p
A / B	linear	1	17	3.237	.161	.088
	quadratic			5.943	.259	.026*
AC / BC	linear	1	17	< 1	-	-
	quadratic			13.913	.450	.002**
AX / BY	linear	1	17	9.453	.357	.007**
	quadratic			3.175	.157	.093
X / Y	linear	1	17	< 1	-	-
	quadratic			< 1	-	-

Note. Results correspond to changes in saccadic curvature as depicted in Figure 8b.

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Professional Experience

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Related Experience

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Publications

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Koenig, S., & Lachnit, H. (2010b). Revisiting the Memory-Based Saccadic Curvature Effect. Manuscript submitted for publication.

Koenig, S., & Lachnit, H. (2010c). Curved Saccade Trajectories Reveal Conflicting Predictions in Associative Learning. Manuscript submitted for publication.

Lachnit, H., Schultheis, H., König, S., Uengoer, M., & Melchers, K. G. (2008). Comparing Elemental and Configural Associative Theories in Human Causal Learning: A Case for Attention *Journal of Experimental Psychology: Animal Behavior Processes*, *34*, 303–313.

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Reinhard, G., König, S., & Lachnit, H. (2005). Der Stimulus-Probability-Effekt bei der Pupille: Eine Untersuchung der Stimulussequenz. In K. Lange, K. Bäuml, M. Greenlee, M. Hammerl, & A. Zimmer (Eds.). *Beiträge zur 47. Tagung experimentell arbeitender Psychologen*. Lengerich: Pabst Science Publishers.

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Erklärung:

Ich versichere, dass ich meine Dissertation

“Modulation of Saccadic Curvature by Spatial Memory and Associative Learning”

selbstständig und ohne unerlaubte Hilfe angefertigt und mich dabei keiner anderen als der von mir ausdrücklich bezeichneten Quellen und Hilfen bedient habe.

Diese Dissertation wurde in der jetzigen oder einer ähnlichen Form noch bei keiner anderen Hochschule eingereicht und hat noch keinen sonstigen Prüfungszwecken gedient.

Marburg, 20.09. 2010

(Ort/Datum) Unterschrift mit Vor- und Zuname