Post-Saccadic Error Processing in Sensorimotor and Visual Recalibration

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Summary

Saccadic eye movements bring stimuli of interest onto the fovea to improve the perception of the environment. However, imprecise saccades lead to post-saccadic errors, defined as the difference between target and saccade landing position. These errors provide crucial spatial information which the sensorimotor system uses to refine movement execution and sustain perceptual stability. In saccadic adaptation paradigms, artificially induced post-saccadic errors elicit saccade amplitude adjustments to compensate for persistent discrepancies. On a single trial level, serial dependence reflects how current perception is shaped by previously perceived sensory input, e.g., by post-saccadic errors. In the present dissertation, post-saccadic errors were systematically manipulated in three behavioral eyetracking studies to examine their contributions to both saccade-to-saccade and saccade-to-vision recalibration processes.

Study 1 assessed whether motor and visual recalibration were better explained by the shared resource model, assuming common neural structures for processing action and perception, or the active recalibration model, assuming independent yet interacting neural structures. To test these contrasting models, saccadic adaptation was induced, followed by “no error” trials, in which either the retinal error, defined as the difference between saccade landing position and visual target, or the prediction error, defined as the difference between predicted and actual saccadic landing position, was set to zero. These “no error” trials were intermixed with visual localization trials. Although saccadic adaptation remained robust, visual mislocalization decreased as a function of the number of trials with the retinal error set to zero and was entirely abolished with the prediction error set to zero. These findings suggest that motor and visual recalibration operate independently yet interactively, supporting the active recalibration model.

Study 2 assessed whether recalibration occurred through trial-by-trial integration of the post-saccadic error by assessing serial dependence under target uncertainty. Participants executed saccades towards Gaussian blobs with manipulated visuospatial uncertainty and target contrast. Reductions in saccade amplitude were observed only when the current target was uncertain, the preceding target was certain and contrast was held constant. These results imply that pre-saccadic target features (e.g., size and contrast) modulate the post-saccadic error influence on subsequent saccade amplitudes.

Study 3 assessed whether endogenous or exogenous attention shifts, elicited by voluntary or delayed saccades, respectively, produced distinct patterns in sensorimotor and visual serial dependence. Sensorimotor serial dependence, assessed via saccate-to-saccade amplitude adjustments, was more pronounced following voluntary saccades whereas visual serial dependence, assessed via an orientation judgment task, did not vary as a function of saccade type. This dissociation underscores the divergent impact of attentional mechanisms on sensorimotor but not visual serial dependence.

To conclude, the results support the active recalibration model over the shared resource model, shown by distinct saccade-to-saccade and saccade-to-vision recalibration patterns and provide novel insights into post-saccadic error integration in a serial-dependent way to stabilize visual perception and motor control.

Introduction

General saccade characteristics

In our daily life, we constantly gather sensory information to perceive our environment and successfully interact with it. The human field of view can cover 180° visual angles horizontally which can further be extended by head movements (Johnson et al., 2011). To successfully perceive our environment *saccadic eye movements* are conducted. Saccades are goal-oriented eye movements to bring objects of interest onto the *fovea*, the retinal area with the highest resolution. Humans perform about three saccades per second which are characterized by a short duration and high velocity with peak velocity values up to $700° s^{-1}$ (Mays, 2009). The sensorimotor system requires approximately 200 ms to initiate a saccadic movement, while saccadic duration is remarkably shorter with around 80 ms (Ilg & Thier, 2012; Purves et al., 2001). Research shows a positive correlation between amplitude size, peak velocity, and duration of a saccade, which is called the *main sequence*. This correlation implies that an increase in one parameter, by, for example, performing larger saccade amplitudes, inherently leads to changes in the other parameters, that is a larger peak velocity and longer duration, as they cannot be independently controlled (Ilg & Thier, 2012). Additionally, saccadic eye movements are affected by stimulus characteristics such as brightness, size, contrast, or direction (Goliskina et al., 2023).

Saccades are differently classified (Ilg & Thier, 2012). *Visual-guided saccades* are eye movements towards a target to project it onto the fovea, with latencies around 200 ms. *Express saccades* are eye movements characterized by significantly shorter latencies, typically ranging from 80 to 130 ms. Their induction requires the removal of the fixation target just before the presentation of a saccade target. This necessity indicates that the decision to disengage fixation contributes to increased saccadic latency. In the case of express saccades, however, this decision appears to have been pre-planned, allowing saccadic execution to occur with smaller delay. In *memory-guided saccades*, a peripheral target stimulus is briefly presented, requiring the observer to execute a saccade towards the target position after it has disappeared. To perform this task accurately, spatial parameters of the target position must be retained in short-term memory. Memory-guided saccades typically exhibit 10 to 20 % slower peak velocities compared to visual-guided saccades, reflecting the additional cognitive demand of recalling the target position from memory (Gnadt & Andersen, 1988). *Antisaccades* are eye movements executed in the opposite direction of a presented target and do not occur naturally in everyday situations but are created for experimental studies (Hallett, 1978). Compared to visual-guided saccades, they are associated with higher latencies and reduced accuracy as they require the inhibition of a reflexive saccadic response towards the target. Successful performance of antisaccades relies on cognitive control to suppress this automatic response and direct gaze to the opposing position.

Neural representation

The main brain areas of saccadic control are governed by a network of cortical and subcortical structures. The *superior colliculus*, which includes visual neurons in its superficial layers and oculomotor neurons in its deeper layers, is primarily responsible for initiating visual-guided saccades while lesions in this area result in increased saccadic latencies (Ilg & Thier, 2012). The *parietal cortex* is involved in visuospatial orientation (Husain & Nachev, 2007), saccade execution, and saccadic adaptation (Gerardin et al., 2012; Zhou et al., 2016). The *frontal eye field* contains visual, motor, and visuomotor neurons and projects to both the superior colliculus and oculomotor regions in the brainstem. The frontal eye field is crucial for the planning and execution of voluntary saccades (Müri & Nyffeler, 2008; Schraa-Tam et al., 2009) and memory-guided saccades (Schlag & Schlag-Rey, 1985), as well as for coding the saccadic goal (Russo & Bruce, 1994) and plays a significant role in visual attention (Hung et al., 2011). The *lateral intraparietal area* is involved in saccadic spatial planning and plays a crucial role in memory-guided saccades (Powell & Goldberg, 2000). Additionally, the *cerebellum* is a central brain region for saccadic movements. Electrophysiological studies (Gerardin et al., 2012; Guillaume et al., 2018; Métais et al., 2022) and positron emission tomography studies (Desmurget et al., 1998, 2000) provided evidence for a cerebellar contribution to saccadic adaptation. Further, the cerebellum is involved in eye movement accuracy (Peterburs et al., 2012), the execution of voluntary saccades (Robinson & Fuchs, 2001), and the detection and processing of post-saccadic errors (Herzfeld et al., 2018). Brain regions controlling eye movements significantly overlap with the *dorsal attention network* which codes the spatial target location and contributes to spatial attention (Ilg & Thier, 2012).

The forward model and the efference copy

The latency for corrective saccades is typically shorter than the latency of the initial saccades and than the time required for processing visual information (Ilg & Thier, 2012). This observation implies that saccadic trajectories are not directly guided by visual information perceived during or after the saccade. Consequently, saccadic eye movements are often described as ballistic: They are initiated based on an internal command, and, once underway, their course cannot be modified by real-time visual input (Ilg & Thier, 2012). However, while saccadic eye movements are not modifiable by external sensory feedback, they can be adjusted through an internally generated error signal if misalignments between the intended and actual saccadic landing position are detected (Blakemore et al., 2001; Miall & Wolpert, 1996; Wolpert & Kawato, 1998; Wolpert et al., 1998).

This internal error detection mechanism is conceptualized within the framework of the *forward model* (Wolpert & Flanagan, 2001). In the forward model, the consequences of motor commands are processed independently of sensory input. When a motor command is sent to the muscles, in the case of saccades those muscles control eye movements, a corresponding *efference copy* is generated simultaneously. This efference copy contains real-time information about the current position of the eye, which is compared to the intended target position. Based on this comparison, a prediction about the anticipated saccade landing position is generated which is then compared with the actual saccade landing position. If a discrepancy between the predicted and actual landing positions is detected, an error signal is generated to correct this landing position. As long as this discrepancy persists, excitatory burst neurons remain active, ensuring that the saccade is completed and that the predicted and actual landing positions align (Ohtsuka & Noda, 1991; Strassman et al., 1986).

The literature suggests that the cerebellum plays a crucial role in processing the efference copy and thereby in coordinating the correction of post-saccadic errors (Ethier et al., 2008; Tanaka et al., 2020; Wolpert et al., 1998). The forward model has been extensively studied across domains, such as tactile stimulation (Blakemore et al., 1999), sounds (Ghio et al., 2018), saccadic movement (Ethier et al., 2008), and even higher-level functions (Kilteni et al., 2020). Recent literature also contributed cognitive tasks to the functional role of the cerebellum (Berlijn et al., 2024; Lesage et al., 2012; Peterburs et al., 2019). Notably, the forward model does not describe a static system; it is adaptable and can optimize movement execution through learning processes (Tanaka et al., 2020). This adaptability becomes evident in saccadic adaptation, during which saccadic behavior is systematically altered in response to imposed errors, thereby facilitating ongoing optimization of movement.

The post-saccadic error and saccadic adaptation

Saccadic eye movements are essential for aligning the fovea with a target of interest. However, saccades typically undershoot the target, resulting in the need for a corrective saccade to complete the movement (Gillen et al., 2013; Ohl et al., 2011). This hypometric amplitude is anticipated by the visual system, leading to corrective saccades with shorter latencies compared to the initial saccades (Ohl et al., 2011). Research indicates that the superior colliculus plays a significant role in generating these corrective movements which are, therefore, no form of execution noise but rather an intended strategy of saccadic control of the visual system (Becker, 1989; Harris, 1995; Lisi et al., 2019). The discrepancy between target position and saccade landing position, known as the *post-saccadic error*, is assessed by comparing the perceived error to internal predictions (Bahcall & Kowler, 2000; Collins & Wallman, 2012; Pélisson et al., 2010; Wong & Shelhamer, 2010). If a mismatch is registered, the sensorimotor system initiates a corrective saccade to reduce this discrepancy (Sedaghat-Nejad & Shadmehr, 2021).

In laboratory settings, artificial post-saccadic errors can be induced to study *saccadic adaptation*, as first demonstrated by McLaughlin (1967). The manipulation, now widely employed, involves shifting the saccadic target mid-flight, thereby creating an unpredicted discrepancy between the target position and the saccadic landing position (for reviews, see Hopp & Fuchs, 2004; Pélisson et al., 2010; Zimmermann & Lappe, 2016). When participants detect this artificially induced error for the first time, they react with an unpredicted corrective saccade to bring the target onto their fovea. Over repeated trials, the oculomotor system gradually adapts by modifying saccadic amplitudes to minimize the artificially induced error (Abel et al., 1978; Bahcall & Kowler, 2000; Collins & Wallman, 2012; Cont & Zimmermann, 2021; Müri & Nyffeler, 2008; Noto & Robinson, 2001; Optican & Robinson, 1980; Pélisson et al., 2010; Pomè et al., 2023; Schraa-Tam et al., 2009; Wallman & Fuchs, 1998; Wong & Shelhamer, 2010; Zimmermann & Lappe, 2009, 2010). This oculomotor plasticity demonstrates that the sensorimotor system can continuously monitor and refine performance to maintain visual precision (Pélisson et al., 2010). The artificially induced target displacements are generally perceived subconsciously and are not noticed by the participants due to *saccadic suppression* (Bridgeman et al., 1975). Through saccadic suppression, perceptual influences during saccades are suppressed to stabilize the representation of the visual space and thereby prevent visual distortions caused by head- or eye movements (Burr et al., 1994; Gremmler & Lappe, 2017; Ilg & Hoffmann, 1993; Krekelberg, 2010; Pomè et al., 2024; Wurtz, 2018; Zimmermann, 2020). Saccadic suppression begins around 50 ms before saccade initiation, peaks around saccade onset, and persists for about 50 ms after the saccade is completed (Volkmann et al., 1978). Because information processing during saccadic execution is suppressed, visual feedback can only be processed after saccade landing. After several trials of saccadic adaptation, the system reaches an asymptotic state, establishing a new relation between saccadic amplitude and target position (Noto & Robinson, 2001; Wallman & Fuchs, 1998). At maximal adaptation, saccadic adaptation reduces approximately 75 % of the induced post-saccadic error (Gillen et al., 2013; Ohl et al., 2011). Adaptive modifications in saccadic amplitudes have been shown to be long-lasting, often persisting for several days after initial induction (Alahyane & Pélisson, 2005; Wang et al., 2012). Therefore, saccadic adaptation operates as a dynamic and adaptive process, enabling the sensorimotor system to correct post-saccadic errors and adjust behavior in response to changing environmental conditions.

During the mediation of saccadic adaptation, the sensorimotor system experiences two distinct error signals. The first error signal is the *retinal error*, which is defined as the difference between the saccade landing position and the visual target. It requires only the detection of deviation from the target rather than precise localization (Collins & Wallman, 2012; Havermann & Lappe, 2010; Wallman & Fuchs, 1998). However, since saccades are typically hypometric, adaptation may function to preserve a small retinal error and remain sensitive to this modest error signal (Havermann & Lappe, 2010; Robinson et al., 2003). The second error signal is the *prediction error*, which emerges from the difference between the predicted and actual saccadic landing position (Bahcall & Kowler, 2000; Chen-Harris et al., 2008) and originates most likely from the forward model (Miall & Wolpert, 1995; Munuera & Duhamel, 2020). Collins and Wallman (2012) directly compared the effects of retinal error and prediction error signals on saccadic adaptation, concluding that while both error types induce saccadic amplitude adaptation the prediction error exerts a greater influence than the retinal error. These findings support the notion that the oculomotor system relies on an internal prediction for adaptation. In Study 1 of this dissertation, this mechanism was further investigated by contrasting conditions in which either the retinal error or the prediction error were selectively set to zero, and by assessing their respective effects on saccadic metrics and visual perception.

Differences in adaptation dynamics imply that distinct mechanisms are at play (Ethier et al., 2008; Hernandez et al., 2008; Panouillères et al., 2009). *Inward adaptation* refers to the gradual decrease in saccadic amplitude observed over repeated trials when the target is displaced towards the initial fixation position during the saccade. It develops more rapidly, typically requiring between 30 and 60 trials to reach maximal oculomotor adaptation (Albano, 1996; Deubel & Schneider, 1996; Frens & Van Opstal, 1994; Watanabe et al., 2003). This process is efficient and effective in reducing post-saccadic errors. However, the faster adaptation rate may be influenced by factors such as muscle fatigue (Golla et al., 2008; Schnier & Lappe, 2011). In contrast, *outward adaptation* refers to the gradual increase in saccadic amplitude observed over repeated trials when the target is displaced away from the initial fixation position during the saccade. It develops more gradually, typically requiring 200 to 400 trials (Bahcall & Kowler, 2000; Deubel et al., 1986; Miller et al., 1981). It demands more effort, is less efficient, and less effective in minimizing post-saccadic error compared to inward adaptation (Ethier et al., 2008; Hernandez et al., 2008; Panouillères et al., 2012; Schnier & Lappe, 2011; Zimmermann & Lappe, 2010, 2016). As a result, outward adaptation often leads to longer perceived post-saccadic errors during the adaptation process (Havermann & Lappe, 2010).

Besides these two adaptation dynamics, saccadic adaptation occurs between saccades of similar amplitude (Collins et al., 2007; Frens & Van Opstal, 1994) and direction (Watanabe et al., 2003). At the same time, saccadic adaptation is independent of color and shape of the target (Collins et al., 2007; Deubel, 1995a; Frens & Van Opstal, 1994). In most studies, target displacement size and direction were kept constant between saccades (for reviews, see Pélisson et al., 2010; Zimmermann & Lappe, 2016). However, studies employing randomized displacement sizes and directions suggest that saccadic adaptation can also occur at the level of single saccades (Collins, 2014; Desmurget et al., 2000; Havermann & Lappe, 2010; Srimal et al., 2008). This finding is particularly relevant for Study 2 and Study 3 of this dissertation, in which post-saccadic errors were varied randomly in their size and direction to examine trial-by-trial influences of these post-saccadic errors.

Studies investigating the performance of visual-guided saccades typically use flashed target stimuli that elicit *reactive saccades*, also known as *delayed*, *reflexive*, or *externally triggered saccades* (Müri & Nyffeler, 2008; Schraa-Tam et al., 2009; Zimmermann & Lappe, 2009, 2010). However, compared to real-life scenarios, these briefly appearing targets create an artificial setting that does not fully represent natural scene perception during which individuals predominantly rely on *voluntary saccades* to explore a stable environment. Voluntary saccades are commonly described as *internally triggered saccades* (Alahyane et al., 2007; Collins & Doré-Mazars, 2006; Erkelens & Hulleman, 1993; Fujita et al., 2002; Walker & McSorley, 2006). In a laboratory setting, voluntary saccades have been tested by presenting the fixation point and target stimulus simultaneously. Participants are instructed to voluntarily perform a saccade, based on an internal “go”-signal. Voluntary saccades show a latency between 250 to 350 ms (Hallett & Adams, 1980). *Scanning saccades* form a variant of voluntary saccades as they occur when scanning a natural scene (Cotti et al., 2007; Deubel, 1995b). Studies investigating scanning saccades often present multiple targets simultaneously and participants fixate all stimuli based on an internal “go”-signal. The latency of scanning saccades ranges between 300 and 500 ms (Alahyane et al., 2007).

Previous research suggests a functional dissociation between voluntary and reactive saccades due to an asymmetric transfer of post-saccadic error information between these types of saccades (Alahyane et al., 2007; Collins & Doré-Mazars, 2006; Deubel, 1995b; Erkelens & Hulleman, 1993; Fujita et al., 2002). Zimmermann and Lappe (2009) examined the dissociation of reactive and scanning saccades with a typical saccadic adaptation paradigm. After adapting reactive and scanning saccades separately, they observed little transfer of the artificially induced post-saccadic errors from reactive to voluntary saccades. However, a more substantial transfer from adapted scanning saccades to reactive saccades was observed, while adaptation to the same type of saccade was high. While reactive saccades primarily activate parietal pathways that project to the superior colliculus and brainstem (Gaymard et al., 2003; Müri & Nyffeler, 2008; Pierrot-Deseilligny et al., 1991), voluntary saccades engage pathways in the frontal cortex leading to activations in the frontal eye field (Gerardin et al., 2012; Schraa-Tam et al., 2009) but also to the superior colliculus and brainstem (Cotti et al., 2007; Müri & Nyffeler, 2008; Rivaud et al., 1994). Thus, voluntary saccades also partially activate brain areas in the reactive pathway. In sum, the involvement of distinct neural pathways underscores the functional differentiation between reactive and voluntary saccades.

The link between the motor and the visual system

Successful interaction with the environment relies on the integration of sensory input into spatial representations that guide accurate motor responses. These representations are hypothesized to take the form of visual and motor maps, which encode spatial parameters essential for linking perception to action (for a review, see Zimmermann & Lappe, 2016). By transforming sensory signals into actionable motor commands, these maps enable precise and coordinated behavior. While the processing of motor commands is well understood, the relationship between visual and motor maps, as well as their role in bridging perception and action, remains ambiguous. Two underlying models regarding this relationship can be contrasted: the model of shared or of independent resources. The *shared resource model* of action and perception may optimize computational efficiency by minimizing the risk of misalignment between the two systems. Existing research highlights a strong link between perception and action (Hommel et al., 2001; James, 1890; Parr & Friston, 2017; Prinz, 1990). The *common coding theory* (Prinz, 1990) posits a shared representational framework for perception and action that facilitates efficient alignment of sensory and motor functions. Similarly, the *predictive coding theory* (Parr & Friston, 2017) suggests that spatial localization is intrinsically linked to motor interaction, such as directing a saccade to a target position. Conversely, independent processing of the motor and the visual space would allow for behavioral dissociation between these systems as they may be represented in distinct brain areas connected through specific neural pathways. Previous research has provided evidence for the independent neural control of various types of saccades which differ in attentional demands and, therefore, in neural processing (Cotti et al., 2007; Gerardin et al., 2012; Müri & Nyffeler, 2008; Rivaud et al., 1994; Schraa-Tam et al., 2009). More precisely, the *active recalibration model* suggests that the systems, while processed independently, interact dynamically to update each other (Cont & Zimmermann, 2021). Theories supporting the assumption of the active recalibration model must address which aspects of sensorimotor processing are shared and how these connections facilitate the integration and coordination of perception and action.

The architecture of the brain provides further insights into the relationship between the visual and motor system. Visual information is organized via isometric mappings, preserving the topological relationships of external objects (Ilg & Thier, 2012). Such mappings are maintained throughout higher levels of visual processing, ensuring that spatial relationships remain intact. However, isometric mapping encodes only relative, not absolute, spatial positions. To determine absolute object positions, recalibration processes are required. Recalibration connects the spatial position of an object in the external space to its corresponding position within the internal map. This connection depends on movements, such as reaching for an object or saccadic eye movements, allowing the verification of spatial distances in the internal representation. The brain evaluates the success or the failure of the movements by processing discrepancies between the intended and the actual movements. These movement-induced errors play a critical role in recalibrating and refining internal and external maps, ensuring that the internal representation aligns with external reality.

Saccadic eye movements offer an ideal model for testing this link between motor and visual parameters. Each saccade aims to gather new visual input which is then used to localize upcoming saccadic targets. Due to the high saccadic frequency of about three saccades per second, saccadic movements provide constant and frequent information required to recalibrate motor and visual space. Motor maps that store spatial information gathered over saccadic parameters contain an accurate representation of saccade target positions, as evidenced by the precision of saccade landing positions (Kowler, 2011). Studies investigating the influence of the post-saccadic error offer valuable insights into recalibration processes between motor and visual maps: When the target is displaced during saccade execution, an artificial post-saccadic error arises, prompting recalibration of spatial perception within motor maps (Awater et al., 2005; Bahcall & Kowler, 1999; Bruno & Morrone, 2007; Collins et al., 2007; Cont & Zimmermann, 2021). This recalibration might extend to visual maps to maintain a coherent environmental perception. The recalibration of action and perception will serve as the primary research focus of Study 1 of this dissertation with the research question: Does the brain rely on the shared resource model, in which motor and visual parameters are processed together, or does it rely on the active recalibration model, in which spatial parameters are processed separately but interact dynamically?

Current research indicates a relationship between saccadic motor parameters and visual localization. Tasks investigating visual localization combined with saccadic adaptation as a motor manipulation are commonly employed to test for visual mislocalization (Awater et al., 2005; Bahcall & Kowler, 1999; Bruno & Morrone, 2007; Collins et al., 2007; Garaas & Pomplun, 2011; Georg & Lappe, 2009; Hernandez et al., 2008; Moidell & Bedell, 1988; Schnier et al., 2010; Zimmermann & Lappe, 2009, 2010). In such tasks, participants should localize a target flashed in their visual periphery. Prior saccadic adaptation, which adjusts motor parameters, usually leads to visual mislocalization in the same direction as the adapted saccades (Awater et al., 2005; Bahcall & Kowler, 1999; Bruno & Morrone, 2007; Collins et al., 2007). These findings suggest a shared coordinate system for motor and visual parameters which then rely on overlapping neural pathways, implying that due to the shared underlying resources, any change in motor parameters and therefore in motor maps should also lead to changes in the spatial information for visual maps and thus in visual localization. Conversely, if the active recalibration model holds true, opposite behavioral results should be observed: This model suggests that motor and visual parameters are processed separately but are constantly recalibrated through post-saccadic errors. Since there is no reason for distinct brain areas specialized in post-saccadic error processing, distinguishable patterns between motor and visual parameters should be measured.

Zimmermann and Lappe (2010) tested the shared resource hypothesis by conducting a saccadic adaptation experiment involving inward and outward adaptation. After saccadic adaptation induction, participants were tasked with localizing a target briefly presented in their visual periphery. If saccadic parameters and visual localization share a common source of information, the expected visual mislocalization should have occurred in the same direction of the adapted saccades. The authors revealed adaptive changes in saccadic amplitudes for both inward and outward adaptation but visual mislocalization aligned with saccadic adaptation only in the outward direction. Since outward adaptation develops gradually (Bahcall & Kowler, 2000; Deubel et al., 1986; Miller et al., 1981) the sensorimotor system is exposed to larger post-saccadic errors for a longer time. As post-saccadic errors are a central mechanism underlying saccadic adaptation, the extended and amplified error exposure during outward adaptation particularly enhances the recalibration of motor maps (Ethier et al., 2008; Hernandez et al., 2008; Panouillères et al., 2009; Schnier & Lappe, 2011, 2012; Semmlow et al., 1989; Straube & Deubel, 1995; Straube et al., 1997; Wallman & Fuchs, 1998; Zimmermann & Lappe, 2010). Therefore, only outward adaptation was induced in Study 1 of the present dissertation to capture a more isolated form of adaptation, minimizing potential confounds such as muscle fatigue and allowing for prolonged exposure to post-saccadic errors. Furthermore, Zimmermann and Lappe (2010) excluded efference copy effects as an explanation for visual mislocalization since no saccades and thus no efference copies were generated during the visual localization task. This finding is consistent with recent research demonstrating that, in visual localization trials conducted without intermixed saccadic trials, visual mislocalization shifts towards the fovea (Cont & Zimmermann, 2021). Therefore, Cont and Zimmermann (2021) proposed the active recalibration model in which external spatial information, conveyed by post-saccadic errors following each saccade, is used to recalibrate both motor and visual localization. Furthermore, the extent of separate recalibration for motor and visual space appears to be influenced by the magnitude of the post-saccadic error, supporting the notion of an active mechanism.

In addition to investigating the asymmetric adaptation transfer between reactive and scanning saccades, Zimmermann and Lappe (2009) explored these transfer effects on a visual localization task. In their study, they used two types of localization targets: flashed targets, which are more suited for reactive saccades, and stationary targets, which better align with scanning saccades. The results revealed that adaptation of both reactive and scanning saccades induced visual mislocalization of flashed targets. However, only the adaptation of scanning saccades resulted in the visual mislocalization of stationary targets. This dissociation further supports the hypothesis that distinct brain regions are involved in processing different types of saccades, even when performing visual perception tasks, aligning with the assumptions of the active recalibration model.

The neural mechanisms underlying post-saccadic error processing are still not fully understood but evidence suggests an important role for both the cerebellum and the *posterior parietal cortex*. The cerebellum has been implicated in the detection of post-saccadic errors and the computation of adaptive modifications in saccade amplitudes (Herzfeld et al., 2018). The posterior parietal cortex plays a role in post-saccadic error detection (for reviews, see Husain & Nachev, 2007; Robinson et al., 1978) and saccadic adaptation (Gerardin et al., 2012; Panouillères et al., 2014). An electrophysiological study in macaques showed that neurons in the posterior parietal cortex receive post-saccadic error signals from the cerebellum, emphasizing that these neurons code the post-saccadic eye position (Zhou et al., 2016). Additionally, lesions in the posterior parietal cortex have been associated with deficits in saccade monitoring resulting in deficits in remapping the visual space (Duhamel et al., 1992; Sapir et al., 2004). This dual representation of post-saccadic error processing in the cerebellum and the posterior parietal cortex suggests that the posterior parietal cortex, which integrates motor and visual signals, is a strong candidate for mediating recalibration processes, supporting the active recalibration model (Husain & Nachev, 2007).

Serial dependence

The perception of sensory features is influenced by recently perceived sensory inputs, a phenomenon known as *serial dependence*. Serial dependence stabilizes the visual experience, producing an attractive bias towards similar stimulus features encountered in the recent past. This mechanism reduces visuospatial uncertainty, creating a smooth and coherent experience of the environment (Cicchini et al., 2014, 2017, 2018, 2024; Fischer & Whitney, 2s014; Manassi & Whitney, 2024). Corbett et al. (2011) proposed that serial dependence helps the brain to maintain object continuity, allowing for a consistent perception without generating new representations, although serial dependence effects gradually decrease over time to permit perceptual updates. Typically, serial dependence effects are short-lived, primarily limited to the last three trials within experiments and lasting up to 15 seconds (Fischer & Whitney, 2014; Manassi et al., 2018; Murai & Whitney, 2021). Cicchini et al. (2014) quantified the serial dependence effect, showing that up to 15 % of the magnitude of a previous stimulus can influence responses in the current trial.

Fischer and Whitney (2014) introduced serial dependence as a distinct perceptual mechanism that biases current perception towards recent input. In their study, participants adjusted the orientation of a response bar to match a *Gabor patch* presented in their visual periphery. A Gabor patch is a visual stimulus that consists of alternating light and dark stripes that smoothly fade at the edges, allowing to precisely control its spatial features. The authors found a positive relationship between adjustment errors, defined as the difference between the perceived and the reproduced orientation, and the orientation for consecutive stimuli, proposing that perception operates through continuity fields, i.e., spatiotemporally tuned mechanisms that promote continuity over time. Cont and Zimmermann (2021) extended these findings to the domain of saccadic eye movements, exploring recalibration processes between motor and visual parameters in a serially dependent manner. In their study, participants alternated between trials involving solely saccades with target displacements and trials combining saccades with target displacements and visual localization. They found that both, current post-saccadic errors and visual mislocalization, were attracted in the direction of the previously perceived post-saccadic error. However, evidence for trial influences in the further past was only descriptive, potentially due to an alternating trial structure between saccadic and visual localization trials, which differs from typical serial dependence studies with uniform trial types.

Serial dependence has been observed across a wide range of perceptual tasks, from basic sensory features to more complex attributes: Early studies focused on visual orientation (Alais et al., 2017; Cicchini et al., 2017; Collins, 2019; Fischer & Whitney, 2014; Fritsche & De Lange, 2019; Fritsche et al., 2017; Murai & Whitney, 2021; Pascucci et al., 2019; Rafiei et al., 2021; Tanrikulu et al., 2023) and numerosity (Bliss et al., 2017; Cicchini et al., 2014; Corbett et al., 2011; Fornaciai & Park, 2018). Further research has documented serial dependence for other visual features such as color (Barbosa et al., 2020; Bays et al., 2009; Foster et al., 2017; Oberauer et al., 2017; van den Berg et al., 2012), shape (Collins, 2022; Manassi et al., 2019, 2021), head movements (Bayer & Zimmermann, 2023; Zimmermann, 2021), time estimates (Schlichting et al., 2023), visual stability (Manassi & Whitney, 2022), facial identity (Liberman et al., 2014; Taubert et al., 2016; Turbett et al., 2021), emotional expression (Liberman et al., 2018; Mei et al., 2019), attractiveness (Kim et al., 2019; Xia et al., 2016), and, most importantly for this dissertation, saccadic eye movements (Cont & Zimmermann, 2021). This broad occurrence across different domains suggests that serial dependence constitutes a fundamental mechanism in perceptual processing.

Serial dependence is modulated by various factors including attention, similarity, and spatial and temporal proximity of consecutive stimuli (Cicchini et al., 2018; Manassi et al., 2023). Cicchini et al. (2018) claimed that this dependence on prior optimizes processing speed and accuracy across different paradigms. However, several prerequisites must be met for serial dependence to occur. Otherwise, the perception would deliver false input if every object were completely biased by the recent sensory history. For instance, temporal and spatial proximity of stimuli is crucial (Fischer & Whitney, 2014; Manassi et al., 2018). Additionally, similarity between successive stimuli enhances the effect. If two stimuli presented in rapid succession are sufficiently alike, the latter stimulus is perceived as more similar to the first one (Barbosa et al., 2020; Burr & Cicchini, 2014; Gallagher & Benton, 2022). Conversely, when the difference between successive stimuli is too large, serial dependence does not occur (Burr & Cicchini, 2014; Cicchini et al., 2024; Fischer & Whitney, 2014).

Influence of target uncertainty on serial dependence

Another determinant of serial dependence is the judgments of physical characteristics, e.g., the *spatial frequency*. The spatial frequency of a stimulus is defined as the number of cycles of light and dark elements per degree of visual angle. Evidence indicates that lower spatial frequencies, which are associated with broader visual representations and increased *visuospatial uncertainty*, elicit stronger serial dependence than higher spatial frequencies that convey fine, gradual changes (Ceylan et al., 2021; Cicchini et al., 2018). Cicchini et al. (2018) induced uncertainty through spatial frequency manipulations in an orientation reproduction task. Their findings showed that cardinal Gabor stimuli, which are associated with high spatial frequency and thus reduced visuospatial uncertainty, elicited weaker serial dependence whereas oblique Gabor stimuli with low spatial frequency showed stronger serial dependence. This aligns with findings by Girshick et al. (2011) who showed that orientation judgments along the cardinal axis were more accurate whereas more uncertain orientations exhibited stronger serial dependence. Cicchini et al. (2018), thus, proposed that serial dependence is reduced when current stimuli are clearer and less uncertain. Recent studies indicate that serial dependence is modulated by the uncertainty of the current stimulus but less by the uncertainty of the preceding stimulus (Fritsche et al., 2020; Gallagher & Benton, 2022; van Bergen & Jehee, 2019). Gallagher and Benton (2022) employed a paradigm by Fritsche et al. (2017), in which uncertainty was manipulated using the spatial frequency of Gabor stimuli, finding a stronger bias towards prior stimuli under higher current stimulus noise. This suggests that higher uncertainty in the current stimulus intensifies serial dependence effects. In contrast, they found little evidence that the uncertainty of prior stimuli has an equivalent effect.

Only a few studies investigated how post-saccadic error information transfers between trials based on the visuospatial uncertainty of the target and how this might affect adaptive saccadic amplitude changes. Research by Souto et al. (2016) examined the influence of target uncertainty on saccadic adaptation by adjusting the spatial constant of a *Gaussian blob target*. A Gaussian blob is a symmetrical two-dimensional luminance distribution with smooth transitions between peak intensity and background and its *spatial constant* defines the spread of this distribution. A low spatial constant is associated with a precise and certain target, whereas a high spatial constant corresponds to a diffuse and uncertain target. The authors revealed only a weak correlation between target uncertainty and adaptation rate, suggesting that visual uncertainty may have a limited effect on saccadic adaptation. A complementary study by Heins et al. (2023) demonstrated that saccadic adaptation can occur without a visible pre-saccadic target, which they interpreted as a function of internal predictive mechanisms rather than the physical presence of a target. Their findings imply that an internal estimate of the target position may hold greater significance for adaptation than the actual physical presentation. Another study by Lisi et al. (2019) indicates that Gaussian blobs with larger visuospatial uncertainty led to a larger variance in saccadic landing positions and a bias towards the average target position. This suggests that under uncertainty the sensorimotor system relies on prior knowledge, putting less weight on post-saccadic errors when predictions are uncertain, potentially reducing serial dependence for the post-saccadic error. In Study 2 of the present dissertation, changes in saccadic amplitudes were investigated in a serial-dependent manner in response to post-saccadic errors, further examining how target uncertainty and target contrast, tested by using Gaussian blob stimuli, influence serial dependence. Specifically, it was tested whether recalibration follows integration processes in which the influence of the previous post-saccadic error on the current one would be modulated by these features of target appearance.

Higher-order factors affecting serial dependence

Despite the findings that serial dependence is influenced by the immediate sensory input, some contradictory findings raise the possibility of cognitive factors at play. Gallagher and Benton (2022) suggested that post-perceptual decision-making processes might modulate serial dependence as they found no effect of prior stimulus uncertainty on serial dependence but increased serial dependence with higher current stimulus uncertainty as shortly addressed previously. Similarly, Cicchini et al. (2018) noted that serial dependence is more strongly influenced by the quality of the current stimulus rather than the past one and depends on the decision in the previous trial.

Serial dependence occurs within both perceptual and decision processes (Cicchini et al., 2017) but distinguishing bottom-up from top-down influences is challenging due to their interconnection (Fritsche et al., 2017). Additionally, serial dependence appears to be influenced not only by bottom-up processes such as stimulus characteristics but also by top-down processes such as expectations, which bias perception based on past behavior (Abreo et al., 2023). Even though most reported serial dependencies exhibit attractive biases, i.e., the features of the current target are biased towards those of the previously perceived target, repulsive biases can also occur. In these cases, the perception of the current target is biased in the opposite direction to the perceptual features of the previous one (Abreo et al., 2023; Rafiei et al., 2021). Abreo et al. (2023) found that no target expectations resulted in attractive serial dependencies, whereas expected targets led to repulsive serial dependencies. Unexpected targets, however, exhibited a mix of both attractive and repulsive serial dependencies. Other factors modulating the strength of serial dependence are awareness (Kim et al., 2020), salience (Lisi et al., 2019), confidence (Abreo et al., 2023; Samaha et al., 2019), and stimulus reliability (Cicchini et al., 2018). These studies suggest that serial dependence may be a flexible, context-sensitive mechanism that prioritizes perceptual stability by adjusting to conditions of uncertainty, enhancing the accuracy of environmental representation.

Influence of attention on serial dependence

Attention also plays a critical role in visual serial dependence as well as in eye movement execution. Two distinct forms of attention, *endogenous* and *exogenous attention*, can be distinguished (Ilg & Thier, 2012). Endogenous, also known as *voluntary attention*, is a goal-directed, top-down process that is oriented towards a target of interest. It requires a longer buildup time (~300 ms) and is typically associated with voluntary saccades (Godijn & Pratt, 2002). In contrast, exogenous, also known as *reflexive attention*, is a bottom-up process triggered by sudden, salient stimuli with a faster buildup (~100 to 120 ms) and a rapid decay. This form of attention is linked to reactive saccades and induces a shift of attention towards the triggering stimuli (Carrasco & Barbot, 2014). Both forms of attention enhance the processing of visual contrast and spatial resolution but their effects differ: Endogenous attention can enhance perception simultaneously at both peripheral and central retinal locations while exogenous attention improves spatial resolution in the visual periphery at the expense of central information (Barbot & Carrasco, 2017; Yeshurun & Carrasco, 1998). Research identified the dorsal attention network associated with spatial attention (Ilg & Thier, 2012), with ventral frontoparietal regions active during tasks in which attention is required. The frontal eye field is connected to visual attention and plays a role in endogenous attention. The superior colliculus, which plays a role in saccadic eye movements, is connected to this attention network (Ilg & Thier, 2012). The *ventral attention network* is strongly connected to the dorsal attention network but is involved in more general attentional processes rather than mediating spatial attention (Ilg & Thier, 2012).

Attention can also be classified based on its deployment mechanism. *Overt attention* involves an observable orientation towards a target, such as through eye movements, while *covert attention* allows focus on a target without corresponding observable behavior, for instance, focusing on one object while directing attention to another (Fernández et al., 2022; Van Der Stigchel & Theeuwes, 2007). While research has focused on covert attention shifts (e.g., Fernández et al., 2022), differences between endogenous and exogenous attention are also evident in overt attention shifts. For example, when a sudden visual target occurs, a reactive saccade is often performed automatically towards this target. Conversely, voluntary saccades involve internally driven decisions to perform eye movements. Notably, attention mandatorily shifts to the saccadic target position just before a saccade is executed (Deubel & Schneider, 1996; Hoffman & Subramaniam, 1995; Kowler et al., 1995; Shepherd et al., 1986; Van Der Stigchel & Theeuwes, 2007). Overt and covert attention shifts are therefore thought to be closely linked (Awh et al., 2006; Corbetta et al., 1998; Smith & Schenk, 2012). A recent study examined the effects of exogenous and endogenous covert attention shifts on sensory tuning for orientation (Fernández et al., 2022). Both endogenous and exogenous attention modulate sensory tuning through gain enhancement, with exogenous attention producing a stronger orientation gain enhancement.

Studies have shown that serial dependence occurred only if the preceding stimulus was attended to (Bae & Luck, 2020; Fischer & Whitney, 2014; Fornaciai & Park, 2018; Liberman et al., 2016; Rafiei et al., 2021). For instance, Fischer and Whitney (2014) demonstrated that serial dependence emerged only when the cue validity for selecting a target and therefore attention to this stimulus was fully provided. Similarly, Bae and Luck (2020) found that serial dependence was observed for motion direction only when participants directed attention to that specific feature; no serial dependence occurred when attention was directed to a different feature (e.g., color) of the same stimulus. In a meta-analysis, Manassi et al. (2023) found that devoting fewer attentional resources to the previous stimulus results in reduced serial dependence. However, some studies reported no effects of attention on serial dependence (Fornaciai & Park, 2018; Goettker & Stewart, 2022). Rafiei et al. (2021) conducted a visual search task combined with an orientation judgment task and found attractive serial dependence for attended targets while actively ignored targets led to a repulsive serial dependence. The authors conclude that both biases simultaneously reduced noise to keep a continuous perception. Fritsche and De Lange (2019) claim that the direction of serial dependence changes depends on the attended features of the previous stimulus. More precisely, an attractive bias was found with similar oriented targets from trial to trial while a repulsive bias was found with different orientations. Additionally, the strength of an attractive bias was reduced when participants attended to the stimulus size, while the repulsive bias remained unaffected. The authors claimed separate attentional sources for attractive and repulsive serial dependence. The aim of Study 3 of the present dissertation was to investigate how different types of saccades, voluntary and reactive saccades, hereafter delayed saccades, affect saccadic amplitude changes in a serial-dependent way, given that these types of saccades are associated with distinct attentional mechanisms: endogenous for voluntary and exogenous for reactive saccades. Specifically, it was examined whether recalibration processes between the previous and the current post-saccadic error differ between these distinct types of saccades.

Overview of studies

In this dissertation, three studies were conducted in which post-saccadic errors were artificially induced to gain insights into the mechanisms underlying error processing and the transfer of spatial information across saccades. In Study 1, either the retinal error or the prediction error was annulled to test whether the shared resource model or the active recalibration model best described the interplay between action and perception. In Study 2, trial-by-trial transfer of post-saccadic error information was investigated using Gaussian blob targets with manipulated spatial uncertainty and target contrast. In Study 3, sensorimotor and visual serial dependence were examined for voluntary versus delayed saccades, which differed in their associated attentional shifts (endogenous versus exogenous, respectively). All experiments were in accordance with the Declaration of Helsinki and were approved by the local ethics committee of the Faculty of Mathematics and Natural Sciences of Heinrich Heine University Düsseldorf. Participants were recruited at the Heinrich Heine University Düsseldorf or via social networks. Written informed consent was obtained prior to their voluntary participation. In all three studies, eye movements were recorded using a desktop-mounted EyeLink 1000 Plus eyetracker operating at a sampling rate of 1000 Hz. Participants performed the task binocularly, with data recorded from the left eye. A standard nine-point calibration was used for each participant before the start of the experiment. All studies were conducted in a completely dark room to prevent visual landmarks. Participants were seated 57 cm away from the monitor with their heads stabilized in a chin rest. In the following, an overview of the three studies is presented. Full details are provided in the original research articles and manuscripts, which can be found in the Appendix.

Study 1

Research question and hypotheses

A fundamental question in vision science is how an accurate visual representation of the external space is maintained. Post-saccadic errors provide crucial information for recalibrating both motor and visual spatial parameters. According to the shared resource model, action and perception rely on a common processing mechanism, leading to parallel adjustments in motor and visual representations (Awater et al., 2005; Bahcall & Kowler, 1999; Bruno & Morrone, 2007; Collins et al., 2007). In contrast, the active recalibration model proposes that these processes operate independently but are continuously updated (Cont & Zimmermann, 2021; Cotti et al., 2007; Gerardin et al., 2012; Müri & Nyffeler, 2008; Rivaud et al., 1994; Schraa-Tam et al., 2009). To test these theoretical frameworks, Study 1 employed a saccadic adaptation paradigm in which artificial post-saccadic errors were induced to examine their effects on visual localization.

Following an adaptation phase, “no error” trials were introduced by predicting saccadic landing positions and eliminating post-saccadic errors. Two variations of “no error” trials were used, one in which retinal errors were eliminated (hereafter “no retinal error” trials) and another in which prediction errors were eliminated (hereafter “no prediction error” trials). Additionally, the number of trials in which the sensorimotor system received “no error” information was varied to assess its influence on visual mislocalization. Using these error variations allows conclusions regarding the two models: A concomitant shift in saccadic parameters and visual localization would support the shared resource model, whereas divergent shifts in motor and visual parameters would indicate independent recalibration processes, in line with the active recalibration model.

Method

Study 1 involved a total of 17 participants across three experiments. Nine participants (mean age = 25.78 years, *SD* = 4.79 years; 5 females, 4 males) took part in the “no retinal error” experiment. Four of these participants, along with five additional participants (mean age = 22.43 years, *SD* = 5.13 years; 6 females, 3 males), took part in the “no prediction error” experiment. Four participants from the previous experiments and three new participants (mean age = 23.89 years, *SD* = 6.83 years; 5 females, 2 males) took part in the “constant error” experiment. Stimuli were presented by a 2014 Mac Mini computer on a 12.9 inch CRT monitor with a resolution of 800 × 600 pixels and a refresh rate of 120 Hz. Stimuli were generated using MATLAB R2016b (v. 7.10.0; The MathWorks, Natick, MA, United States) and PsychToolbox routines (v. 3.0.17; Kleiner et al., 2007). Before starting the experiment, participants underwent a three-minute dark adaptation period.

In all three experiments, each trial began with the presentation of a fixation square, displayed 6.5° to the left of the screen center. Trials were categorized either as saccade trials, indicated by a red fixation square, or as localization trials, indicated by a blue fixation square. In the saccade trials participants should perform a saccadic eye movement towards a suddenly appearing target 6.5° on the right of the screen center. In the visual localization task, participants perceived a briefly flashed target with the same physical characteristics as the target used in the saccade trials and should indicate its perceived position via mouse click. They were instructed to permanently fixate on the left blue fixation square to prevent influences from saccadic movements in the visual localization task. The experimental procedure followed a block structure: Each block consisted of a set of saccade trials, followed by a set of localization trials. In the *baseline* block, 20 saccade trials and 20 localization trials were conducted to establish baseline saccadic and visual localization performance. The *post-adaptation* block involved 100 saccadic adaptation trials. The saccade velocity was calculated online and as soon as it was bigger than 30° s^{-1} in five consecutive samples the saccade target was displaced 3° outward to induce an artificial post-saccadic error. Adaptation effects on the visual localization were measured in the following 20 localization trials. This block was followed by two “*no error*” blocks in which saccade trials without induced post-saccadic error alternated with 20 visual localization trials. The number of saccadic trials varied across three conditions: short (12 trials), medium (25 trials) and long (50 trials). Each condition concluded with a *de-adaptation* block of 20 trials to cancel out any saccadic adaptation effect. Each participant underwent all three conditions. This resulted in 244 total trials in the short condition, 270 total trials in the medium condition, and 320 total trials in the long condition. Each condition lasted approximately 20 minutes, with a minimum 15-minute break between conditions to prevent adaptation transfer across conditions.

In the three experiments, the post-saccadic error was systematically set to zero in the "no error" trials, a method devised by Robinson et al. (2003). In the "no retinal error" experiment, the saccade landing position was predicted online (defined as saccade velocity slower than 30° s^{-1} in three consecutive samples) and the target was presented at this predicted position to ensure no retinal post-saccadic error was experienced. In the "no prediction error" experiment, the mean retinal error in the individual baseline trials of each participant, that is the difference between the saccade landing position and the target position, was calculated and added to the predicted landing position in the "no error" trials. In the "constant error" experiment, the target was displaced 3° beyond the predicted saccade landing position, resulting in a consistent outward error which led to ongoing saccadic adaptation. For this last experiment, only the long condition (320 trials) was conducted.

Data from saccades with blinks or anticipatory eye movements and outliers (defined as saccade landing positions smaller than 3.5° or larger than 9.5°) were excluded from the analyses. Data from localization trials were excluded if fixation was not maintained during the task or if the reported localization was smaller than 3.5° or larger than 9.5°. Participants were included in the analyses if at least 60 % of their trials were valid. For the mean saccadic adaptation effect in each block, the difference between the mean of the last ten valid trials in each saccadic block and the saccadic baseline was computed. For the mean localization adaptation effect in each block, the difference between the mean localization of each localization block and the localization baseline was computed. For both, the mean saccadic adaptation effect and the mean localization adaptation effect, positive values indicate an outward shift, whereas values close to zero indicate no adaptation effect. For statistical analyses, non-parametric repeated-measures ANOVAs were conducted, using aligned rank transformations for factorial data. For the "no retinal error" and the "no prediction error" experiment, separate analyses were conducted. To test adaptation effects in saccade landing positions or visual localization, a 2 × 3 non-parametric repeated-measures ANOVA with factors *block* (baseline, manipulation) and *condition* (short, medium, long) was performed. Additionally, a 2 × 3 non-parametric repeated-measures ANOVA with factors *task* (saccade, localization) and *condition* (short, medium, long) was performed to compare saccade and localization adaptation effects. Finally, for the "constant error" experiment, Bonferroni-corrected one-sample *t*-tests against zero were performed on mean saccade and localization adaptation effects.

Results and discussion

The processing of the saccadic and visual space was examined to determine whether it relies on shared or independent resources by introducing "no error" trials and analyzing their effect on saccadic adaptation and visual mislocalization in three experiments ("no retinal error", "no prediction error", and "constant error") and three conditions (short: 12 trials; medium: 25 trials; and long: 50 trials). In the *post-adaptation* block, saccadic adaptation was induced by using a typical intra-saccadic target displacement paradigm followed by visual localization trials. Note that during the *post-adaptation* block, the number of trials did not vary across conditions; the different trial lengths were introduced with the first *"no error"* block.

Replicating previous literature, saccadic landing positions consistently shifted outward in all experiments and conditions, indicating robust saccadic adaptation (Bahcall & Kowler, 2000; Collins & Wallman, 2012; Noto & Robinson, 2001; Pélisson et al., 2010; Pomè et al., 2023; Wallman & Fuchs, 1998; Wong & Shelhamer, 2010; Zimmermann & Lappe, 2009, 2010). Similarly, visual localization shifted outward, aligning with the adapted saccadic landing. These findings support the shared resource model, according to which the motor and visual system rely on common spatial coordinates (Awater et al., 2005; Cheviet et al., 2022; Garaas & Pomplun, 2011; Moidell & Bedell, 1988; Zimmermann & Lappe, 2009, 2010). Study 1 thus joins a series of previous studies that corroborate that saccadic adaptation alters spatial perception, reinforcing the role of the motor system in constructing visual space.

To further test how the adapted state responds to the absence of post-saccadic error signals, "no error" trials ("no retinal error", "no prediction error", and "constant error" condition) were mimicked directly after the *post-adaptation* block. In all experiments of Study 1, saccades maintained their adapted state across short (12 trials), medium (25 trials), and long (50 trials) trial periods, even without post-saccadic error information. Interestingly, while saccadic adaptation persisted, visual localization effects varied across experiments, that is, across error types. In the "no retinal error" experiment, visual mislocalization effects diminished only after a long exposure phase with no retinal error information (50 trials) with localization shifting foveal. This aligns with previous findings which suggest that recalibration depends on the duration without post-saccadic information transfer (Cont & Zimmermann, 2021). By contrast, in the "no prediction error" experiment, visual localization rapidly decayed already after a short exposure phase with "no error" trials (12 trials), indicating a pattern that diverged from saccadic adaptation in all conditions. In the "constant error" experiment, visual mislocalization remained strongly aligned with saccadic adaptation due to the consistent outward post-saccadic error signal. In contrast, after a second block of "no error" trials, mixed results were observed with both saccade and localization adaptation effects showing decay. This suggests that prolonged exposure to "no error" information gradually disrupted both motor and visual recalibration. This dissociation between saccades remaining adapted and visual localization shifting towards the fovea when experiencing no post-saccadic error information challenges the shared resource model and instead supports a framework proposing separate resources for visual and motor space. Importantly, these findings, however, do not align with models positing completely independent resources for motor and visual space, either, as they point to a more nuanced interaction between the two systems.

Comparing the effects of the "no retinal error" and the "no prediction error" condition, stronger recalibration between motor and visual parameters occurred in the "no retinal error" condition. Retinal error annulment appeared to be more potent in driving changes in visual perception, as its effects persisted across longer trial durations (up to 50 trials), unlike the prediction error, which decayed more rapidly. Thus, while both error types effectively induced saccadic adaptation, their impact on visual localization diverged.

Conclusion

In conclusion, the data provide evidence in support of the active recalibration model. The post-saccadic error, induced by saccadic adaptation, recalibrated both the metrics of saccadic eye movements and visual localization, aligning with the shared resource model. However, dissociations between these processes were observed: While saccadic adaptation remained unaffected by “no error” trials, visual localization reverted to baseline levels depending on error type and number of trials. This finding highlights the existence of connected yet independently functioning systems of motor and visual recalibration: the active recalibration model.

Study 2

Research question and hypotheses

An increase in visuospatial uncertainty has been shown to amplify serial dependence (Ceylan et al., 2021; Cicchini et al., 2018). Serial dependence is particularly pronounced when the current stimulus is highly uncertain, while the uncertainty of the preceding stimulus plays a comparatively smaller role in the modulation of serial dependence (Fritsche et al., 2020; Gallagher & Benton, 2022; van Bergen & Jehee, 2019). This asymmetry in serial dependence can be investigated via post-saccadic error influences from trial to trial. In Study 2, Gaussian blobs were used as pre- and post-saccadic targets, with their spatial constant varied to manipulate target visibility and thereby inducing different levels of visuospatial uncertainty. If target visibility primarily affects perceptual localization, serial dependence should be stronger when the pre-saccadic target has a high spatial constant, as such targets are more challenging to fixate accurately. In this scenario, the visual system would rely more heavily on prior stimuli to estimate the position of the target. Conversely, if target visibility influences motor targeting, serial dependence should decrease when the post-saccadic target has a high spatial constant as reduced visibility of the post-saccadic target would impair amplitude adjustments for subsequent saccades.

Method

Study 2 involved a total of 44 participants across two experiments with three conditions each. Twenty-two participants (mean age = 22 years, *SD* = 2.99 years; 14 females, 8 males) took part in the “constant contrast” experiment. Twenty-two new participants (mean age = 23.50 years, *SD* = 4.28 years; 17 females, 5 males) took part in the “adjusted contrast” experiment. Stimuli of the “constant contrast” experiment were presented by a 2014 Mac Mini computer on a 12.9 inch CRT monitor with a resolution of 800 × 600 pixels and a refresh rate of 120 Hz, generated using MATLAB R2016b (v. 7.10.0; The MathWorks, Natick, MA, United States) and PsychToolbox routines (v. 3.0.17; Kleiner et al., 2007). Stimuli of the “adjusted contrast” experiment were presented by a Windows 10 computer on a 23.6 inch Acer XB272 monitor with a resolution of 1920 × 1080 pixels and a refresh rate of 120 Hz, generated using MATLAB 2020b (v. 9.9.0; The MathWorks, Natick, MA, United States) and PsychToolbox routines (v. 3.0.18; Kleiner et al., 2007).

Each trial began with the presentation of a red fixation square displayed 6.5° to the left of the screen center. A two-dimensional Gaussian blob (pre-saccadic target; T1) was presented 6.5° to the right of the screen center. Participants were instructed to perform a saccade towards T1 as soon as it appeared. During saccade execution, defined as eye velocity bigger than $30° \, s^{-1}$ in five consecutive samples, T1 was displaced to one out of six different positions (−2.5°, −1.5°, −0.5°, 0.5°, 1.5°, 2.5°), becoming the post-saccadic target (T2). To manipulate spatial uncertainty, the Gaussian blob's spatial constant (σ) could either be low (σ = 0.3°), that is, more focused and associated with smaller visuospatial uncertainty, or high (σ = 1.5°), that is, broader and associated with larger visuospatial uncertainty. Each participant underwent three different conditions: (a) both T1 and T2 had a small spatial constant, (b) T1 had a small spatial constant and T2 had a large spatial constant and (c) T1 had a large spatial constant and T2 had a small spatial constant. Each condition consisted of 400 trials and lasted for approximately 20 minutes, with condition order randomized across participants.

Between the two experiments, the target contrast (peak luminance of the stimulus divided by total screen luminance) was varied to further modulate spatial uncertainty. In the "constant contrast" experiment, the same contrast level was applied to both spatial constants (∼27 %) in which targets with a higher spatial constant appeared more luminous. In the "adjusted contrast" experiment, targets with a lower spatial constant (σ = 0.3°) were paired with higher contrast (∼27 %), while those with a higher spatial constant (σ = 1.5°) were paired with lower contrast (∼3 %). This adjustment ensured that targets with a higher spatial constant appeared less luminous, thereby increasing target uncertainty.

Trials were excluded from analyses if no saccade was executed, if the saccadic amplitude was smaller than half the required distance of 6.5°, or if the peak velocity exceeded $800° \, s^{-1}$. On average, about 10 % of trials were excluded per participant. For both experiments, the post-saccadic error for each trial was computed as the difference between the saccadic landing position and the position of T1 (6.5°). Negative values indicate an undershoot of saccadic performance whereas values close to zero indicate perfect saccadic performance. The influence of the previous post-saccadic target ($T2_{n-1}$) on the current pre-saccadic target ($T1_n$) was analyzed offline. This analysis focused on three conditions: (a) $T2_{n-1}$ small/$T1_n$ small, (b) $T2_{n-1}$ large/$T1_n$ small, and (c) $T2_{n-1}$ small/$T1_n$ large. Serial dependence effects were assessed via the relationship between the post-saccadic error influence in the previous and the current trial with linear regression analyses. Bonferroni-corrected one-sample *t*-tests against zero were performed to test significant serial dependence effects. Additionally, a one-way ANOVA with the three-level within-subjects factor *condition* ($T2_{n-1}$ small/$T1_n$ small, $T2_{n-1}$ large/$T1_n$ small, $T2_{n-1}$ small/$T1_n$ large) was performed for both experiments to assess differences in the serial dependence strength. Last, a 2 × 3 repeated-measures ANOVA with the between-subjects factor *experiment* (constant contrast, adjusted contrast) and the within-subjects factor *condition* ($T2_{n-1}$ small/$T1_n$ small, $T2_{n-1}$ large/$T1_n$ small, $T2_{n-1}$ small/$T1_n$ large) was performed to identify differences in the serial dependence strength between the two experiments.

Results and discussion

The role of post-saccadic errors in shaping serial dependence was investigated by systematically manipulating the spatial constant and contrast, and therefore the visuospatial uncertainty, of pre-saccadic and post-saccadic targets (T1 and T2, respectively). Specifically, the effect of the previous post-saccadic error ($T2_{n-1}$) on the current one ($T1_n$) was investigated. Two-dimensional Gaussian blobs were used as saccadic targets and their visuospatial uncertainty varied across two experiments, the “constant contrast” experiment in which target contrast was uniform across target sizes, and the “adjusted contrast” experiment in which the contrast decreased with increasing spatial constant of the targets.

Linear regression analyses revealed trial-by-trial influences of the post-saccadic error independent of visuospatial uncertainty. These findings align with saccadic amplitude changes observed in typical saccadic adaptation paradigms which minimize the post-saccadic error (Study 1; Bahcall & Kowler, 2000; Collins & Wallman, 2012; Noto & Robinson, 2001; Pélisson et al., 2010; Pomè et al., 2023; Wallman & Fuchs, 1998; Wong & Shelhamer, 2010; Zimmermann & Lappe, 2009, 2010). They further support previous findings of adaptive saccade amplitude adjustments from trial to trial (Cont & Zimmermann, 2021).

However, the serial dependence strength varied between experiments. In the “constant contrast” experiment, a pre-saccadic target ($T1_n$) with a high spatial constant in the current trial preceded by a low and therefore certain post-saccadic target ($T2_{n-1}$) resulted in weaker trial-by-trial influences of the post-saccadic error compared to the other two conditions, supporting that target visibility influences motor targeting rather than perceptual localization. In contrast, in the “adjusted contrast” experiment, serial dependencies were equally strong regardless of target size. Notably, when the current pre-saccadic target ($T1_n$) had a high spatial constant and the previous post-saccadic target ($T2_{n-1}$) had a low spatial constant, the serial dependence strength differed significantly between the two experiments. These findings suggest that the size and the contrast of the pre-saccadic target (T1) were key factors in determining the impact of post-saccadic errors on serial dependence. Specifically, larger and more visible pre-saccadic targets led to greater tolerance in post-saccadic error evaluation, resulting in weaker adaptive changes. In contrast, small pre-saccadic targets induced stronger serial dependencies, which highlights the crucial role of target size and visibility in shaping trans-saccadic processes. The observed asymmetry, that is, the contingency of serial dependence strength on the change in target size across saccades, further emphasizes that serial dependence strength was driven by pre-saccadic target characteristics rather than the change in target size itself.

Additionally, given the ballistics characteristics of saccadic movements and the absence of saccadic amplitude changes during saccade execution, the post-saccadic target of the current trial ($T2_n$) did not influence current saccadic performance. Further, a stronger undershoot of saccadic amplitudes occurred in the “adjusted contrast” experiment compared to the “constant contrast” experiment which aligns with the results of Lisi et al. (2019). Their findings indicated that the strength of saccadic undershoot scales with the visuospatial uncertainty of the target: Saccadic eye movements towards uncertain targets result in a stronger saccadic undershoot. In Study 2 of this dissertation, no differences in serial dependence strength were observed between the two experiments and conditions, nor was consistent temporal tuning of *n*-back trials found.

Conclusion

In conclusion, Study 2 demonstrated that the characteristics of a pre-saccadic target played a crucial role in determining the extent to which post-saccadic errors induced adaptive amplitude changes. Specifically, an increase in the size of the pre-saccadic target and a constant contrast led to a reduction in the serial dependence strength. In contrast, the uncertainty of the previously experienced post-saccadic target did not lead to changes in serial dependence strength. This finding underscores the significant influence of stimulus characteristics on the serial dependence strength.

Study 3

Research question and hypotheses

Previous research has demonstrated distinct neural mechanisms and attentional demands for voluntary and reactive, hereafter delayed, saccades (Gerardin et al., 2012; Müri & Nyffeler, 2008; Schraa-Tam et al., 2009), suggesting that endogenous and exogenous overt attention shifts may affect serial-dependent influences of the post-saccadic errors divergently. For sensorimotor serial dependence, evidence from saccadic adaptation points to differences in the serial dependence strength for endogenous and exogenous overt attention shifts. For visual serial dependence, however, literature provides no prior expectations for an interpretation. Thus, it remains unclear whether serial dependence is selective for one mode of attention allocation. Study 3 addressed this research gap with two experiments designed to investigate how voluntary and delayed saccades differ in attentional demands and how these types of saccades shape sensorimotor and visual serial dependence.

Method

Study 3 involved a total of 44 participants across two experiments with three conditions each. Twenty-two participants (mean age = 21.13 years, *SD* = 2.49 years; 19 females, 3 males) took part in Experiment 1. Twenty-two new participants (mean age = 22.71 years, *SD* = 5.22 years; 18 females, 4 males) took part in Experiment 2. Stimuli were presented by a 2014 Mac Mini computer on a 12.9 inch CRT monitor with a resolution of 800 × 600 pixels and a refresh rate of 120 Hz, generated using MATLAB R2016b (v. 7.10.0; The MathWorks, Natick, MA, United States) and PsychToolbox routines (v. 3.0.17; Kleiner et al., 2007).

In Experiment 1, sensorimotor serial dependence was investigated by saccade-to-saccade amplitude adjustments while in Experiment 2, visual serial dependence was investigated by an orientation judgment task. In both experiments, participants performed three conditions: Condition A with only voluntary saccades, Condition B with only delayed saccades, and Condition C with alternating voluntary and delayed saccades. The order of conditions was fixed across participants to allow the calculation of the mean voluntary saccade latency (Condition A) which was used to match the mean trial length of voluntary and delayed saccade trials (Condition B and Condition C). Condition A and Condition B each comprised 400 trials (20 minutes per condition), while Condition C included 800 trials (40 minutes).

In Experiment 1, voluntary saccade trials began with the simultaneous presentation of a fixation square (6.5° left of the screen center) and a target square (T1; 6.5° right of the screen center). Participants were instructed to perform a saccade towards T1 at their own pace. For delayed saccade trials, the same visual input was presented but participants were required to perform a saccade towards T1 as soon as they heard an auditory “go”-signal. This “go”-signal was calculated based on the mean voluntary saccade latency of each participant in Condition A to ensure that the mean delayed saccade latency in Condition B and Condition C was, on average, equally long as the mean voluntary saccade latency. For both, voluntary and delayed saccade trials, T1 was displaced to one out of six positions (T2; −2.5°, −1.5°, −0.5°, 0.5°, 1.5°, 2.5°) during saccade execution, defined as eye velocity bigger than $30° s^{-1}$ in five consecutive samples.

In Experiment 2, a Gabor patch served as the target stimulus (T1). Its orientation was randomized between five possible orientation values (25°, 35°, 45°, 55°, 65°). As in Experiment 1, participants performed only voluntary saccade trials (Condition A), only delayed saccade trials (Condition B), or alternatingly voluntary and delayed saccade trials (Condition C) towards T1 and maintained fixation after saccade landing, defined as eye velocity slower than $30° s^{-1}$ in five consecutive samples. After saccade landing, the Gabor patch disappeared, and a response bar appeared. The retinal distance between this response bar and the last fixation (T1) was identical to the retinal distance from the first fixation square to T1. Participants aligned the response bar to match the perceived orientation of the Gabor patch.

Trials were excluded from the analyses if no saccade was executed, if the saccadic amplitude was smaller than half the required distance of 6.5°, or if the peak velocity exceeded $800° s^{-1}$. For delayed saccade trials, anticipatory saccades before the auditory “go”-signal were excluded. For Experiment 2, fixation losses at the Gabor patch position were excluded (defined as gaze position exceeding a radius of 2.5° around the Gabor patch). On average, 5 % of trials were excluded per participant. For offline analyses, the three experimental conditions were split into four condition types: (a) voluntary saccades from Condition A, (b) delayed saccades from Condition B, (c) voluntary saccades preceding delayed saccades from Condition C and (d) delayed saccades preceding voluntary saccades from Condition C. For Experiment 1, the post-saccadic error for each trial was computed as the difference between the saccadic landing position and the actual position of the target (6.5°). Negative values indicate an undershoot of saccadic performance, while values close to zero indicate a perfect saccadic performance. For Experiment 2, the deviation error between the Gabor patch orientation and the reproduced orientation was computed. A deviation error of zero implies a perfect reproduction of the perceived orientation. Serial dependence effects were assessed via the relationship between the post-saccadic error in the previous and the current trial (Experiment 1) or the deviation error and the reproduced orientation (Experiment 2) in the previous and the current trial with linear regression analyses. Bonferroni-corrected one-sample *t*-tests against zero were performed to test for significant serial dependencies. Additionally, a 2 × 2 repeated-measures ANOVA with the within-subjects factor *previous trial* (voluntary, delayed) and the within-subjects factor *current trial* (voluntary, delayed) was conducted for both experiments to assess differences in the serial dependence strength.

Results and discussion

In this Study 3, it was investigated how voluntary and delayed saccades, which are assumed to differ in attentional demands and neural processes, affect serial dependence. It was differentiated between sensorimotor serial dependence (Experiment 1) and visual serial dependence (Experiment 2) to uncover potential divergent effects of the type of saccade.

Linear regression analyses revealed sensorimotor serial dependence effects on post-saccadic errors and visual serial dependence effects on orientation judgments, independently of the type of saccade combination. This aligns with previous findings on saccadic amplitude changes in a serial-dependent manner (Study 2; Cont & Zimmermann, 2021) and from typical adaptation paradigms which minimize the post-saccadic error (Study 1; Bahcall & Kowler, 2000; Collins & Wallman, 2012; Noto & Robinson, 2001; Pélisson et al., 2010; Pomè et al., 2023; Wallman & Fuchs, 1998; Wong & Shelhamer, 2010; Zimmermann & Lappe, 2009, 2010), as well as with previous findings on visual orientation judgments in a serial-dependent manner (Alais et al., 2017; Cicchini et al., 2017; Collins, 2019; Fischer & Whitney, 2014; Fritsche & De Lange, 2019; Fritsche et al., 2017; Murai & Whitney, 2021; Pascucci et al., 2019; Rafiei et al., 2021; Tanrikulu et al., 2023).

Further, differences between the sensorimotor serial dependence strength (Experiment 1) and the visual serial dependence strength (Experiment 2) were observed. In Experiment 1, sensorimotor serial dependencies were significantly stronger when only voluntary saccades were performed (Condition A), compared to all other combinations of types of saccades. More precisely, when voluntary saccades were performed in both the previous and the current trial, saccadic amplitudes exhibited a stronger trial-to-trial influence compared to consecutive trials involving delayed saccades (preceding, following, or solely). The asymmetric transfer of sensorimotor serial dependencies between voluntary and delayed saccades may reflect different programming stages for both types of saccades (Deubel, 1995b), possibly due to different modes of attention allocation.

As elucidated, this asymmetric transfer might also be observed for visual features like visual orientation judgments. In Experiment 2, therefore, voluntary and delayed saccades were each combined with visual orientation judgments. Visual serial dependencies were equally strong across different types of saccades thereby crucially differing from sensorimotor serial dependencies. As the absence of differences in the serial dependence strength in Experiment 2 could be due to differences in attention shifts for undershooting and overshooting saccades, a median split of saccadic amplitudes in the current trial was conducted. Specifically, attention shifts for undershooting saccades were expected to be less precise, leading to stronger serial dependencies than for overshooting saccades. A higher uncertainty in saccadic performance should further increase trial-by-trial influences. Study 3 revealed higher serial dependence strengths for undershooting saccades compared to overshooting saccades but only when solely voluntary saccades were performed (Condition A). Further, for consecutive voluntary saccades (Condition A), a stronger baseline deviation error for undershooting saccades compared to overshooting saccades was found, indicated by more negative intercepts.

Conclusion

In conclusion, Study 3 showed an asymmetric transfer of post-saccadic error information: Exclusively for voluntary saccades, sensorimotor serial dependence increased, suggesting a stronger influence of endogenous attention compared to exogenous attention. By contrast, visual serial dependence seemed to be insensitive to post-saccadic error information transfer resulting from different types of saccades, indicating that exogenous and endogenous overt attentional shifts involved in delayed and voluntary saccades, respectively, impact sensorimotor serial dependence to a stronger degree than visual serial dependence.

General discussion

The general scientific objective underlying this dissertation was to assess the recalibration processes driven by post-saccadic errors. Visual perception is closely linked to saccadic eye movements, suggesting an information transfer between motor and visual parameters (Awater et al., 2005; Bahcall & Kowler, 1999; Bruno & Morrone, 2007; Collins et al., 2007; Garaas & Pomplun, 2011; Georg & Lappe, 2009; Hernandez et al., 2008; Moidell & Bedell, 1988; Schnier et al., 2010; Zimmermann & Lappe, 2009, 2010). This raises the question of whether this transfer follows the shared resource model or the active recalibration model. To successfully stabilize the perception of the world, serial dependence emerges, indicating that previous perception influences current perception. The post-saccadic error provides crucial information for this recalibration process (Awater et al., 2005; Bahcall & Kowler, 1999; Bruno & Morrone, 2007; Collins et al., 2007; Cont & Zimmermann, 2021), such as information on saccadic success, offering insights to improve or maintain current behavior in the environment. To gain a deeper understanding of the interaction between action and perception, this dissertation addressed the questions of how the connection between motor and visual parameters is represented, tested via saccadic adaptation, and how post-saccadic errors are processed in a serial-dependent way.

Impact of saccadic adaptation on visual localization

The link between action and perception might rely on the shared resource model, aligning spatial information more effectively (Awater et al., 2005; Bahcall & Kowler, 1999; Bruno & Morrone, 2007; Collins et al., 2007). However, evidence for separate neural pathways processing motor and visual spatial parameters suggests a more distinct but interacting relationship between these systems (Cont & Zimmermann, 2021; Cotti et al., 2007; Gerardin et al., 2012; Müri & Nyffeler, 2008; Rivaud et al., 1994; Schraa-Tam et al., 2009). Insights into the underlying theories can be gained by examining the behavioral patterns: A shift in motor and visual parameters in the same direction would support the shared resource model, whereas divergent shifts would indicate independent recalibration processes, supporting the active recalibration model. In Study 1, participants completed conditions with varying number of trials in which either the retinal or prediction error was set to zero. Following these trials, participants localized visual targets while maintaining fixation.

The first central finding of Study 1 was a replication of prior research in line with the assumptions of the forward model. It was observed that saccadic amplitudes adapt to minimize the spatial mismatch between predicted and actual saccade landing positions (Bahcall & Kowler, 2000; Collins & Wallman, 2012; Noto & Robinson, 2001; Pélisson et al., 2010; Pomè et al., 2023; Wallman & Fuchs, 1998; Wong & Shelhamer, 2010; Zimmermann & Lappe, 2009, 2010). Second, visual localization shifted following saccadic adaptation, consistent with evidence that motor signals influence visual spatial perception (Awater et al., 2005; Cheviet et al., 2022; Garaas & Pomplun, 2011; Moidell & Bedell, 1988; Zimmermann & Lappe, 2009, 2010), which supports the shared resource model. One could argue that visual mislocalization originated from motor information encoded in the efference copy. Research on trans-saccadic localization, in which the stimulus location is perceived before saccade execution and reported afterwards, has documented concomitant shifts in motor and visual parameters, albeit predominantly near the saccade target (Awater et al., 2005; Bahcall & Kowler, 1999; Bruno & Morrone, 2007; Collins et al., 2007). If the efference copy were the sole factor underlying this observation, one would expect visual mislocalization to be uniformly distributed across the visual field (Bahcall & Kowler, 1999). Thus, an explanation based exclusively on the efference copy appears insufficient to account for these spatially confined errors. Instead, the evidence suggests that an integrated neural mechanism, which links motor signals and visual localization, underlies this phenomenon.

When post-saccadic error information was unavailable, however, visual mislocalization shifted towards the fovea, replicating Cont and Zimmermann (2021). To investigate the relationship between action and perception in the “no error” trials of Study 1, the number of these trials was manipulated and the effects of the retinal error and the prediction error on visual localization were compared. While saccadic adaptation remained stable over the course of “no error” trials, visual mislocalization was dynamic: Without perceived prediction error, visual localization shifted towards the fovea, independently of the number of “no error” trials. However, without perceived retinal error, visual mislocalization stabilized on an adapted level over a short period of “no error” trials but shifted in foveal direction over a long period of “no error” trials. This dissociation between motor and visual parameters yields evidence in support of independent but interacting recalibration mechanisms. Evidence for this dissociation has also been observed when saccadic adaptation was experimentally suppressed but visual mislocalization persisted (Heins & Lappe, 2022). The present results suggest that visual recalibration does not occur in early visual brain areas. If early visual brain areas mediated visual recalibration, the observed changes in saccade amplitudes should reflect visual rather than motor adaptation. However, since visual mislocalization in Study 1 decayed while saccadic adaptation remained stable, this possibility of early visual brain areas mediating visual recalibration can be ruled out. This underscores the assumption of distinct processes governing motor and visual recalibration. The best neural candidate for recalibration of the motor and visual system is the parietal cortex as a hub for constructing visual space (Husain & Nachev, 2007; Zhou et al., 2016), generating saccades (Pierrot-Deseilligny et al., 1991), and mediating saccadic adaptation (Panouillères et al., 2012).

Motor adaptation involves at least two components: a fast learning process with rapid decay and a slower process with prolonged retention (Ethier et al., 2008; Körding et al., 2007; Krakauer et al., 2019). Visual recalibration in Study 1 exhibited faster decay compared to motor adaptation. This raises the question of whether visual recalibration generally demonstrates faster learning dynamics or whether its faster decay reflects distinct neural processes. Importantly, the comparable magnitude of motor parameters and visual mislocalization immediately after saccadic adaptation indicates that saccadic adaptation is not a purely visual phenomenon but involves distinct mechanisms for motor and visual recalibration. The temporal dynamics of error signals further provide relevant insights. The persistent adaptation effects after error annulment contrast with the rapid decay of visual mislocalization. These different patterns may reflect distinct temporal properties of the reliance of the motor and visual systems on error signals for recalibration. It needs to be noted that perfect "no error" trials are challenging to achieve. However, in Study 1, error signals were effectively minimized, with the observed mismatch between predicted and actual landing positions being small (~0.2°), indicating a high level of alignment. Future research should explore how neural representations of retinal and prediction errors evolve over time and divergently impact motor and visual parameters.

In conclusion, the findings presented in Study 1 favor the active recalibration model for motor and visual spatial recalibration over the shared resource model. While shared resources may facilitate initial alignment between action and perception, the dissociation observed in the data highlights the existence of separate recalibration processes and thus separate resources. These insights contribute to a deeper understanding of the mechanisms underlying saccadic adaptation and visual localization and provide a foundation for investigating their interaction in more complex spatial tasks.

Serial dependence in post-saccadic error processing

Saccadic adaptation paradigms typically employ consistent target displacements which lead to saccadic amplitude adjustments to minimize the post-saccadic error (Bahcall & Kowler, 2000; Collins & Wallman, 2012; Noto & Robinson, 2001; Pélisson et al., 2010; Pomè et al., 2023; Wallman & Fuchs, 1998; Wong & Shelhamer, 2010; Zimmermann & Lappe, 2009, 2010). However, studies using randomized displacements indicate that adaptation can also occur on a single-trial basis (Collins, 2014; Desmurget et al., 2000; Havermann & Lappe, 2010; Srimal et al., 2008), thereby allowing for the assessment of trial-by-trial influences of post-saccadic errors (Cont & Zimmermann, 2021). This phenomenon, known as serial dependence, functions to stabilize visual perception by smoothing sensory input over time, reducing noise, and facilitating a coherent representation of the external environment (Cicchini et al., 2017; Fischer & Whitney, 2014). This effect has been demonstrated across various domains of perception and action, underscoring its universal nature. To further examine serial dependence effects for post-saccadic errors, Study 2 and Study 3 of this dissertation investigated the influence of two key factors on serial dependence within saccade-related contexts: stimulus size in relation to visuospatial uncertainty (Ceylan et al., 2021) and attentional effects induced by different types of saccades (Bae & Luck, 2020; Müri & Nyffeler, 2008; Schraa-Tam et al., 2009).

In Study 2 of this dissertation, it was examined how post-saccadic errors shape serial dependence by varying the spatial constant and contrast of both pre-saccadic and post-saccadic targets. Using Gaussian blobs as stimuli, target size and target contrast were manipulated to simulate varying levels of visuospatial uncertainty. Hypotheses about the serial dependence strength were two-sided. If target visibility primarily affected perceptual localization, serial dependence should be stronger when the pre-saccadic target had a high spatial constant. Contrary, if target visibility influenced motor targeting, serial dependence should decrease when the post-saccadic target had a high spatial constant. The results revealed overall trial-by-trial influences. Notable differences in the strength of serial dependence emerged depending on the manipulated stimulus characteristics. Specifically, serial dependencies were weaker when pre-saccadic targets with a high spatial constant (implying larger visuospatial uncertainty) were paired with post-saccadic targets with a low spatial constant but only with a constant contrast due to which the saccadic target appeared more luminant than with an adjusted contrast. This asymmetry suggests that spatial properties and visibility of pre-saccadic targets, rather than trans-saccadic changes in size, shaped serial, supporting that target visibility influences motor targeting rather than perceptual localization.

The observed connection between saccadic dynamics and serial dependence can be interpreted within the framework of behavioral optimization and perceptual stabilization. Highly precise saccadic movements, facilitated by well-defined targets, minimize the need for adaptive error correction, thereby reducing the serial dependence strength. Conversely, erratic or highly variable saccades resulting from target uncertainty may disrupt optimization processes, weakening serial dependence. The findings obtained in Study 2 highlighted that the strongest serial dependence occurred in scenarios in which saccades were precise but still flexible enough to allow subtle corrections based on prior experience. To examine the role of target uncertainty, the spatial constant of targets was manipulated as a proxy for visuospatial uncertainty. The data does not support the hypothesis that higher spatial constants, indicating larger visuospatial uncertainty, increase saccadic variability or reliance on serial dependence for stabilization. Saccade variability did not increase for targets with larger uncertainty, nor did saccadic amplitudes more extensively incorporate post-saccadic error under these conditions. Instead, the findings suggest that pre-saccadic target visibility and spatial properties were the critical factors determining the impact of post-saccadic error on serial dependence. It was observed that large pre-saccadic targets induced weaker adaptive changes compared to small, spatially focused pre-saccadic targets. This suggests that large targets offer more tolerance for landing errors as multiple positions could be perceived as correct. Conversely, small pre-saccadic targets provide precise spatial anchoring, reducing the influence of post-saccadic target size on the serial dependence strength. Interestingly, high-contrast post-saccadic targets amplified the observed asymmetry, thereby underscoring the necessity of sufficient target contrast for effective error evaluation. These findings suggest that trans-saccadic changes in target size alone did not fully explain the variations in the serial dependence strength. Instead, pre-saccadic target properties serve as a critical anchor for post-saccadic error evaluation.

Voluntary saccades involve endogenous attention allocation while delayed saccades rely on exogenous attention. In Study 3 of this dissertation, it was tested how these different attentional demands of voluntary and delayed saccades affected serial dependence. Two experiments were conducted: Experiment 1 assessed sensorimotor serial dependence by analyzing trial-by-trial effects of artificially induced post-saccadic errors while Experiment 2 assessed visual serial dependence in an orientation judgment task, investigating whether the type of saccade influenced the serial dependence strength. For sensorimotor serial dependence, differences in the serial dependence strength for endogenous and exogenous overt attention were hypothesized while for visual serial dependencies, no prior hypotheses were set. The results revealed trial-by-trial influences across all experiments and types of saccades, however, the serial dependence strength varied: Sensorimotor serial dependencies exhibited a stronger transfer of saccadic amplitude for voluntary saccades while visual serial dependencies were equally strong regardless of the type of saccades. The asymmetric transfer observed in saccadic performance likely reflects differences in motor programming between voluntary and delayed saccades. Notably, the symmetry in the visual orientation task suggests that visual serial dependence operates independently of attentional mechanisms governing saccade programming. This dissociation supports the hypothesis that sensorimotor serial dependence arises primarily from motor-specific processes, whereas visual serial dependence stabilizes perception by smoothing sensory input based on prior experience.

These findings align with previous studies demonstrating an asymmetric transfer of motor error information in saccadic adaptation (Alahyane et al., 2007; Collins & Doré-Mazars, 2006; Deubel, 1995b; Erkelens & Hulleman, 1993). Taken together, these studies suggest that the differences in motor error transfer between voluntary and delayed saccades originate at the saccade programming stage. Early models, such as the one proposed by Deubel (1995b), attributed voluntary saccadic adaptation to higher-order frontal areas while attributing delayed saccadic adaptation to the superior colliculus. According to this hierarchical framework, delayed saccades should remain unaffected by changes in the motor plan for voluntary saccades. By contrast, voluntary saccades should be influenced by adaptation processes occurring in the superior colliculus, as the planning signals for voluntary saccades would traverse this structure. This model is supported by evidence that the frontal eye field is associated with the execution of voluntary saccades (Müri & Nyffeler, 2008; Schraa-Tam et al., 2009) while the superior colliculus is associated with both, the processing of voluntary and delayed saccades (Ilg & Thier, 2012). This hierarchical organization could explain the asymmetric patterns observed in motor error transfer in Study 3. However, neural activation patterns associated with saccadic adaptation present a more complex and nuanced scenario than these assumptions suggest: Delayed saccades primarily engaged ventral regions of the frontal and parietal cortex, whereas voluntary saccades predominantly activated more dorsal areas within these cortical regions (Gerardin et al., 2012; Panouillères et al., 2012). This dissociation aligns with the dorsal and ventral attention network (Corbetta & Shulman, 2002), underscoring the role of attentional and motor programming pathways in shaping saccadic behavior. Recent evidence emphasized a central role of the cerebellum in detecting post-saccadic errors and adjusting amplitudes (Desmurget et al., 2000; Desmurget et al., 1998; Gerardin et al., 2012; Guillaume et al., 2018; Herzfeld et al., 2018; Métais et al., 2022).

In the visual orientation judgment task, serial dependencies were equally strong regardless of the combination of voluntary or delayed saccades. This lack of motor-type specificity suggests that the stabilization of perception via visual serial dependence is independent of whether objects were attended to endogenously or exogenously. Unlike sensorimotor serial dependence, visual serial dependence is likely used to interpret the external environment and to generalize across different attentional mechanisms. However, findings from paradigms involving covert attention shifts should be compared to overt attention shifts with caution. Evidence from Casteau and Smith (2020) indicates that only exogenous overt attention is coupled tightly to eye movement programming whereas endogenous overt attention could operate independently, focusing on visual field regions beyond the involvement of eye movements. This independence of endogenous overt attention might also explain the asymmetric transfer of motor adaptation observed in Study 3.

Taken together, Study 2 and Study 3 of the present dissertation demonstrate that serial dependence is a pervasive characteristic of both visual and motor processes, modulated by factors such as uncertainty and attention. The results indicate that pre-saccadic target features act as critical anchors for error evaluation. Further, they reveal distinct behavioral mechanisms underlying sensorimotor versus visual serial dependence. This supports the notion that serial dependence operates as a selective smoothing process to stabilize representations of sequentially attended features, thereby enhancing the efficiency of the perceptual system in processing the history of a perceived scene. Future research should investigate the interplay of overt and covert attention in shaping serial dependence and explore the generalizability of these findings across different sensory modalities and task domains.

Limitations and future directions

Despite the valuable insights provided by the three studies of the present dissertation, several limitations need to be addressed in future research to deepen the understanding of the perceptual and neural mechanisms underlying saccadic adaptation, serial dependence, and post-saccadic error processing. One limitation of this dissertation lies in its reliance on behavioral data only, which, while informative, does not allow an investigation of the underlying brain structures. Neuroimaging techniques such as functional magnetic resonance imaging or electrophysiological recordings like electroencephalography could unravel the neural correlates of saccadic adaptation, specifically the roles of the parietal cortex and the cerebellum in prediction error processing and visual localization. Studies have shown that electrical stimulation of deeper layers of the superior colliculus can induce saccadic motor learning (Kaku et al., 2009) and that neurons in the superior colliculus alter their firing patterns following adaptation (Takeichi et al., 2007). Additionally, studies highlight the involvement of the posterior parietal cortex in post-saccadic error detection (Husain & Nachev, 2007; Robinson et al., 1978), saccadic adaptation (Gerardin et al., 2012; Panouillères et al., 2014), and coding of post-saccadic eye position (Zhou et al., 2016). Lastly, mounting evidence points to the cerebellum, particularly its oculomotor vermis, as a key brain area for saccadic adaptation: Lesions in the cerebellar vermis impair saccadic adaptation in both humans and monkeys (Optican & Robinson, 1980; Straube et al., 2001; Takagi et al., 1998) and Purkinje cell population bursts may underly mechanisms of inward and outward adaptation (Catz et al., 2008). Although the involvement of these brain areas in both action and perception has been documented, their connectivity and combined influence on the integration of saccadic adaptation and visual localization remain to be fully elucidated. Future research should use neuroimaging techniques to identify a neural network that integrates oculomotor and visual inputs independently yet interactively.

Moreover, the role of temporal dynamics in saccadic adaptation requires further exploration. While the effects of immediately preceding saccades (i.e., *n*-1 effects) have already been studied, it remains unclear how more distant preceding saccades (e.g., *n*-2, *n*-3 effects) influence visual perception during adaptation. While Zimmermann and Lappe (2010) report descriptive serial dependence effects of more distant preceding saccades on visual localization, more ambiguous effects were found in Study 2 of this dissertation. However, this may be due to the alternating trial structure between saccadic and visual localization trials, which differs from typical investigations of temporal dynamics with uniform trial types. Future research should examine whether post-saccadic error processing and visual localization evolve across successive trials and how these dynamics impact perceptual experience in the context of visual-motor integration. Additionally, future studies could investigate how long saccadic amplitudes persist adapted depending on the duration of perceived “no error” trials (see Study 1).

Study 2 and Study 3 provide valuable insights into sensorimotor serial dependence by examining saccade-by-saccade influences. Although these findings support predictions regarding the effects of post-saccadic errors on visual localization, it remains unclear how these dependencies ultimately modulate visual outcomes. Under conditions of increased saccadic target uncertainty (Study 2), the internal representation of the target position became less reliable. This reduced reliability may lead to a diminished weighting of target information during the active recalibration process, resulting in a smaller bias of saccadic metrics in visual localization. Consequently, visual localization might be less influenced by post-saccadic error signals, yielding a greater dissociation between action and perception, an effect that is comparable to the outcomes observed in the “no error” trials of Study 1. For the findings reported in Study 3, previous literature suggests an interaction between the type of saccade and the localization performance. For instance, Zimmermann and Lappe (2009) demonstrated that both saccadic adaptation and visual mislocalization vary with the nature of the saccade (voluntary versus delayed) and the type of localization target (stationary versus flashed), reporting an asymmetric transfer of effects: Adapted delayed saccades led to visual mislocalization when flashed targets are used, while adapted voluntary saccades led to visual mislocalizations in both flashed and stationary targets. Complementarily, studies have revealed distinct neural control mechanisms and attentional demands involving delayed and voluntary saccades (Gerardin et al., 2012; Müri & Nyffeler, 2008; Schraa-Tam et al., 2009). According to the active recalibration model, these differences should be reflected in the visual localization, with exogenously driven spatial representations being recalibrated independently from endogenously driven ones, specifically for more similar types of localization targets. Future research should aim to clarify the interplay between saccade dynamics and visual localization, particularly in the context of target uncertainty and varying types of saccades.

Moreover, since saccadic eye movements have been suggested to be behavioral indicators of psychiatric disorders, future studies should analyze how post-saccadic errors are processed in clinical populations. For example, Pomè et al. (2023) have recently suggested that the trans-saccadic updating, as well as serial dependence for saccadic eye movements, might be compromised in the autism spectrum disorder due to dysfunctions in the efference copy. They found slower saccadic outward adaptation and weaker transfer of saccadic metrics to visual parameters. Dysfunctions in the efference copy were also found in patients with schizophrenia (Spering et al., 2013), underlying potential impairments in trans-saccadic updating that might be a phenomenon across different psychiatric disorders. Investigating how motor parameters adapt in the clinical population, particularly in relation to saccadic movements, could provide critical insights into the role of sensory-motor integration and error processing.

General conclusion

The findings presented in this dissertation provide novel insights into the role of post-saccadic errors in recalibrating motor and visual space. By systematically manipulating post-saccadic errors, it was demonstrated that saccadic adaptation and serial dependence are fundamental mechanisms for maintaining sensorimotor accuracy and perceptual stability. In three experimental studies, it was examined how post-saccadic errors drive recalibration, how target uncertainty modulates serial dependence, and how different types of saccades influence sensorimotor and visual serial dependence. Study 1 revealed that motor and visual recalibration rely on distinct yet interacting mechanisms, as adaptation persisted despite the absence of post-saccadic errors while visual mislocalization was modulated by error type, supporting the active recalibration model rather than the shared resource model. Study 2 showed that target appearance played a crucial role in serial dependence, with post-saccadic errors dynamically weighted based on the uncertainty of the current target. Study 3 demonstrated that sensorimotor serial dependence was specific to the type of saccade whereas visual serial dependence remained unaffected, indicating distinct underlying processes. Together, these findings highlight the adaptability of the sensorimotor system, which flexibly integrates error information to optimize both motor execution and spatial perception. Understanding these recalibration processes advances our knowledge of sensorimotor learning and provides a basis for future research on adaptive mechanisms in oculomotor control and perception.

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Affidavit

Eidesstattliche Erklärung gemäß § 5 der Promotionsordnung vom 15.06.2018 der Mathematisch-Naturwissenschaftlichen Fakultät der Heinrich- Heine-Universität Düsseldorf:

Ich versichere an Eides Statt, dass die Dissertation von mir selbständig und ohne unzulässige fremde Hilfe unter Beachtung der „Grundsätze zur Sicherung guter wissenschaftlicher Praxis an der Heinrich-Heine-Universität Düsseldorf“ erstellt worden ist. Die Dissertation wurde in der vorliegenden oder ähnlichen Form noch bei keiner anderen Institution eingereicht. Ich habe bisher keine erfolglosen Promotionsversuche unternommen.

Düsseldorf, den 11.03.2025 ____________________

Sandra Tyralla

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Dear **Mensa Gruppe**, you made my lunch breaks unforgettable. Balancing our schedules around teaching duties and the 12 o’clock rush was always a challenge, but you made it fun. **Larissa**, your mastery of balancing excellent advice combined with a calm perspective continues to impress me. I am delighted that we have managed to remain gym buddies for so long. Over the past years, a special group has formed: *die Zelle*. **Carolin** and **Fabian**, you have not only brightened my life but also transformed it for the better. It is always a pleasure sharing a single brain cell with you two. Also, thanks to *Barbie for the Win*: **Carolin**, **Nadine,** and **Hebli**. I cherished our chaotic attempts to create overly complicated, Instagram-inspired meals, often without even watching the pre-planned Barbie movie.

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To *Die Coolen*: **Steffi**, **Tina**, and my wife **Ina**: you are the best friends I could have asked for. Together, we have navigated through the challenges of adulthood, without none of us being ready for it. It is a true pleasure to hear from you every day. **Anna** and **Valle**, my favorite *Lama(r)s*, thank you for always having an open ear; I am looking forward to more chaotic holidays and upcoming half marathons (yay). **David**, you are always the anchor of calm, and your football team *GEschmack* is truly outstanding. Dear **DNA**, my longest-standing friends, many of whom I have known since fifth grade. I cannot imagine a life without the brilliant (and occasionally less brilliant) ideas that emerge from this group. Thank you for being such *MVPs* in my life. **Georg**, sometimes you just know when you have met your soulmate. Thank you for enduring my annoying tendencies (like minute-long voice messages) with your calm and endearing nature. I love you. **Papa**, dziękuję, że zawsze mnie wspierałeś, bez względu na wszystko. Jesteś najlepszym ojcem, jakiego mogłam sobie wymarzyć. Kocham cię.

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Appendix

Original article of Study 1

Tyralla, S., Pomè, A., & Zimmermann, E. (2023). Motor recalibration of visual and saccadic maps. *Proceedings of the Royal Society B: Biological Sciences*, *290*(1994), Article 2566. https://doi.org/10.1098/rspb.2022.2566

I was the first author of this article along with the second first author Dr. Antonella Pomè. I contributed to the development of the paradigm, created the experimental setup, conducted the data acquisition and analysis, contributed to the interpretation of the results, wrote the initial draft of the manuscript, and cooperated with all co-authors on the following versions of the manuscript.

Original article of Study 2

Tyralla, S., & Zimmermann, E. (2024). Serial dependencies in motor targeting as a function of target appearance. *Journal of Vision*, *24*(13), Article 6. https://doi.org/10.1167/jov.24.13.6

I was the corresponding and first author of this article. I contributed to the development of the paradigm, created the experimental setup, conducted the data acquisition and analysis, contributed to the interpretation of the results, wrote the initial draft of the manuscript, and cooperated with Prof. Dr. Eckart Zimmermann on the following versions of the manuscript.

Original article of Study 3

Tyralla, S., & Zimmermann, E. (2025). Serial dependencies and overt attention shifts. *Manuscript submitted for publication at Journal of Vision*.

I was the corresponding and first author of this article. I contributed to the development of the paradigm, created the experimental setup, conducted the data acquisition and analysis, contributed to the interpretation of the results, wrote the initial draft of the manuscript, and cooperated with Prof. Dr. Eckart Zimmermann on the following versions of the manuscript.

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Research

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Motor recalibration of visual and saccadic maps

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How does the brain maintain an accurate visual representation of external space? Movement errors following saccade execution provide sufficient information to recalibrate motor and visual space. Here, we asked whether spatial information for vision and saccades is processed in shared or in separate resources. We used saccade adaptation to modify both, saccade amplitudes and visual mislocalization. After saccade adaptation was induced, we compared participants' saccadic and perceptual localization before and after we inserted 'no error' trials. In these trials, we clamped the post-saccadic error online to the predicted endpoints of saccades. In separate experiments, we either annulled the retinal or the prediction error. We also varied the number of 'no error' trials across conditions. In all conditions, we found that saccade adaptation remained undisturbed by the insertion of 'no error' trials. However, mislocalization decreased as a function of the number of trials in which zero retinal error was displayed. When the prediction error was clamped to zero, no mislocalization was observed at all. The results demonstrate the post-saccadic error is used separately to recalibrate visual and saccadic space.

1. Introduction

Successful interaction with our environment requires that sensory input is organized into a representation of the external world that accurately encodes spatial relations. Neurons representing objects of interest in a perceptual map must convey information to corresponding neurons in motor maps such that precise actions can be generated. The success of each action is implicitly measured in the errors of movement production. Any deviation from a desired movement trajectory will be corrected by a modification of the following movement [1,2]. Minimization of movement errors thus recalibrates space in the motor map. The perceptual spatial map demands for a likewise recalibration. This process would require feedback about the actual state of the external world and a frequent occurrence in order to guarantee maintenance of precision.

Saccade eye movements are the prime candidate to satisfy the urgent need to recalibrate internal spatial maps for action and perception. They are the movements we perform most often per day (about three times per second) and their errors reveal mismatches between internal and external space. Saccades bring the fovea onto objects of interest, although they usually undershoot intended objects and land short of the target [3,4]. A corrective saccade will compensate for the undershoot such that the eye fixates the target [4]. The undershoot has been linked to the magnification factor in the superior colliculus [5]. Research has suggested that this inaccuracy does not reflect motor execution noise but a strategy of saccade control [6–8]. Saccade planning generates a prediction of where the eye will land and the sensed post-saccadic error, i.e. the distance of the fovea to the position of the target, will be compared against the prediction [9–11]. Prediction errors can become effective only after the movement has been completed since saccades are too fast to be adjusted online by visual feedback. Any deviation between the predicted landing error and the actual post-saccadic error will shape the amplitude of the following saccade such as to re-establish the predicted saccade landing. Laboratory experiments in

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previous research have presented an artificial post-saccade error of the same direction and size after execution of the same saccade amplitude (for a review see [12]). In these experiments, saccade adaptation gradually increases or decreases saccade amplitudes across trials until it reaches an adaptation steady state. The functional role of saccade adaptation has been seen in a compensation of eye muscle fatigue or damage. However, it has repeatedly been shown that adaptive modifications can be found on single saccades [13–16] and on top-down task-relevant information even in the absence of bottom-up visual error [17].

We have recently demonstrated that post-saccadic errors from single saccades recalibrate, in a serially dependent manner, saccade and visual space [18]. Post-saccadic errors might provide the signal that recalibrates the spatial metric not only in motor but also in visual maps (figure 1*a*). The crosstalk between action and perception gives rise to a fundamental question about how space is processed in the brain: Is there one resource of space, shared between perception and action, or are spatial coordinates for action and perception processed separately?

On one perspective, motor actions might produce necessary error signals to recalibrate internal spatial estimates to external space, such that both, action and perception, rely on a shared resource. In this view, any change in the motor adaptation must be reflected in visual space. Electrophysiological studies have suggested that the detection of the post-saccadic error is a function of the cerebellum [19]. Although adaptive modifications of saccade amplitudes are computed within the cerebellum, cortical processing of the post-saccadic error has been reported [20]. Neurons in posterior parietal cortex with persistent pre- and post-saccadic responses reflect the intended saccade landing based on efference copy information, whereas neurons with late post-saccadic responses represent the actual saccade ending position. Brain imaging studies in humans suggested that the parietal cortex is one of the sites where saccade adaptation takes place [21]. The parietal cortex represents a likely candidate to host a shared resource for action and perception, given its functional role as a hub for spatial coordinates [22]. The parietal cortex might alternatively relay error signals further into visual areas in order to recalibrate spatial maps separately. This route might serve the purpose of visual recalibration via post-saccadic errors.

A straightforward strategy to address how space is processed in the brain consists in an attempt to drive motor space and visual space in different directions. A singular shared resource would not allow such a dissociation. If motor and visual coordinates can be manipulated such as to shift in opposite directions, neural resources of space must be separate. Studies have shown that saccade adaptation is accompanied by a comparable shift in spatial perception (e.g. [23–26]) and in pointing movements [27].

In our experiments, we adapted saccade amplitudes. After adaptation, we applied trials in which we predicted saccade landing during the execution of the eye movement, in order to present the saccade target such that no error signal is ensued. In our previous study, we found that in the absence of motor errors, visual localization shifts in direction of the fovea [18]. In the current study we varied the number of trials in which the sensorimotor system received ‘no error’ information and wondered whether we would find concomitant or different shifts in localization between action and perception. Concomitant shifts would reinforce claims of a shared resource whereas different shifts would present clear evidence to the contrary.

2. Methods

(a) Participants

Nine subjects (mean age 25.78 years, s.d. = 4.79 years; 5 women) participated in the ‘no retinal error’ experiment (experiment 1). Four of them plus five additional (mean age = 22.43 years, s.d. = 5.13 years; 6 women) were tested on a second experiment (‘no prediction error’ experiment). Finally, four subjects (who also participated in the previous two experiments) plus three new subjects (mean age = 23.89 years, s.d. = 6.83 years; 5 women) took part in a third experiment (‘constant error’ experiment). Subjects were all German native speakers with no neurological or psychiatric diseases. Participants either reported to have normal vision or they wore lenses during their acquisition. All participants were recruited through the Heinrich-Heine University Düsseldorf and received either course credit or payment of 10 euros per hour.

(b) Setup

Stimuli were generated under Matlab R2016b (v. 7.10.0; The MathWorks, Natick, MA, United States) using PsychToolbox routines (v. 3.0.17 [28]) run by a Mac Mini, 2014 and presented on screen (CRT, 12.9 inches, Diamond Pro 2070) with a resolution of 800 × 600 pixel and a refresh rate of 120 Hz, placed at 57 cm from the observer. To avoid the potentially confounding influence of any visual references the room was completely dark (except for the EyeLink IR illuminator which emitted some diffuse light that could be potentially used as visual reference. However, the adaptation and mislocalization effects reported in the present study are comparable to the ones reported in literature [29,30]). A transparent foil reduced the luminance of the monitor by 2 log units and prevented the visibility of the monitor borders. Subjects were stabilized in a chin rest to prevent head movements. Before the experiment started subjects were dark adapted for 3 min. The background colour of the screen was dark (0.01 cd m^{-2}) to reduce illumination.

Eye movements were recorded by a desktop-mounted eye tracker (EyeLink 1000 Plus), sampling eye position at 1000 Hz. Subjects performed the task binocular but only the left eye was used for eye movement recording. At the beginning of the experiment a standard 9 points calibration routine was run. A standard keyboard and mouse were used to collect participants responses.

(c) Experimental procedure

Figure 1*b* shows the trial structure. Each trial began with a fixation square (0.55 × 0.55°) presented 6.5° left of the screen centre. Its colour indicated whether subjects were required to perform a saccade (red, *saccade trials*) or to keep fixation on the fixation point (blue, *localization trials*). The protocol consisted of the following blocks of trials: baseline localization and baseline saccades (*baseline*), adaptation followed by visual localization (*post-adaptation*), one block of ‘no error’ trials followed by visual localization (*post no error trials early*), a second block of ‘no error’ trials followed by visual localization (*post no error trials late*) and saccade de-adaptation (*de-adapt*). A condition started with 20 baseline localization trials and 20 baseline saccade trials. These were followed by 100 saccade adaptation trials in which the saccade target was displaced outward by 3° as soon as saccade execution was detected. In the next 20 trials, localization performance was measured (post-adaptation). Then, in the ‘no error’ trials (‘post no error trials early’ and ‘post no error trials late’), we manipulated the size of the post-

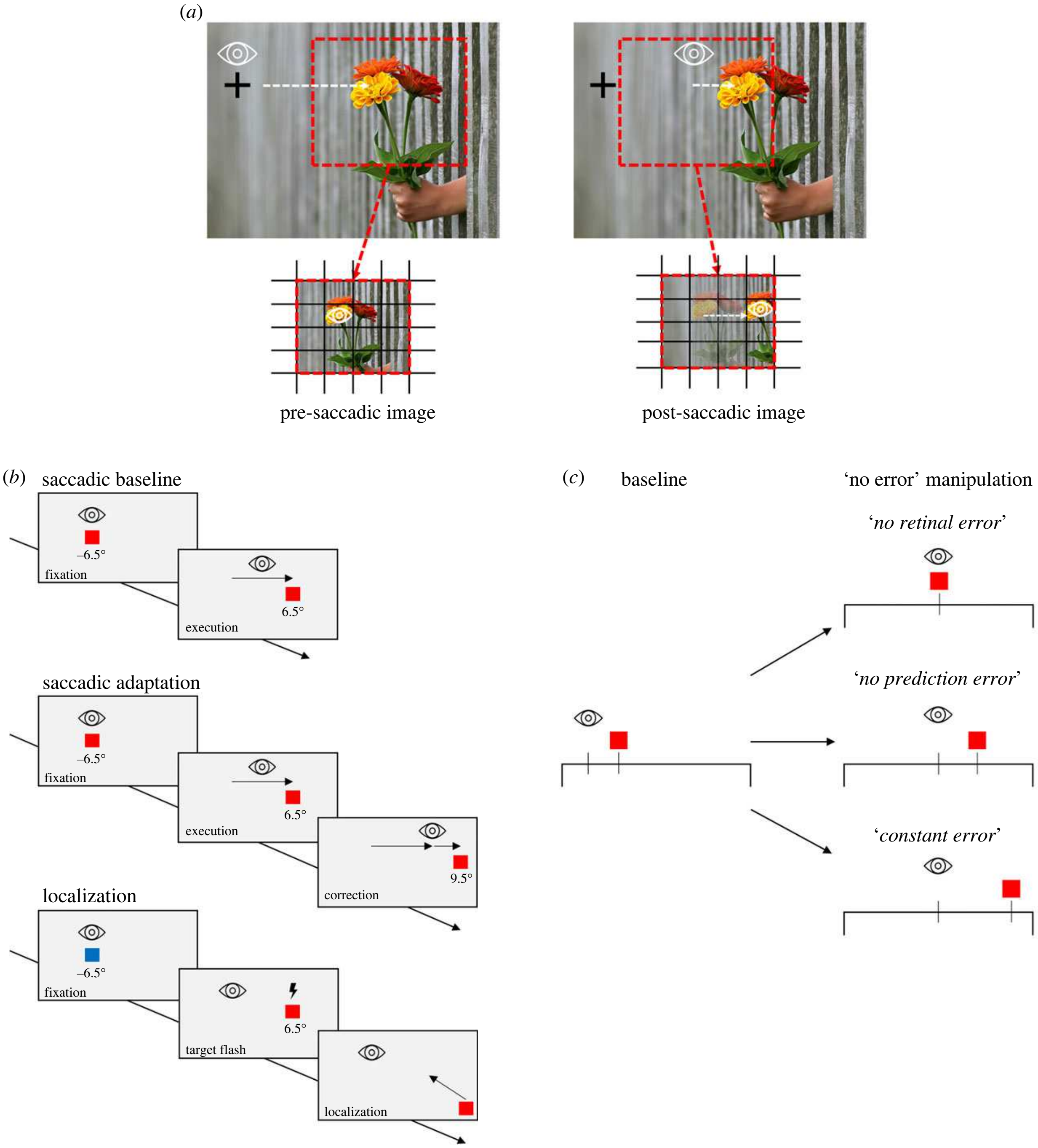

Figure 1. (*a*) Illustration of visual recalibration by the post-saccadic error. When making a saccade toward the flower on the right, its retinal projection travels from a peripheral location (pre-saccadic image) to a foveal location (post-saccadic image). Any error in foveate the predicted position of the object (i.e. saccade falling shorter on the flower) will be corrected by a modification of the following movement. (*b*) Saccade and localization task. Saccade baseline: subjects perform a saccade from a fixation square (–6.5°) to a target square (+6.5°), as soon as the fixation target disappears. Saccade adaptation: subjects saccade to the target, which is displaced 3° outwards from its pre-saccadic position, as soon as the saccade has been detected. Localization: subjects fixate a blue fixation square (–6.5°). After pressing the space bar, fixation disappears and a red target flash for 24 ms at the same position of the saccade target (+6.5°). Subjects indicate the perceived target position via mouse click. Background's colour during the experiment was dark. (*c*) Examples of the 'no error' manipulation applied. The initial saccade target is always at 13° (filled red square). In baseline (the same for all 'no error' manipulations), the eye undershoots the intended target position (saccade amplitude of 12°), which correspond to a retinal and prediction error. In the 'no error' manipulations (right panels), we predicted the saccade endpoint to apply different 'no error' trials. To obtain a retinal error of zero (no retinal error), the target had to be stepped to the predicted saccade landing position (red square). In the second example (no prediction error), the baseline prediction error of each subject has been added to the predicted eye landing position. Therefore, the prediction error is set to zero while the retinal error still occurs. 'Constant error' example: eye's landing position was predicted, and the target was displaced by adding a constant error of 3° to this prediction.

saccadic error systematically in separate experiments (figure 1*c* for an example of error modifications applied). To this end, we predicted the saccade landing position online and stepped the target according to the post-saccadic error provided. In separate conditions, we varied the number of 'no error' trials: in condition 'short' 12 'no error' trials, in condition 'medium' 25 'no error' trials and in condition 'long' 50 'no error' trials were presented. After these, 20 localization trials were tested. Blocks with 'no error' trials and localization trials blocks alternated two times. At the end of the session, 20 de-adaptation trials were applied. These trials were identical to the saccadic baseline block and were performed to cancel out the adaptation.

Each condition lasted for around 20 min. A minimum of 15 min break between each condition was conducted to ensure no saccadic adaptation transfer from one condition to the other. The short condition (12 'no error' trials) resulted in a total trial length of 244 trials, the medium condition (25 'no error' trials) resulted in 270 trials and the long condition (50 'no error' trials) resulted in 320 trials. The order of the conditions was randomized within participants.

(d) Saccadic baseline

At trial onset, a red fixation square (0.55 × 0.55°) was presented horizontally 6.5° left from the screen centre. The vertical position for all targets was always centred. A trial was initiated by disappearance of the fixation target if the subject had fixated it for a randomly selected time interval drawn from a uniform distribution between 500 and 1200 ms. After this time, a red target square (T1) of the same size as the fixation square was presented 6.5° right from the screen centre for 1200 ms. Subjects were instructed to perform a saccade toward the saccade target as soon as it appears. The next trial started with a new fixation square. One trial lasted around 3000 ms (figure 1*b*, saccadic baseline).

(e) Saccadic adaptation

After completing 20 trials of baseline, saccadic adaptation was induced (figure 1*b*, saccadic adaptation). The trial structure was identical to saccadic baseline, except that the target was displaced during execution of the saccade. Eye velocity was calculated online and the target then stepped 3° to the right (T2) as soon as the eye reached a velocity bigger than 30° s^{-1} in 5 consecutive samples. A new fixation square then appeared after the saccade was completed and the second target was extinguished (1200 ms after saccade completion).

(f) Localization trials

A blue fixation square (0.55 × 0.55°; presented 6.5° left from the centre) indicated a localization trial (figure 1*b*, localization task). Subjects were instructed to fixate the fixation square during the whole trial to prevent motor influences. Subjects started the trial by pressing the spacebar. A red target with the same physical characteristics and the same screen coordinates as in saccade trials was flashed for 24 ms. Simultaneously to the disappearance of the target, a red square with the same physical properties as the target appeared in the lower right screen corner. Subjects were instructed to match the location of the mouse target with the location of the flashed target and confirm their answer via mouse click. A localization trial lasted around 3000 ms.

(g) 'No retinal error' experiment (experiment 1)

The 'no retinal error' trials started as the saccadic adaptation trials. Instead of displacing the saccade target outwards, we predicted the eye landing position online and presented the target at the predicted position (see [31]; figure 1*c*, upper panel). Saccade velocity was calculated online. When saccade velocity fell below 30° s^{-1} in three consecutive samples, we selected the current gaze position as the prediction of the saccadic landing point. The mean error between the predicted landing position and the actual landing position resulted over all trials and subjects in 0.19° (s.d. = 0.32°). As described before, 'no retinal error' trials were intermingled with localization trials every 12, 25 or 50 trials depending on the session tested.

(h) 'No prediction error' experiment (experiment 2)

The 'no prediction error' experiment is identical to the 'no retinal error' experiment (experiment 1), except that instead of setting the post-saccadic retinal error to zero, we mimicked the retinal error observed in the baseline trials. We used the mean error of each subject in their baseline saccade trials (difference between the saccade landing position and actual target position) and added this error to the predicted gaze landing position (see [31]; figure 1*c*, middle panel). Prediction errors contain a certain variance since motor execution noise cannot be precisely foreseen by the sensorimotor system. However, on average this noise should cancel out. The mean baseline prediction error over all conditions was 2.03° (s.d. = 0.44°), while the mean error between prediction and actual landing was 0.19° (s.d. = 0.15°). As for the 'no retinal error' experiment (experiment 1), 20 localization trials were intermingled with 12, 25 or 50 'no prediction error' trials, depending on the condition.

(i) 'Constant error' experiment (experiment 3)

In 'constant error' trials, we used a target displacement that was clamped to the end point of the eye movement [31]. The saccade landing position was predicted online (see section 'no retinal error' trials), and the saccade target was displaced 3° to the right of the predicted saccade landing position (figure 1*c*, lower panel). The trial structure was the same as in the previous two experiments, but only one condition (long error trials) was tested. 'Constant error' trials were intermingled with localization trials after saccade trials were completed. Over all subjects, the mean error between prediction and actual landing was 0.19° (s.d. = 0.17°).

(j) Target displacements

We checked the timing of the target displacement relative to saccade performance in an offline analysis. Independent of the condition, displacements were presented well within the period of saccade execution: over all blocks, displacements took place on average 45.6 ms (± 3.4) after the saccade onset (on average saccades lasted approximatively 54.1 ms (± 5.9)) in experiment 1; 39.7 ms (± 4.3) after the saccade onset (on average saccades lasted 51.1 ms (± 2.4)) in experiment 2 and 42.1 ms (± 3.8) after the saccade onset (on average saccades lasted approximatively 49.2 (± 2.1)) in Exp 3. Moreover, participants were asked to report after the conclusion of the experiment whether they saw a displacement of the target during the session. None of the participants tested reported to have noticed the target moving.

(k) Data analyses

For all three experiments we excluded saccade trials in which the subject blinked during saccade execution or if a saccade was initiated before the fixation target disappeared (anticipatory saccades). In order to check for saccades occurring soon after the disappearance of the target, saccade latency was calculated as time from target offset to saccade onset: in less than 1.5% of the valid trials, saccade latencies were shorter than 80 ms. Since their very rare occurrence, these trials were not excluded from the analyses. We excluded trials in which the saccade landing position was smaller than 3.5° or bigger than 9.5°. For the localization trials, trials were excluded in which a perceived localization was smaller than 3.5° or bigger than 9.5°. Additionally, we investigated if the subject fixated during the localization task. If they dissolved fixation during the target flash or during their response of the perceived localization, these trials were excluded from the data analyses. On average, a participant was included in the analyses if at least the 60% of trials was considered valid. Saccadic gain, expressed as the ratio of the saccade landing position over the target position, was calculated for each trial, as well as localization gain (cursor position divided by target position). To compare localization changes over the course of conditions, we calculated the deviation of each localization block from the pre-adaptation baseline trials as the difference between mean localization in the baseline trials and mean localization in each of the three blocks

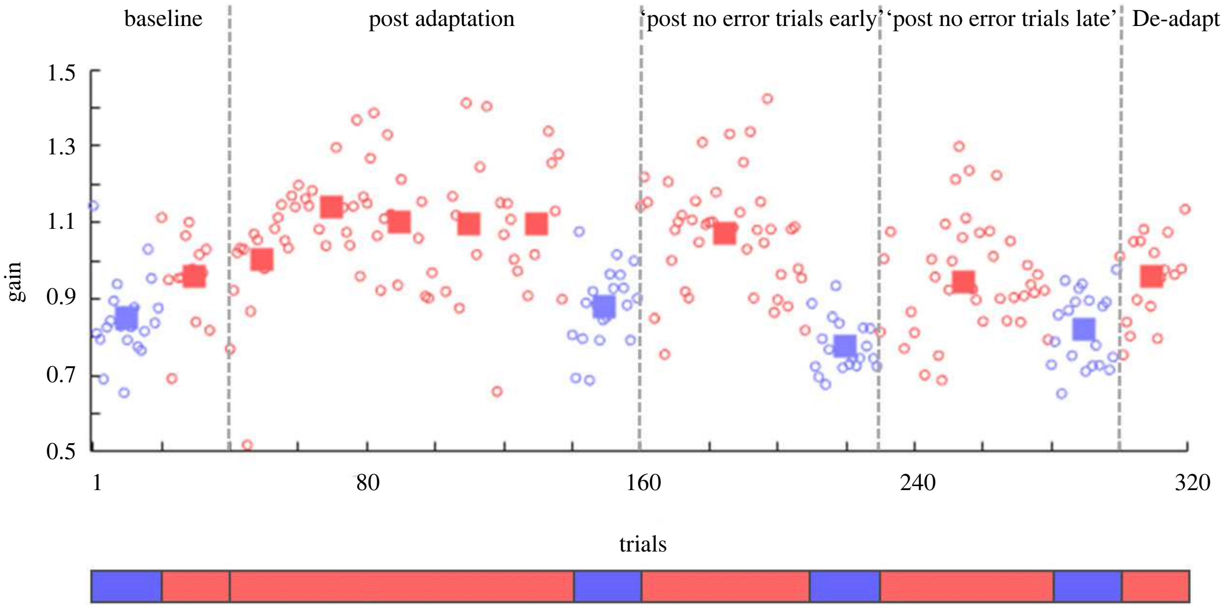

Figure 2. Saccadic and localization gain as a function of trial number for one subject in the long 'no retinal error' condition (experiment 1). A gain of 1 indicates no difference between the eye/mouse position and the target. Circles represent single data points (saccade in red, localization in blue). Rectangles in baseline, adaptation and de-adaptation symbolize the mean over 20 trials, with the exception of red rectangles in 'post no error trials early' and 'post no error trials late' which are the mean over the trial length of the condition (here: bins of 50 trials). The subject first performed 20 trials of baseline localization, followed by 20 saccade baseline trials (baseline). Hundred trials of saccadic adaptation then started, followed by a localization block of 20 trials (post-adaptation). Thereafter, 50 'no retinal error' trials and 20 localization trials were conducted (post no error trials early). This was followed by another block of 50 'no retinal error' and 20 localization trials (post no error trials late). Lastly, 20 de-adaptation trials were performed (de-adapt).

of visual localization. Saccadic landing position changes were obtained, similarly, by computing the difference between subjects' performance in baseline trials and the last 10 valid trials of each adaptation blocks, in order to reduce the temporal offset between saccade and localization. In both cases, positive values indicate an outward shift, while values that fall close to zero indicate no effect of adaptation.

We calculated non-parametric repeated-measures ANOVAs, entailing aligned rank transforms for nonparametric factorial data (ARTool [32]; version 2.1.2) for both, the 'no retinal error' experiment (experiment 1) and the 'no prediction error' experiment (experiment 2).

First, we analysed if saccade landing positions or target localization adapted outward. We, therefore, performed a 2 × 3 non-parametric repeated-measures ANOVA with factors 'block' (baseline, manipulation) and 'condition' (short, medium, long) on either saccade landing positions or target localizations.

Second, we analysed if saccade and visual localization performance differ from each other. We performed a 2 × 3 non-parametric repeated-measures ANOVA with factors 'task' (saccade, localization) and 'condition' (short, medium, long) on the mean saccade adaptation effect (saccade landing positions in the current block minus saccade baseline) and the mean localization adaptation effect (target localization in the current block minus target localization baseline).

Students t-tests against zero on the mean saccade and localization adaptation effect were conducted for the 'constant error' experiment (experiment 3).

3. Results

To investigate if visual and saccadic space is processed by shared or separate resources, we inserted 'no error' trials and observed how those affected saccade adaptation and visual mislocalization. Figure 2 shows saccadic and target localization data for one example participant, tested in the long 'no retinal error' experiment (experiment 1). Saccade gain (red dots) changed from 0.96 (± 0.12) in the pre-adaptation baseline trials to 1.08 (± 0.16) over the course of the adaptation period. However, saccadic gain did not remain stationary. Rather, it slowly decayed starting from the 'post no error trials early' (1.07 ± 0.15) to the 'post no error trials late' (0.95 ± 0.16) up to the very last block (0.95 ± 0.11), where the participant was brought back to its initial de-adapted state.

After completing each block of saccade adaptation, participants localized with the screen cursor the position of a brief flashed target presented at 6.5° to the right of the centre of the screen. Similar to saccades, mean localization gain (blue dots) changed from 0.85 in baseline trials (± 0.11) to 0.88 (± 0.10) after the post-adaptation, and from 0.78 (± 0.07) to 0.82 (± 0.08) respectively after saccade blocks of 'post no error trials early' and 'post no error trials late'.

Saccade adaptation was elicited in post-adaptation by an intrasaccadic target displacement paradigm: Participants performed saccades to a target which was presented at 6.5° to the right of the centre of the screen and always displaced by 3° outward (to 9.5°) once a saccade has been detected. Figure 3 plots the amount of adaptation (difference in saccade landing position relative to the baseline landing position, red dots) and the amount of mislocalization (difference in localization position relative to the baseline localization position, blue dots) averaged over subjects for the three condition types, separately for the 'no retinal error' experiment (experiment 1), the 'no prediction error' experiment (experiment 2) and the 'constant error' experiment (experiment 3). Short (12 trials), medium (25 trials) and long (50 trials) labels in figure 3 refer to the number of trials in which the motor system received an error; however, data shown in figure 3 are relative to the trials before this manipulation was applied.

Modification of saccadic amplitudes with this adaptation method produced a clear shift in gaze positions. In all three

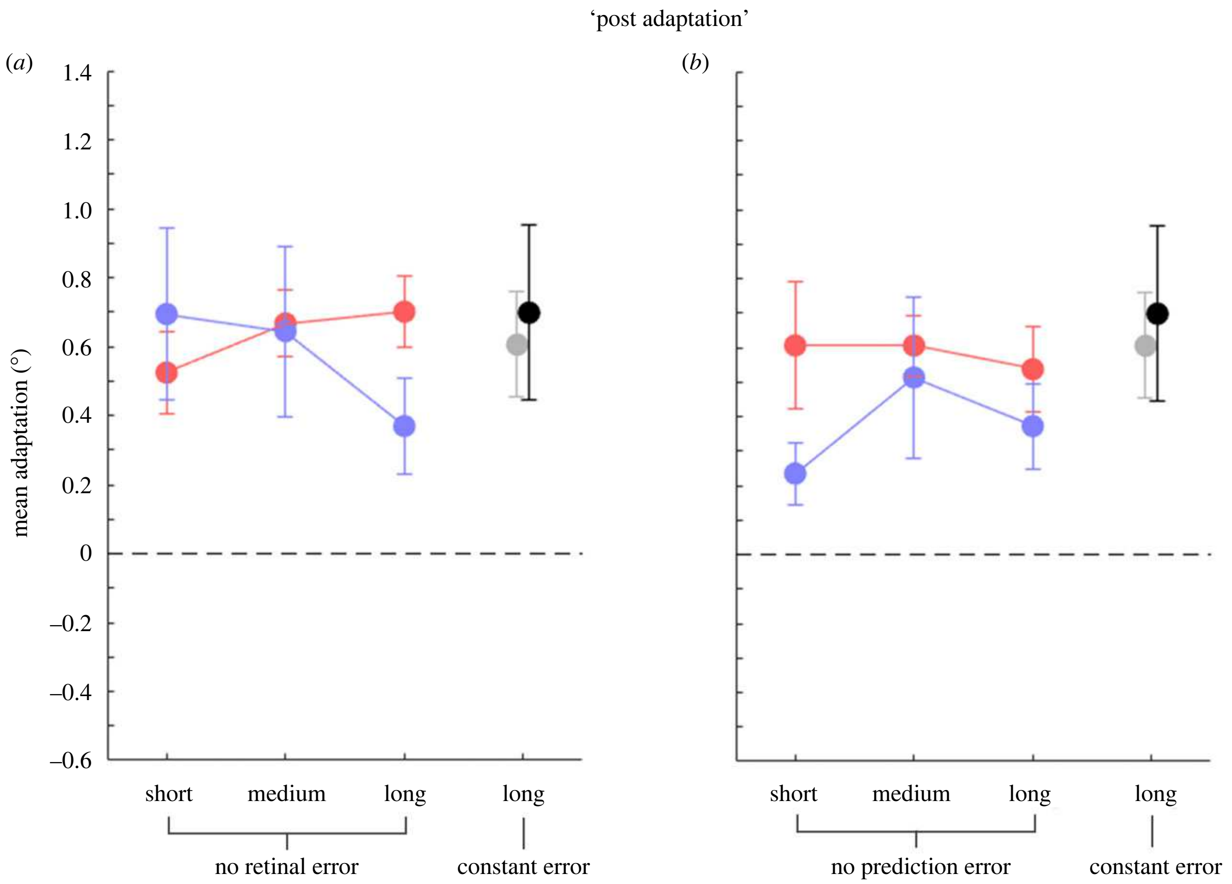

Figure 3. Mean adaptation (°) for saccade (red) and the following localization trials (blue) over subjects in the post-adaptation trials, before the 'no error trials' were performed: (*a*) 'no retinal error' trials; (*b*) 'no prediction error' trials. Mean saccade adaptation in the 'constant error' condition (experiment 3) is shown in grey, corresponding localization trials in black. The dotted line indicates a null effect of adaptation. Positive values indicate an outward shift. Error bars are standard errors of the mean. Modification of saccade amplitudes via saccade adaptation resulted in a positive shift in the direction of the target displacement, which was followed by visual localization.

experiments, a 2 × 3 non-parametric repeated-measures ANOVA with the factors 'block' (baseline, manipulation) and 'condition' (short, medium, long) on the saccade landing positions revealed a main effect of factor 'block' ('no retinal error' experiment [experiment 1]: $F_{1,8} = 69.16$, $p < 0.001$; 'no prediction error' experiment [experiment 2]: $F_{1,8} = 43.39$, $p < 0.001$). Therefore, we found large changes in saccade vectors for the 'post-adaptation' block. For the 'constant error' experiment (experiment 3), on which 7 subjects were tested, a Student's *t*-test against zero on the saccade landing positions also revealed increased saccade vectors, $t_6 = 4.26$, $p = 0.005$.

Moreover, saccadic adaptation affected visual localization, leading to an outward shift of the perceived target position in space, as revealed by a 2 × 3 non-parametric repeated-measures ANOVA with the factors 'block' (baseline, manipulation) and 'condition' (short, medium, long); ('no retinal error' experiment [experiment 1]: $F_{1,8} = 20.51$, $p = 0.002$; 'no prediction error' experiment [experiment 2]: $F_{1,8} = 13.86$, $p = 0.006$).

Again, for the 'constant error' experiment (experiment 3), a Student's *t*-test against zero on the target localization revealed outward shifted perceived localization, $t_6 = 3.25$, $p = 0.018$).

These results show that modifications of saccadic amplitude by saccadic adaptation are paralleled by associated changes in visual localization, consistent with the idea of a common manipulation of motor and perception [23–26].

A key question for our study is how the adapted state of saccades and of the following mislocalization develops when no post-saccadic error is provided anymore. Figure 4 plots the mean adaptation magnitude for trials following the 'no error' trials (post no error trials early), for modified saccade vectors (in red) and target localization (in blue), as a function of the saccade trials length. In both experiments, a 2 × 3 non-parametric repeated-measures ANOVA with the factors 'block' (baseline, manipulation) and 'condition' (short, medium, long) on the saccade landing positions revealed that saccades stayed adapted, irrespective of the trial length and of the error variations applied (significant main effect of factor 'block'; 'No retinal error' experiment [experiment 1]: $F_{1,8} = 14.87$, $p = 0.005$; 'No prediction error' experiment [experiment 2]: $F_{1,8} = 23.31$, $p = 0.001$).

Moreover, a 2 × 3 non-parametric repeated-measures ANOVA with the factors 'task' (saccade, localization) and 'condition' (short, medium, long) on the mean saccade or localization adaptation effect revealed that, after performing saccades without a retinal error (experiment 1; figure 4*a*), mislocalization shifts occur as a function of the number of 'no error' trials (significant interaction between factor 'task' and 'condition'), $F_{2,16} = 4.51$, $p = 0.028$. Indeed, applying 50 'no error' trials, a shift of localization in foveal direction was found, confirming previous results [18].

Visual mislocalization performance in the 'no prediction error' experiment (experiment 2; figure 4*b*), however, diverged from saccade adaptation, irrespective of the condition tested ($F_{1,8} = 25.80$, $p = 0.001$), as a significant main effect of factor 'task' in a 2 × 3 non-parametric repeated-measures ANOVA with the factors 'task' (saccade, localization) and 'condition' (short, medium, long) on the mean saccade or localization adaptation effect revealed. This result shows no significant

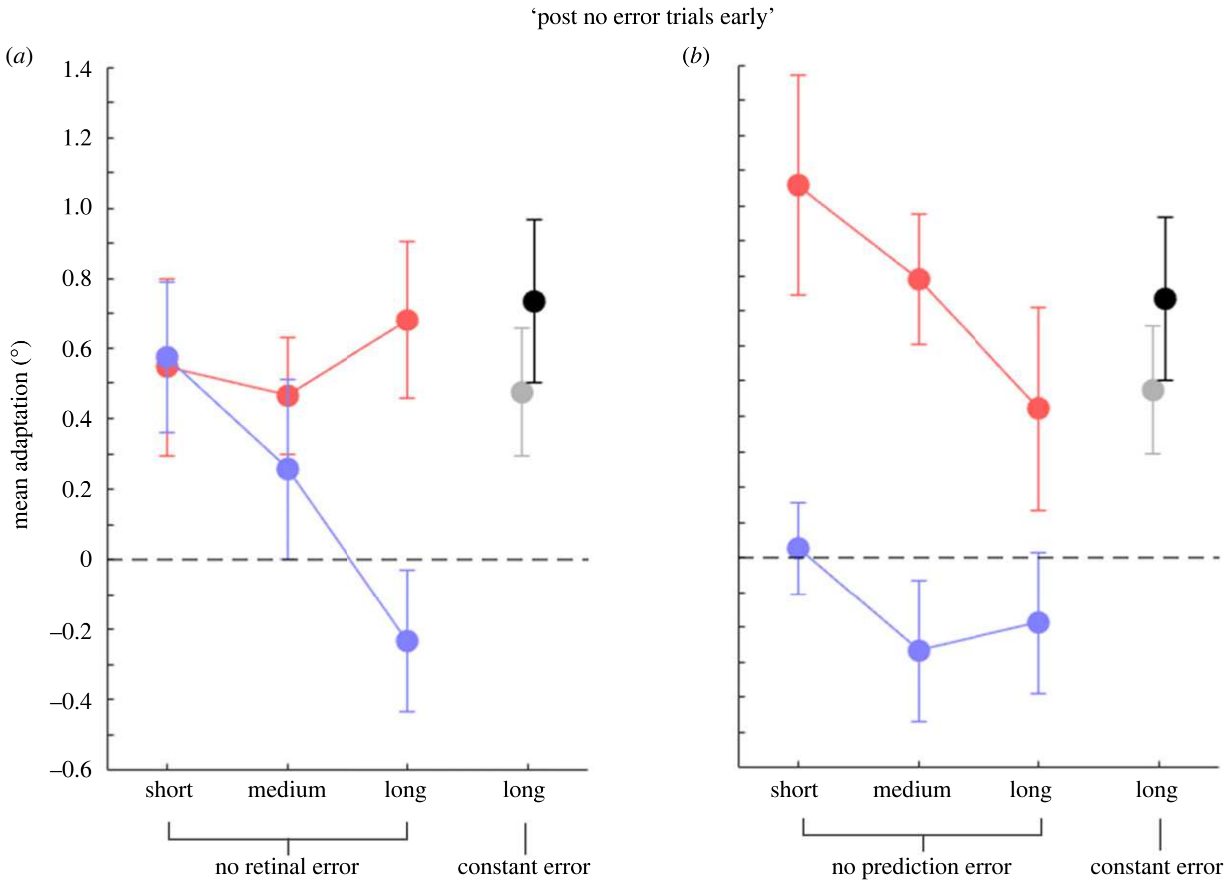

Figure 4. Mean saccade and localization adaptation (°) for 'post no error trials early' in the three conditions tested. (*a*) Mean saccade (red dots) adaptation and visual localization (blue dots) in the 'no retinal error' trials experiment (experiment 1). (*b*) The same convention as in (*a*), for trials in the 'no prediction error' experiment (experiment 2). Grey and black circles represent the mean saccade adaptation and visual localization, respectively. Error bars are standard errors of the mean. The dotted line indicates a null effect of adaptation.

localization shifts in the direction of the adapted saccades, independently of the condition (no significant main effect of factor 'condition'; $F_{2,16} = 1.34$, $p = 0.287$; 2 × 3 non-parametric repeated-measures ANOVA with the factors 'block' (baseline, manipulation) and 'condition' (short, medium, long) on the target localization).

Moreover, when a constant error was provided (experiment 3; long 'constant error' condition) visual mislocalization (black dot) developed very similar to saccade adaptation (grey dot): the constant post-saccadic error method succeeded in inducing saccade adaptation ($t_6 = 2.81$, $p = 0.031$), which was accompanied by mislocalization in favour of an outward displacement ($t_6 = 3.40$, $p = 0.015$).

We additionally compared the amount of mislocalization evoked by the 'post no error trials early' in the 'no retinal error' experiment (experiment 1) and the 'no prediction error' experiment (experiment 2). Shifts in localization depended on the nature of the error signal. Indeed, a 2 × 3 non-parametric between-subjects ANOVA with the factors 'experiment' (no retinal error, no prediction error) and 'condition' (short, medium, long) on the mean localization adaptation effect showed a main effect of factor 'experiment' ($F_{1,8} = 7.35$, $p = 0.027$). The absence of retinal error during saccade execution led, therefore, to a higher recalibration of visual space compared to when 'no prediction error' was provided. Moreover, the absence of prediction error caused a decay in visual localization just after 12 no prediction error trials. Therefore, both errors were equally effective in inducing shifts in saccade amplitudes but it appears that annulling the retinal error was more powerful in evoking visual perception changes, which remained significant up to 50 trials no error trials.

In order to quantify how the 'no error' trials affected visual localization, we calculated the difference between localization in the 'post-adaptation' trials and 'post no error trials early' for all three experiments and all conditions (short, medium, long). A 2 × 3 non-parametric repeated-measure ANOVA with the factors 'experiment' (no retinal error, no prediction error) and 'condition' (short, medium, long) revealed a significant main effect for the factor 'condition' ($F_{2,16} = 7.44$, $p = 0.005$) and no significant main effect of factor 'experiment' ($F_{1,8} = 0.59$, $p = 0.466$) nor a significant interaction ($F_{2,16} = 1.47$, $p = 0.26$). In experiment 3, where a constant post-saccadic error is applied, the difference in localization did not differ significantly from zero ($t_6 = 0.71$, $p = 0.506$), indicating that in both blocks, localizations in the 'constant error' experiment were outward adapted.

We then investigated mean adaptation magnitude for trials at the very end of the condition ('post no error trials late'; figure 5). A 2 × 3 non-parametric repeated-measure ANOVA with the factors 'block' (baseline, manipulation) and condition (short, medium, long) revealed no significant saccade or localization adaptation anymore (no significant factor 'block') in the 'no retinal error' condition (experiment 1; figure 5*a*; saccades: $F_{1,8} = 2.09$, $p = 0.187$; localization: $F_{1,8} = 0.87$, $p = 0.374$) but only adapted saccades in the 'no prediction error' condition (saccades: $F_{1,8} = 16.93$, $p = 0.003$; localization: $F_{1,8} = 1.14$, $p = 0.317$) and a significant difference in the saccade and localization landing positions in

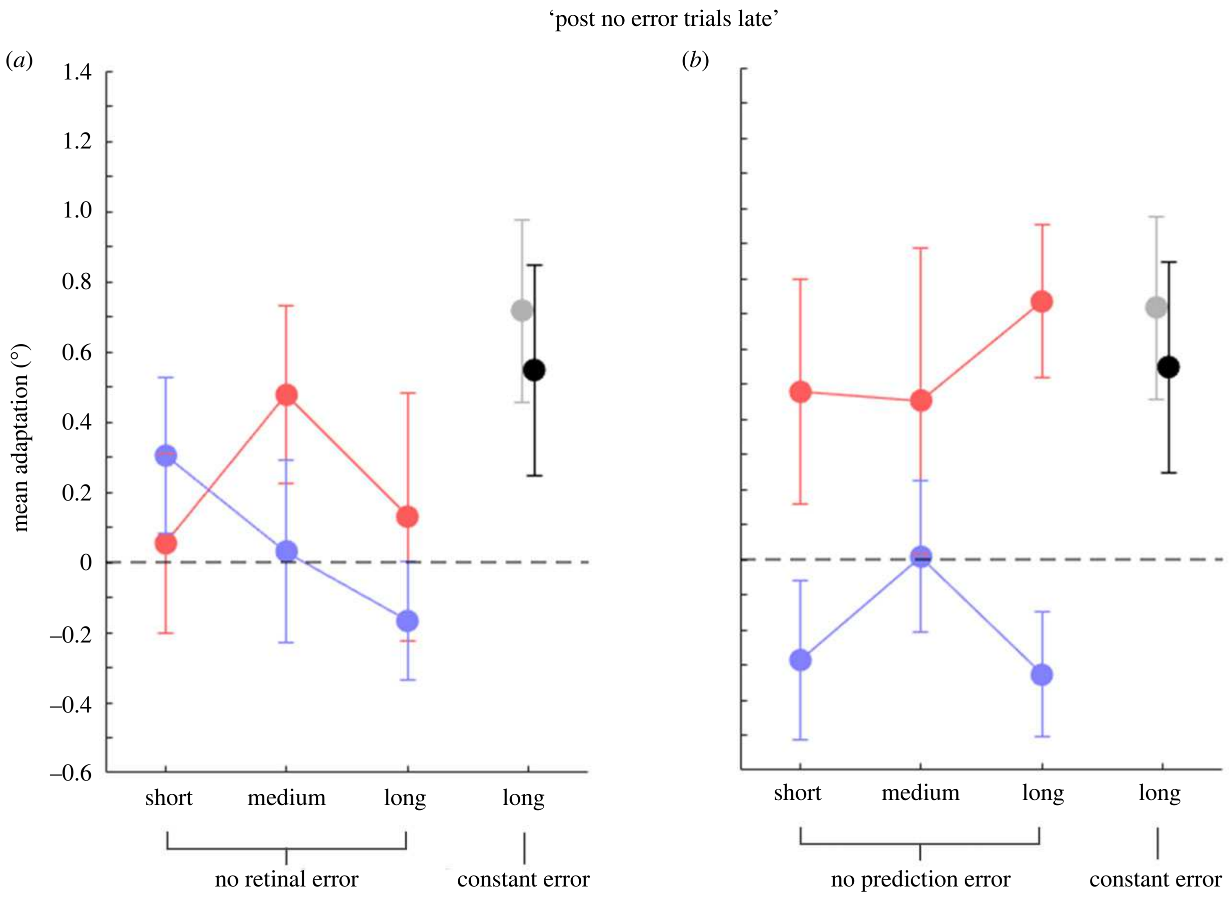

Figure 5. Mean saccade and localization adaptation (°) for 'post no error trials late' in the three conditions tested. (*a*) Mean saccade (red dots) adaptation and visual localization (blue dots) in the 'no retinal error' experiment (experiment 1). (*b*) Same convention as in (*a*), for trials in the 'no prediction error' experiment (experiment 2). Grey and black circles represent the mean saccade adaptation and visual localization, respectively, of experiment 3. Error bars are standard errors of the mean. The dotted line indicates a null effect of adaptation, while dots falling below the dotted lines indicates a shift in the opposite direction on the target.

experiment 2 (figure 5*b*; $F_{1,8} = 10.41$, $p = 0.012$). Applying a constant post-saccadic error modified saccade vectors ($t_6 = 2.98$, $p = 0.025$) while localization vectors showed no outward modification anymore ($t_6 = 1.97$, $p = 0.097$).

4. Discussion

In the present study, we present evidence that saccade adaptation modifies saccadic and visual space separately. In our experiments, various amounts of saccadic error-clamp trials, in which we artificially abolished the post-saccadic error, were followed by trials in which participants localized a visual target while maintaining ocular fixation. While saccade adaptation remained at a steady state level across, visual mislocalization decreased to the baseline level.

The established model of saccade generation involves a motor control architecture in which an inverse model computes the saccade plan and a forward model predicts the sensory consequences following saccade execution. Several studies have reported that consistent with this scheme, saccade amplitudes adapt when the sensorimotor system detects a spatial mismatch between the predicted and the actual saccade landing [9–11]. In a laboratory setting, which usually reduces the visual scene to a saccade target, the prediction of the sensory consequences following a saccade consists in the spatial location of the post-saccadic target. In our baseline trials we confirmed the well-known undershoot in saccade landing positions (e.g. [3–5]). Our target displacement in outward direction increased this undershoot, thus urging the sensorimotor system into an adaptive increase of saccade amplitudes.

Many studies have shown that saccade adaptation changes space perception [23–26]. Motor signals are thus involved in the construction of visual space. In the shared resource model, the metric of visual space could derive directly from motor structures, such that both, action and perception, rely on same spatial coordinates. In this view, any change in the motor adaptation must be likewise reflected in visual space. Alternatively, resources for saccade and visual space might be recalibrated by the same post-saccadic error signal but processed in separate areas in the brain.

In order to decide between the two models, we applied two major manipulations. First, before measuring visual localization, we varied the number of trials with either no retinal or no prediction error. In our previous study [18], we found that visual space compresses toward the fovea if deprived of recalibration by post-saccadic errors. If visual localization would reveal compression to the fovea following the 'no error' trials while saccades maintain adaptation, clear evidence for a dissociation between saccade and visual space would be found. Such a dissociation would rule out the shared resource model and favour the model involving separate resources for visual and motor space. Second, we contrasted the influence of the two different sources of error information on visual localization, i.e. the retinal error (distance between the saccade landing position and the visual target), and the prediction error (difference between the predicted post-saccadic retinal error and the observed post-saccadic retinal error).

Our data clearly speak in favour of the model positing separate resources for saccade and visual space. In all conditions, we found significant changes in visual localization when measured directly after saccade adaptation trials. However, when we measured localization after 'no error' trials had been presented, we found that mislocalization strength depended on the persistence (i.e. the number of 'no error' trials): while a small number of 'no error' trials did not change mislocalization magnitude, increasing the number led to a disappearance of mislocalization. In conditions with the highest number of 'no error' trials we even found that localization drifted toward the fovea, in agreement with previous results [18]. These changes in localization demonstrate a dissociation between motor and visual targeting because saccade adaptation remained unchanged by the insertion of the 'no error' trials. In line with this dissociation, a recent study found significant mislocalization even though subjects, following the instructions, successfully inhibited saccade adaptation [29]. Moreover, evidence of a dissociation between motor and visual targeting have been shown also for saccade directed to moving targets (instead of static targets as in this study), in which the difference between perception and action is suggested to rely on an accumulation of position error over the temporal integration window of motion and position signals, which is much reduced in the motor system [33,34].

Electrophysiological work has demonstrated that the parietal cortex contains a representation of post-saccadic error [20]. It is thus likely that the parietal cortex processes the post-saccadic error in order to recalibrate both, saccade and visual space. The involvement of the parietal cortex in the construction of visual space [22] and in saccade generation [35] and adaptation [21] is well documented. Our data exclude that visual recalibration by the post-saccadic error occurs in early visual areas. If visual recalibration would occur in early visual areas, the changes in saccade amplitudes that we observed would actually be visual in nature. Since the magnitude of visual and saccadic adaptation was identical, it would be impossible under this model that visual mislocalization decays while saccadic adaptation remains stable. However, the latter scenario describes exactly what we found in the present study.

Multiple processes contribute to learning in motor adaptation [36]. Studies suggest that at least two components control learning: an initial process that learns fast and decays quickly and a more gradual process that adapts slow and has a long retention [37,38]. Summing learning from both components yields the final adaptation magnitude. In our study, visual recalibration decayed more quickly than motor recalibration. Since faster adaptive learning is connected to a shorter retention (i.e. a quicker decay), it would be interesting to find out if also recalibration learning is faster for vision than for motor. However, it is unlikely that our data are due to separate components of a single learning process. Since visual and motor changes are of equal strength, when measured immediately after adaptation, the only option remains that saccadic adaptation would be purely visual. However, as argued above, this interpretation must be rejected because motor adaptation was observed when visual mislocalization returned to the baseline level.

In conclusion, our data suggest that resources of motor and visual space are separate and that the post-saccadic error recalibrates the metric of both, saccades and visual localization.

Ethics. Participants gave informed consent prior to participation. Experimental procedures were approved by the local ethics committee of the mathematics and natural sciences faculty of the Heinrich-Heine University Düsseldorf (ethics approval number: ZI01-2021-01). Written informed consent was obtained prior to the experiments in accordance with the declaration of Helsinki.

Data accessibility. Data used for this publication have been deposited into the Dryad Digital Repository: https://doi.org/10.5061/dryad.rbnzs7hft [39].

Authors' contributions. S.T.: conceptualization, formal analysis, investigation, methodology, visualization, writing—original draft; A.P.: conceptualization, formal analysis, funding acquisition, investigation, methodology, supervision, validation, visualization, writing—review and editing; E.Z.: conceptualization, funding acquisition, investigation, methodology, project administration, resources, supervision, validation, visualization, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

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Serial dependencies in motor targeting as a function of target appearance

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In order to bring stimuli of interest into our central field of vision, we perform saccadic eye movements. After every saccade, the error between the predicted and actual landing position is monitored. In the laboratory, artificial post-saccadic errors are created by displacing the target during saccade execution. Previous research found that even a single post-saccadic error induces immediate amplitude changes to minimize that error. The saccadic amplitude adjustment could result from a recalibration of the saccade target representation. We asked if recalibration follows an integration scheme in which the impact magnitude of the previous post-saccadic target location depends on the certainty of the current target. We asked subjects to perform saccades to Gaussian blobs as targets, the visuospatial certainty of which we manipulated by changing its spatial constant. In separate sessions, either the pre-saccadic or post-saccadic target was uncertain. Additionally, we manipulated the contrast to further decrease certainty, changing the spatial constant mid-saccade. We found saccade-by-saccade amplitude reductions only with a currently uncertain target, a previously certain one, and a constant target contrast. We conclude that the features of the pre-saccadic target (i.e., size and contrast) determine the extent to which post-saccadic error shapes upcoming saccade amplitudes.

Introduction

When we gaze around in the environment, we perform saccade eye movements. Saccades are fast displacement of the eye ball whose function it is to bring visual objects of interest onto the region of highest retinal resolution (i.e., the fovea). Because rapid reception of sensory information is of survival value, performing accurate saccades is mandatory.

Inaccuracies in saccade landing can result from three sources, as the visual localization, motor localization of the target, or execution of the saccade might be inappropriate. After the saccade is finished, the error between saccade landing and the target reveals the movement success. The sensorimotor system monitors its performance and aims to minimize the error in saccade landing (Pélisson, Alahyane, Panouillères, & Tilikete, 2010). Oculomotor plasticity can be studied in the laboratory with the paradigm of saccadic adaptation, in which a saccade target is displaced while the eye is on flight (Hopp & Fuchs, 2004; McLaughlin, 1967; Pélisson et al., 2010). Due to visual transduction latencies, the saccade cannot be corrected online. Instead, after registering the post-saccadic error, the sensorimotor system triggers a corrective saccade to reach the desired target location (Sedaghat-Nejad & Shadmehr, 2021). Every experience of a post-saccadic error is followed by an adaptive change in the amplitude of the immediately following saccade. If the same artificial post-saccadic error is repeatedly presented, the adaptive amplitude change increases gradually until it reaches an asymptotic value (Noto & Robinson, 2001; Wallman & Fuchs, 1998). Maximal saccade adaptation minimizes about three quarter of the post-saccade error (Gillen, Weiler, & Heath, 2013; Ohl, Brandt, & Kliegl, 2011). In most experiments on saccade adaptation, the target is displaced in the same direction and distance. Only a few studies have investigated adaptive amplitude changes when the direction and distance of the target jump were determined randomly in every trial (Collins, 2014; Desmurget et al., 2000; Havermann & Lappe, 2010; Srimal, Diedrichsen, Ryklin, & Curtis, 2008). These studies found consistently that adaptive amplitude changes occur on the single-saccade level. Assessing the functional role of saccade adaptation requires knowing why post-saccadic errors accrue in natural vision. A putative source of post-saccadic errors would be eye muscle damage or fatigue (Abel, Schmidt, Dell'Osso, & Daroff, 1978; Kommerell, Olivier, & Theopold, 1976; Optican, Zee, & Chu, 1985). Because such changes would alter saccade dynamics

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permanently and thus produce constant post-saccadic errors, accumulating saccade adaptation would provide the countermeasure. Much more probable in natural vision, however, are inaccuracies in visual or saccadic targeting.

Visual estimates of object features are constantly relying on sensory input of the recent past. Serial dependencies are attractive biases toward similar stimuli previously experienced and have been observed in actions, perception, decisions, and memory (Cicchini, Mikellidou, & Burr, 2024; Manassi & Whitney, 2024). The first studies on serial dependencies used either visual orientation (Fischer & Whitney, 2014) or numerosity (Cicchini, Anobile, & Burr, 2014) as stimuli. However, serial dependencies have been reported for almost all visual features, such as luminance (Fründ, Wichmann, & Macke, 2014), orientation (Alais, Leung, & Van der Burg, 2017; Collins, 2019; Fischer & Whitney, 2014; Fritsche & de Lange, 2019; Fritsche, Mostert, & de Lange, 2017; Murai & Whitney, 2021; Pascucci et al., 2019; Rafiei, Hansmann-Roth, Whitney, Kristjansson, & Chetverikov, 2021; Tanrikulu, Pascucci, & Kristjánsson, 2023), color (Barbosa & Compte, 2020; Bays, Catalao, & Husain, 2009; Foster, Bsales, Jaffe, & Awh, 2017; Oberauer & Lin, 2017; van den Berg, Shin, Chou, George, & Ma, 2012), and shape (Collins, 2022; Manassi et al., 2021; Manassi, Kristjánsson, & Whitney, 2019).

Manassi, Liberman, Kosovicheva, Zhang, and Whitney (2018) showed that even spatial localization is subject to biases from the recent stimulation history. Subjects were required to localize objects in space, and their estimate shifted to the direction in which objects were previously encountered. We have shown that such dependencies also exist between saccade targeting and visual space. We found that artificial post-saccadic errors in the preceding trials modify visual target localization in the current trial (Cont & Zimmermann, 2021). Participants had to perform a saccade in the previous trial (Trial$_{n-1}$), and the saccade target was displaced during saccade execution. In the next trial (Trial$_n$), subjects had to fixate and localize a briefly flashed target with the mouse pointer. Visual localizations were shifted in the direction of the previous post-saccadic target. Do these interactions between post-saccadic errors and visual and motor localization imply that visual and motor space relies on a shared resource? In a follow-up study, we first induced saccade adaptation. After adaptation was established, we clamped the post-saccadic error online to the predicted endpoints of saccades, effectively annulling the error (Tyralla, Pomè, & Zimmermann, 2023). Although saccade motor adaptation remained undisturbed by the experiences of zero post-saccadic error, visual adaptation–induced mislocalization gradually declined. A shared resource of visual and motor space would have dictated that motor and visual localization changes concomitantly. However, this was not the case, suggesting that motor errors recalibrate motor and visual space separately.

In the present study, we wondered how the visuospatial certainty of the saccade target would affect adaptive amplitude changes. Souto, Gegenfurtner, and Schütz (2016) measured the effect of uncertainty by using Gaussian blobs as targets for which the spatial constant was varied. They found little correlation between target uncertainty and saccade adaptation rates. Heins, Masselink, Scherer, and Lappe (2023) have shown that saccade adaptation can even be induced without presenting a pre-saccadic target. After training participants to perform a saccade to a visible target, they asked them to perform saccades to a location at which they expected the target to appear. After saccade execution, the target appeared at a position slightly shifted inward. Over the course of trials, saccade amplitudes adapted to the post-saccadic error and became smaller. These data show that the physical presentation of the saccade target might be irrelevant for adaptation to occur as long as an internal prediction about the position of the target exists.

In the current study, we aimed to test the influence of the saccade target visibility on serial dependencies in saccade targeting. We used Gaussian blobs as targets for which the spatial constant was varied. We manipulated target visibility separately for the pre-saccadic and the post-saccadic targets such that either the pre-saccadic or the post-saccadic target had a high spatial constant. We recorded saccade landing positions as a combined measure of perceptual and motor localization. The manipulation could affect either the perceptual localization of the target or the motor error correction. If the manipulation would affect perceptual localization, serial dependencies should be stronger if the spatial constant of the pre-saccadic target is high. Because such a target is unfocused and therefore more difficult to localize, the visual system should rely on past stimulations when estimating its position. If the manipulation would affect motor targeting, serial dependencies should decrease when the post-saccadic target has a high spatial constant. In that case the post-saccadic error should be less visible, thus inducing less amplitude change of the upcoming saccade.

Methods

Participants

Twenty-two subjects (mean ± *SD* age, 22 ± 2.99 years; 14 women) participated in the first experiment ("constant contrast" experiment) in three different sessions. Twenty-two different subjects (mean ± *SD* age, 23.50 ± 4.28 years; 17 women) took part in the second experiment ("adjusted contrast" experiment), again in three different sessions. Participants were native German speakers, reported to have normal vision or wore lenses during the experiment, and indicated no psychiatric or neurological diseases. Participants were recruited at the Heinrich-Heine University Düsseldorf. Experimental procedures were approved by the local ethics committee of the mathematics and natural sciences faculty of the Heinrich-Heine-University Düsseldorf (approval no. ZI01-2021-01). Written informed consent was given prior to the experiments in accordance with the tenets of the Declaration of Helsinki. They either received course credits or 10 euros per hour for participation.

Setup

The first experiment ("constant contrast") ran on a Mac Mini (2014; Apple, Cupertino, CA), presented on a cathode-ray tube (CRT) screen (Diamond Pro 2070; 12.9 inches, 800 × 600-pixel resolution, 120-Hz refresh rate; Mitsubishi, Tokyo, Japan). MATLAB R2016b (version 7.10.0; MathWorks, Natick, MA) and Psychtoolbox routines (version 3.0.17) were used for stimulus generation. The second experiment ("adjusted contrast") ran on a Windows 10 computer (Microsoft, Redmond, CA) presented on an Acer XB272 screen (23.6 inches, 1920 × 1080-pixel resolution, 120-Hz refresh rate; Acer, New Taipei City, Taiwan). MATLAB R2020b (version 9.9.0) and Psychtoolbox routines (version 3.0.18) were used for stimulus generation. Subjects were placed 57 cm in front of the screen in a dark room. We used a black background (0.01 cd/m^2). Participants placed their head in a chin rest to prevent head movements. Eye movements were recorded by a desktop-mounted eye tracker (EyeLink 1000 Plus; 1000-Hz sampling rate; SR Research, Ottawa, Canada). Participants performed the task binocularly, but only the left eye was recorded. A standard nine-point calibration routine was conducted. For measuring participants' responses, a standard keyboard and mouse were used.

Structure of trials

We asked subjects to perform a saccade to a target. We manipulated the relative spatial uncertainty of the pre-saccadic target (T1) and the post-saccadic target (T2). Both targets consisted of a two-dimensional (2D) Gaussian blob. T1 was shown before saccade execution. During saccade execution, we displaced the target (post-saccadic target T2). By changing spatial constant of the target intrasaccadically between two values ($\sigma = 0.3°$ and $\sigma = 1.5°$) we aimed to manipulate the spatial certainty of the target. The lower spatial constant (0.3°) resulted in a more focused target that was connected to a higher visuospatial certainty. The higher spatial constant (1.5°) resulted in a broader target that was connected to a lower visuospatial certainty.

We created three session types: (a) both targets were small, (b) T1 was small and T2 was large, or (c) T1 was large and T2 was small. In the offline analysis, we took into account the influence of the previously seen post-saccadic target ($T2_{n-1}$) on the currently visible pre-saccadic target ($T1_n$). For the three sessions, we therefore considered three dependencies: (a) $T2_{n-1}$ small/$T1_n$ small, (b) $T2_{n-1}$ large/$T1_n$ small, and (c) $T2_{n-1}$ small/$T1_n$ large. Each session resulted in 400 trials (duration of 20 minutes each). The session order was randomized across subjects.

In two separate experiments, we varied the contrast of the target (peak luminance of the stimulus divided by maximum luminance the screen can reach) to further modify the spatial certainty of the saccade target. Therefore, our values reflect the percentage of maximum stimulus contrast that the monitor could show. In the "constant contrast" experiment, the same contrast was used for both spatial constant values, thus creating a constant stimulus contrast (contrast, ∼27%; minimum luminance, 0.01 cd/m^2; maximum luminance, 3.2 cd/m^2) that leads to targets with higher spatial constant appearing more luminant. In the "adjusted contrast" experiment, a spatial constant of 0.3° was paired with a higher contrast (contrast, ∼27%), and a spatial constant of 1.5° was paired with a lower contrast (contrast, ∼3%; minimum luminance, 0.01 cd/m^2; maximum luminance, 157.7 cd/m^2) to adjust for the higher spatial constant. Targets with a higher spatial constant then appeared less luminant compared with the "constant contrast" experiment and, therefore, resulted in a more uncertain target.

Experimental procedure

Figure 1A schematically shows the structure of a trial. Each trial began with the presentation of a red fixation square (0.55° × 0.55° diameter) on the horizontal meridian, 6.5° to the left of the screen center. The fixation square disappeared after a random duration between 500 and 1200 ms and, simultaneously, a Gaussian blob (see Figure 1B for specifications) was presented 6.5° to the right of the screen center, serving as saccade target T1. Subjects were instructed to perform a saccade toward saccade target T1 as soon as it appeared. Eye position was recorded by the eye tracker and analyzed online by the stimulus program. As soon as the eye velocity exceeded 30°/s, the target was displaced to a new saccade target position, T2. In

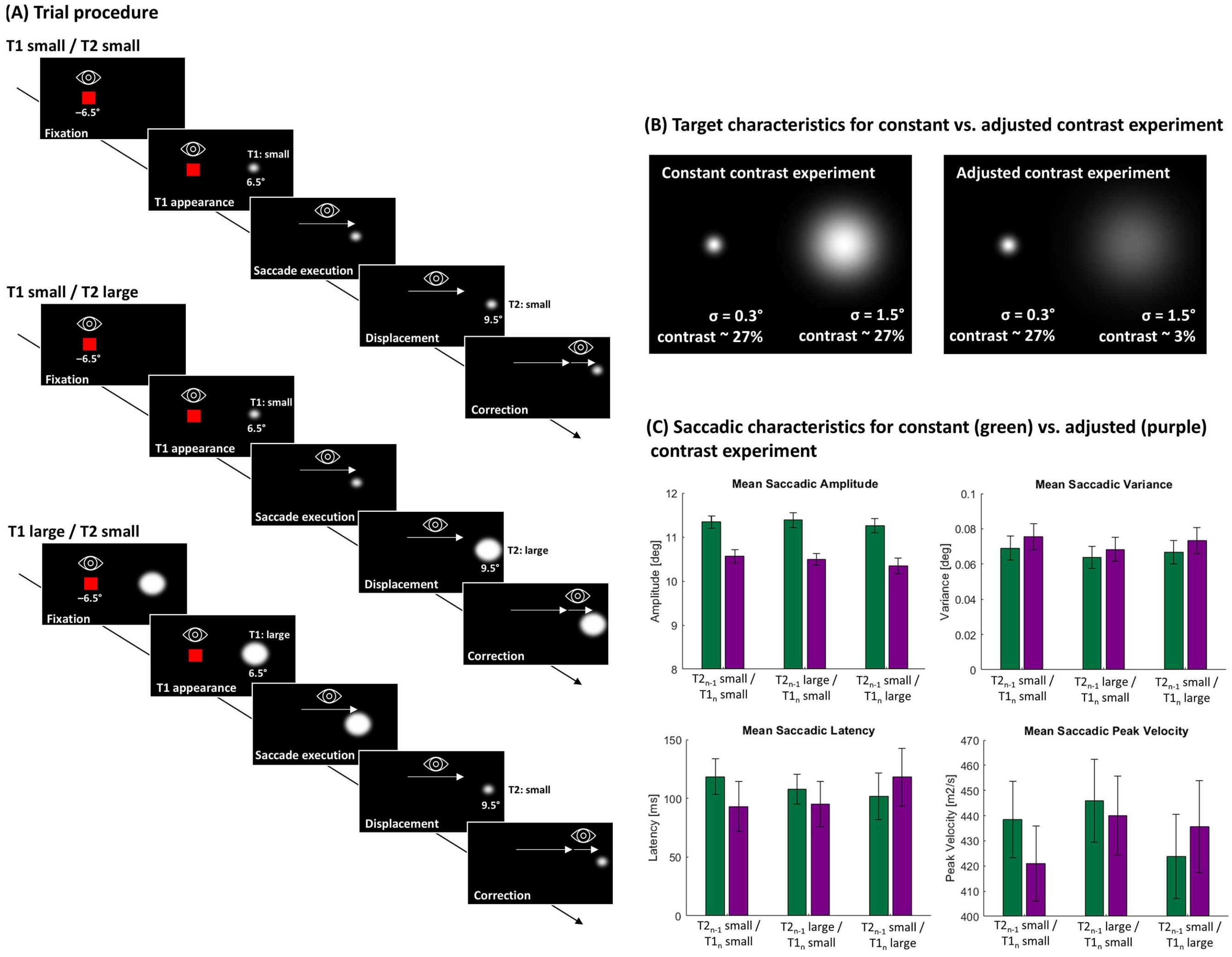

Figure 1. (**A**) Schematic illustration shows the procedure of one trial for each of the three sessions for the "constant contrast" experiment. Subjects performed a saccade toward the target. During saccade execution, the target was displaced in one out of six different locations (−2.5°, −1.5°, −0.5°, 0.5°, 1.5°, 2.5°). In the T1 small/T2 small session, saccadic targets were always indicated by a small diameter. In the T1 small/T2 large session, the initial target showed a small diameter and the displaced target T2 was indicated by a larger diameter. In the T1 large/T2 small session, the target identities were switched. (**B**) The stimulus characteristics for small and large targets for both experiments are specified. (**C**) The saccadic characteristics for saccadic amplitude, variance, latency, and peak velocity were specified for both, the "constant contrast" experiment (green) and the "adjusted contrast" experiment (purple). Error bars represent the standard error of the mean.

each trial, one displacement size and direction were selected equiprobably out of six equidistant steps (−2.5°, −1.5°, −0.5°, 0.5°, 1.5°, 2.5°). The second target disappeared 1200 ms after saccade completion, and a new trial started.

Data analyses

A trial was excluded from the analyses if no saccade was performed, the saccadic amplitude was smaller than half the required distance (i.e., 6.5°) or its peak velocity exceeded 800°/s. This resulted in a trial exclusion of ∼10% per participant. For both experiments, we computed the post-saccadic error for each trial as the difference between the actual target position of T1 (6.5°) and the saccadic amplitude. We performed linear regression analyses to examine the strength between the post-saccadic error in the previous and the current trial in each session. Student's *t*-tests against zero were conducted to investigate serial dependency effects. A one-way ANOVA with the factor session ($T2_{n-1}$ small/$T1_n$ small, $T2_{n-1}$ large/$T1_n$ small, $T2_{n-1}$ small/$T1_n$ large) was calculated for both experiments separately to investigate differences in the strength of trial-by-trial influences.

Results

We manipulated the pre-saccadic and the post-saccadic visuospatial spatial constants of Gaussian blobs that served as saccade targets. By reducing the contrast of the larger target, we aimed to further increase visuospatial uncertainty. Figure 2 shows saccadic amplitudes for one example subject for the “constant contrast” experiment and another example subject for the “adjusted contrast” experiment for each of the three sessions. Subjects were instructed to perform a horizontal saccade of 13° (indicated by the dashed line in all panels) toward the pre-saccadic target (dashed empty circle). We varied the spatial constant of the targets, resulting in a pre-saccadic target of $\sigma = 0.3^\circ$ (followed by a post-saccadic target of $\sigma = 0.3^\circ$; Figure 2, left panel), a pre-saccadic target of $\sigma = 0.3^\circ$ (followed by a post-saccadic target of $\sigma = 1.5^\circ$; Figure 2, middle panel), or a pre-saccadic target of $\sigma = 1.5^\circ$ (followed by a post-saccadic target of $\sigma = 0.3^\circ$; Figure 2, right panel). Both subjects undershot the target systematically, resulting in mean saccadic amplitudes of 10.95° for the first subject and 10.04° for the second subject, regardless of the spatial constant of the pre-saccadic target. We found a stronger saccade undershoot in the “adjusted contrast” experiment, agreeing with the results of Lisi, Solomon, and Morgan (2019) indicating that saccade undershoot magnitude scales with the visuospatial uncertainty of saccade targets.

Post-saccadic serial dependency differences

We calculated the error between the actual target position and the saccadic amplitude. In Figure 3, two example subjects for each session and each experiment are presented to visualize the magnitude of saccade-by-saccade influences. Note that negative numbers indicate an undershoot of saccadic amplitude, whereas positive numbers indicate a saccadic performance overshooting the target. To investigate serial dependencies of the post-saccadic error from the previous trial (Trial$_{n-1}$) to the current trial (Trial$_n$), we fit a linear regression model for each subject in every session and in each experiment separately. More precisely, we took the influence of the previously seen post-saccadic target (T2$_{n-1}$) on the currently visible pre-saccadic target (T1$_n$) into account. Therefore, post-saccadic target T2 in the current trial (T2$_n$) is irrelevant for the current saccadic performance: T2$_n$ is presented during saccade

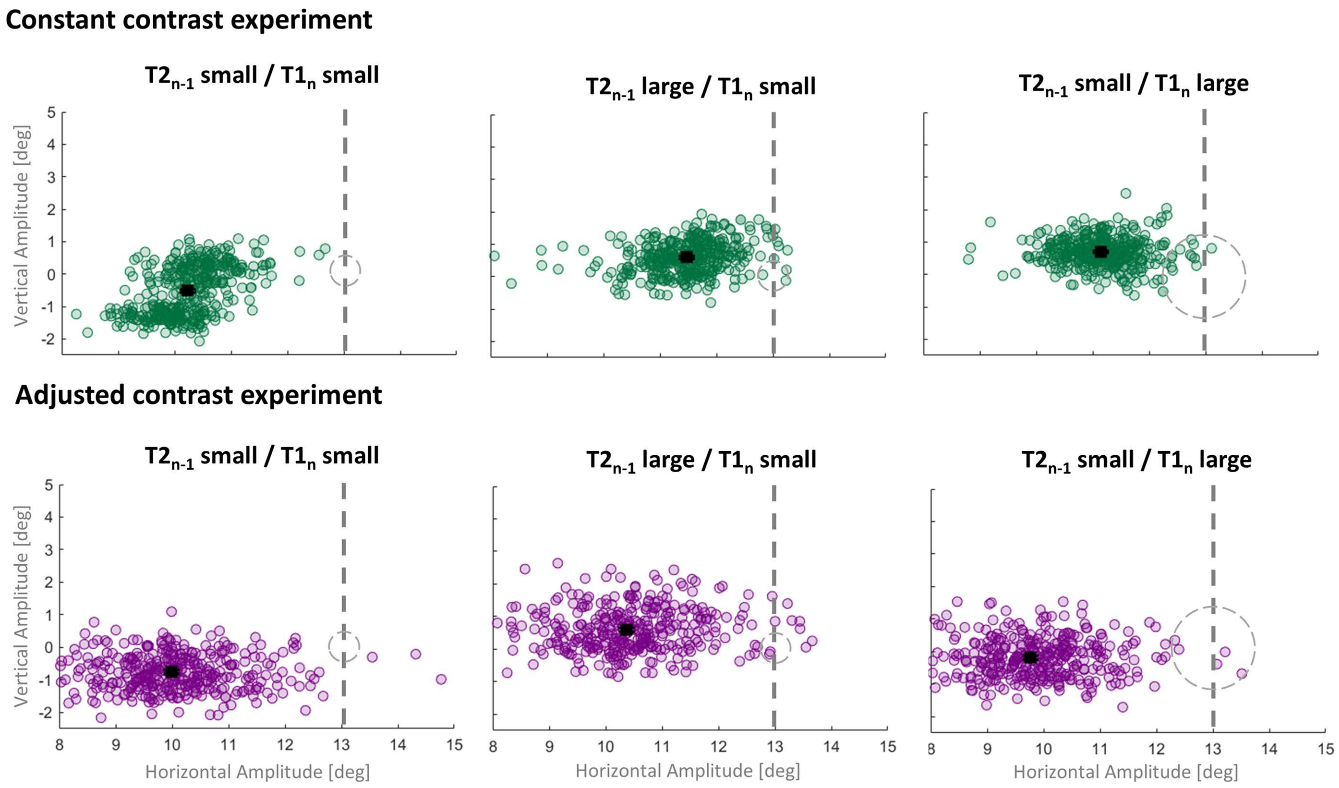

Figure 2. Saccadic amplitudes (degree) of two example subjects for each session and each experiment. The dashed line represents the optimal amplitude to reach the target. The empty dashed circle represent the spatial constant and position of T1. The black square represents the mean saccadic amplitude. Error bars represent standard error of the mean.

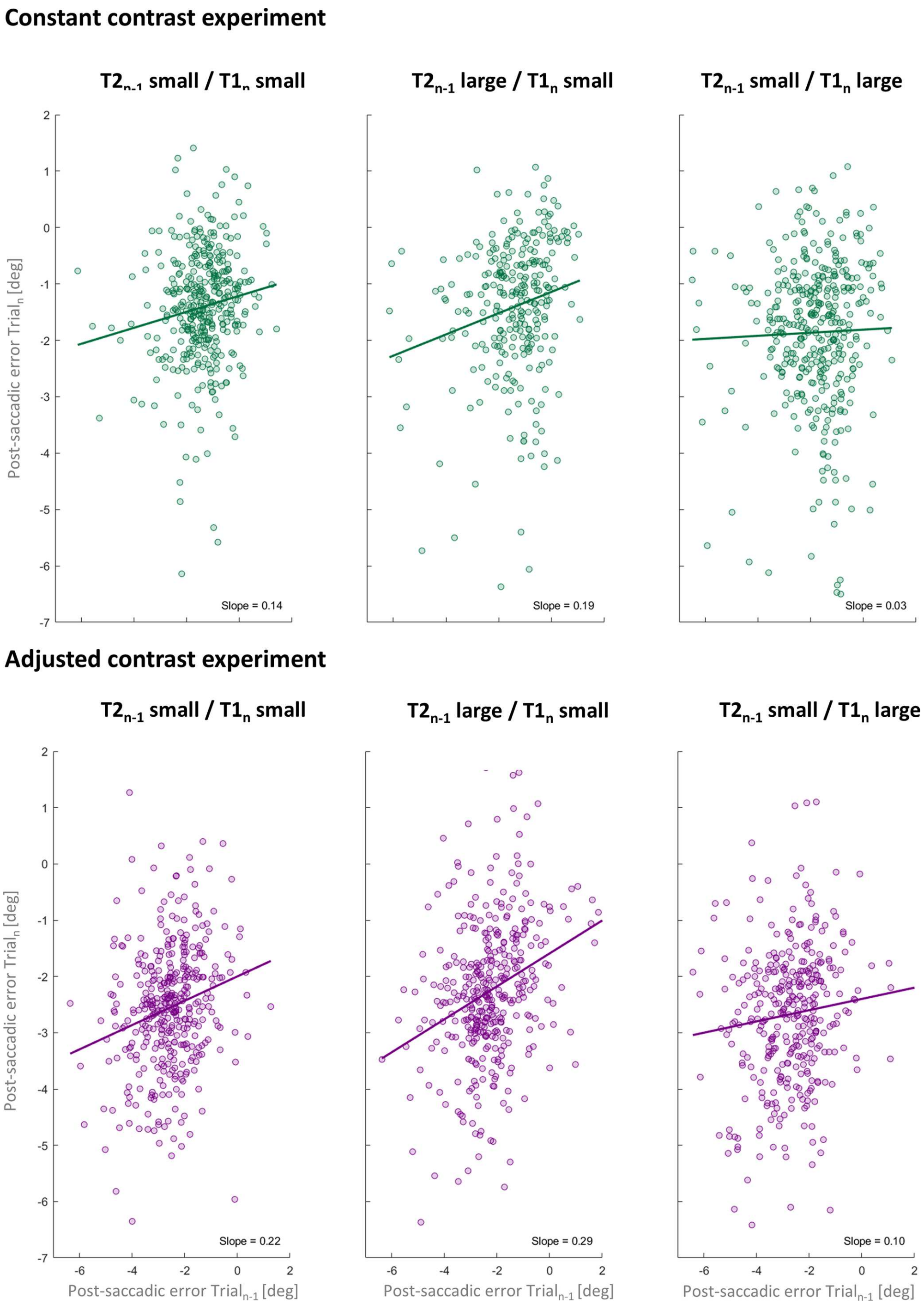

Figure 3. Post-saccadic error in Trial$_n$ (degree) as a function of the post-saccadic error of Trial$_{n-1}$ of two example subjects for each session and each experiment ("constant contrast" experiment, green; "adjusted contrast" experiment, purple). Positive errors are interpreted as saccades overshooting the target, and negative numbers represent a saccadic undershoot. The positive slope (solid line) reveals that larger post-saccadic errors in the previous trial led to larger post-saccadic errors in the current trial.

execution and, because of the ballistic characteristics of saccades, its amplitude cannot be changed mid-flight. We used the slopes to quantify the magnitude of saccade-by-saccade influences. Positive slopes indicate a positive serial dependency between post-saccadic errors, as larger post-saccadic errors in the previous trial led to larger post-saccadic errors in the current trial.

Figure 4 shows the mean slopes for each session and each experiment. Descriptively, we saw a diminished serial dependency magnitude in the "adjusted contrast" experiment when the current target T1 was large

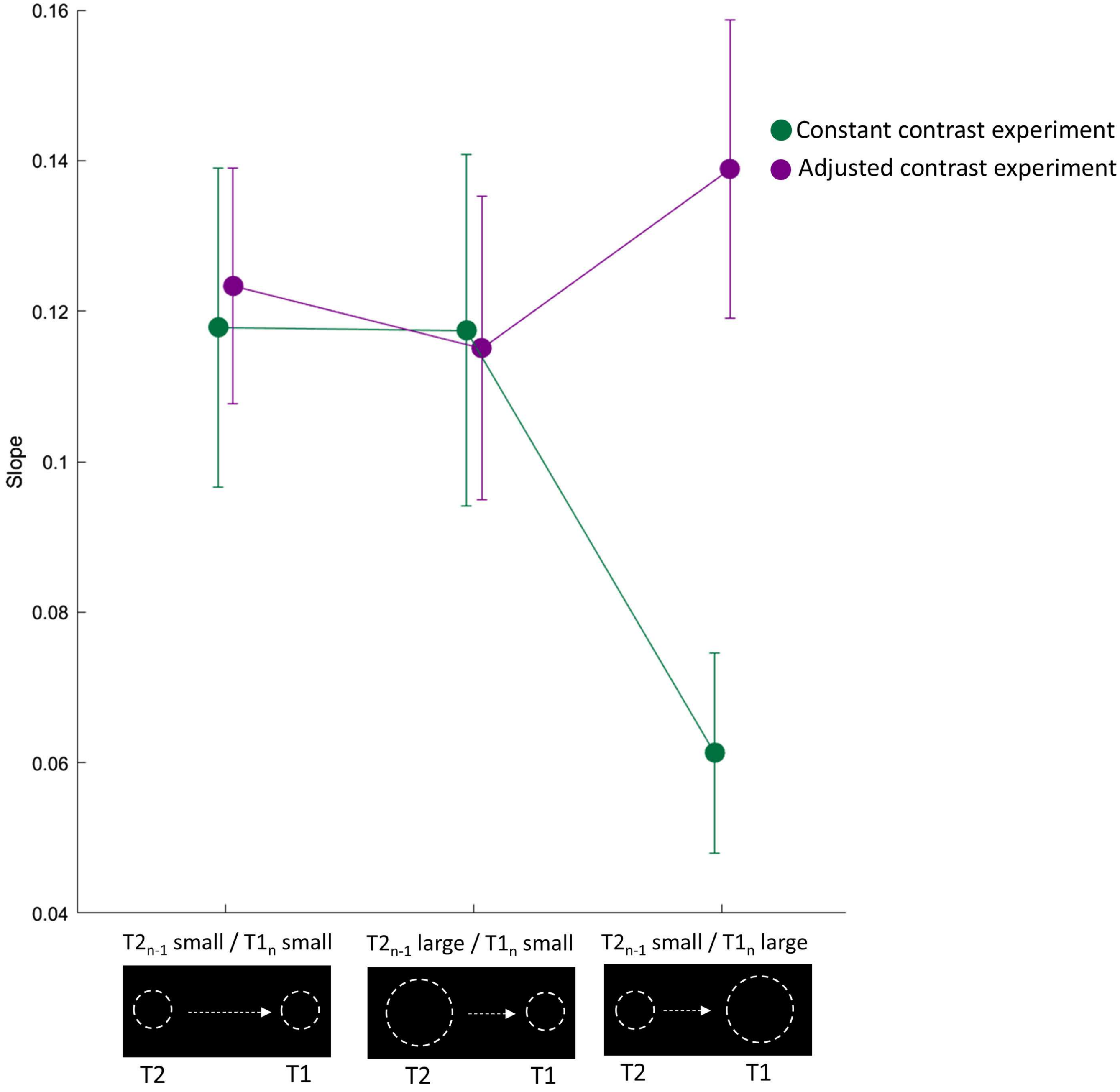

Figure 4. Mean slopes for the linear regression between the predictor Trial$_{n-1}$ (either small or large post-saccadic target) and the criterion Trial$_n$ (small or large pre-saccadic target) for both experiments. Only when pre-saccadic target information was interpreted as too ambiguous did past behavior not influence current behavior, resulting in smaller serial dependency strengths. Error bars represent the standard error of the means.

and the previous post-saccadic target T2 was small. In the "constant contrast" experiment, we first investigated if influences from trial to trial could be observed independently of the session. To test this, *t*-tests against zero for the mean slopes were applied, indicating a significant serial dependency of the previous post-saccadic error on the current one, independently of the visuospatial uncertainty of the pre- or post-saccadic target (Figure 4, green; all $p <$ 0.001, Bonferroni corrected). Additionally, we were interested in whether the serial dependency strength differed dependent on the uncertainty of the target. A one-way ANOVA with the factor session (T2$_{n-1}$ small/T1$_n$ small, T2$_{n-1}$ large/T1$_n$ small, T2$_{n-1}$ small/T1$_n$ large) showed a significant effect, $F(2, 42) = 4.26$, $p = 0.021$. Bonferroni-corrected post hoc *t*-tests indicate that, when perceiving a highly uncertain pre-saccadic target in the current trial, preceded by a highly certain

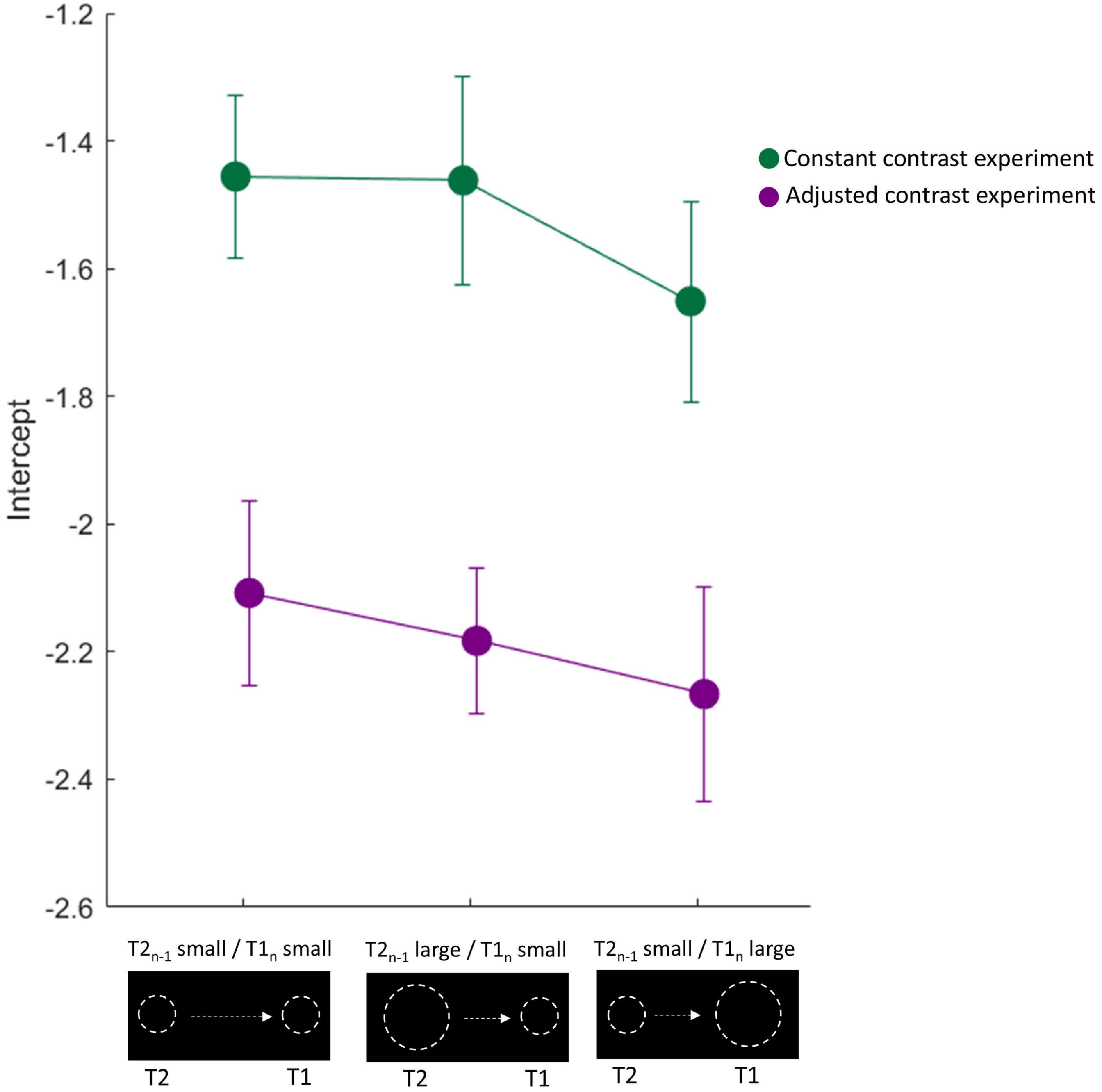

Figure 5. Mean intercepts for the linear regression between the predictor Trial$_{n-1}$ (either small or large post-saccadic target) and the criterion Trial$_n$ (small or large pre-saccadic target) for both experiments. We found stronger saccadic undershoots for the "adjusted contrast experiment," in agreement with the research of Lisi et al. (2019). Error bars represent the standard error of the means.

post-saccadic target, significantly less trial-by-trial influences occurred compared with the other sessions (T2$_{n-1}$ small/T1$_n$ small: $t = 2.54$, $p = 0.045$; T2$_{n-1}$ large/T1$_n$ small: $t = 2.52$, $p = 0.047$). No difference was found between session T2$_{n-1}$ small/T1$_n$ small and session T2$_{n-1}$ large/T1$_n$ small ($t = 0.02$, $p > 0.999$).

In the "adjusted contrast" experiment, we decreased target contrast with increasing spatial constant. Overall, serial dependency influences were found, independently of combination of the initial and the displaced target as t-tests against zero for the mean slopes indicate (Figure 4, purple; all $p < 0.001$, Bonferroni corrected). Additionally, we were interested in whether the serial dependency strength differed dependent of the visuospatial uncertainty of the target. A one-way ANOVA with the factor session (T2$_{n-1}$ small/T1$_n$ small, T2$_{n-1}$ large/T1$_n$ small, T2$_{n-1}$ small/T1$_n$ large) did not show a significant effect, $F(2, 42) = 0.54$, $p = 0.588$). A

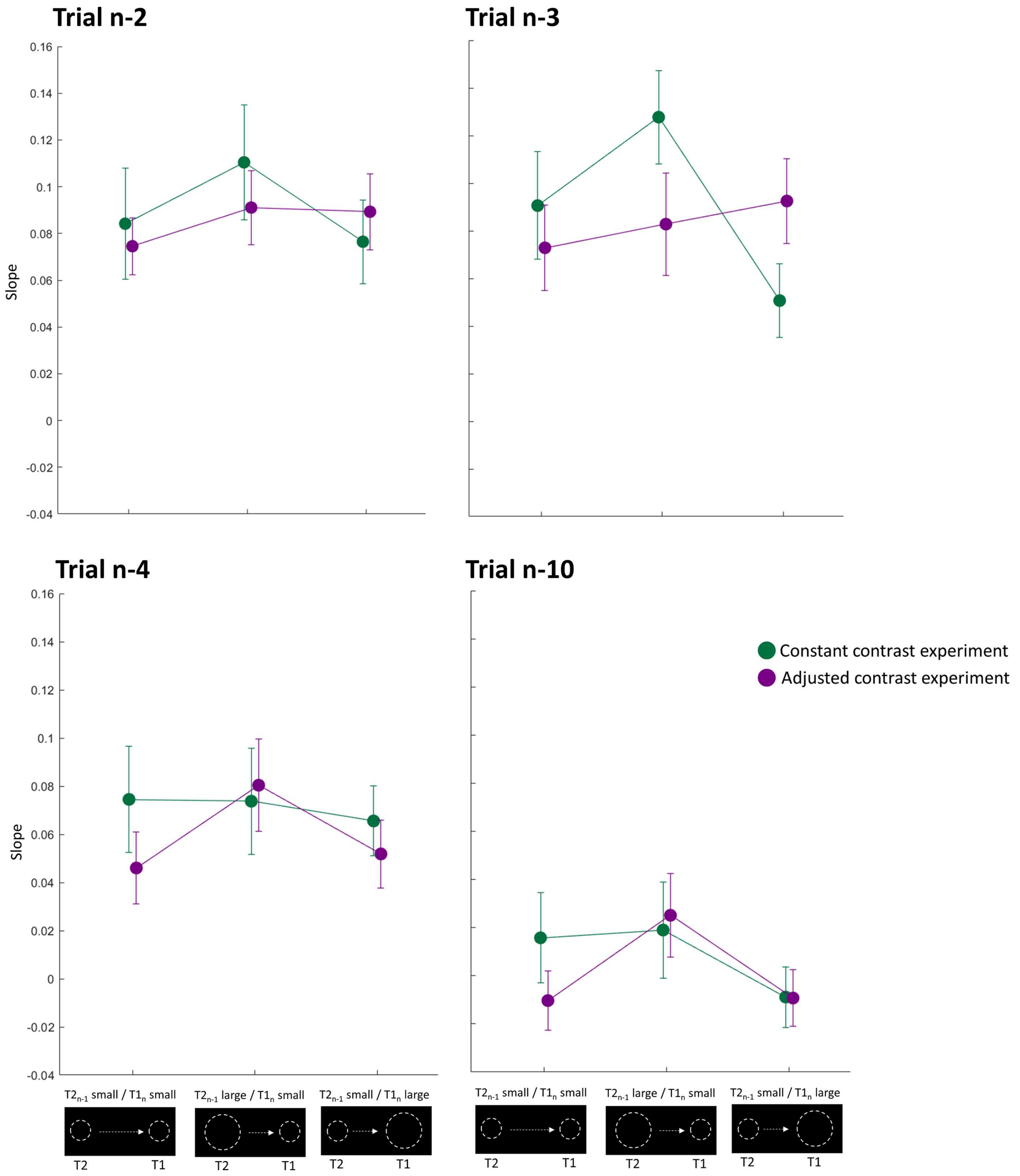

Figure 6. Mean slopes for the linear regression between the predictor Trial$_{n-\text{back}}$ (either small or large post-saccadic target) and the criterion Trial$_{n}$ (small or large pre-saccadic target) for both experiments. We investigated the temporal tuning of the serial dependency by investigating the influence of Trial$_{n-2}$ (upper left), Trial$_{n-3}$ (upper right), Trial$_{n-4}$ (lower left), and Trial$_{n-10}$ (lower right). Error bars represent the standard error of the means.

pre-saccadic target with reduced spatial constant (and therefore implied lower spatial uncertainty) yielded indistinguishable serial dependency strengths compared with the other sessions.

Additionally, we performed a 2 × 3 ANOVA with the factors experiment (constant contrast, adjusted contrast) and session ($T2_{n-1}$ small/$T1_n$ small, $T2_{n-1}$ large/$T1_n$ small, $T2_{n-1}$ small/$T1_n$ large) was calculated to identify differences in the serial dependence strength between the two conditions, revealing a significant interaction effect, $F(2, 42) = 4.55$, $p = 0.016$, but no significant main effect of experiment or session (all $p > 0.265$), Bonferroni-corrected post hoc *t*-tests indicated no difference among groups (all $p > 0.156$).

We performed a 2 × 3 ANOVA with the factors experiment (constant contrast, adjusted contrast) and session ($T2_{n-1}$ small/$T1_n$ small, $T2_{n-1}$ large/$T1_n$ small, $T2_{n-1}$ small/$T1_n$ large) for the intercepts of the fits (see Figure 5). This analysis revealed a significant main effect of experiment, indicating a stronger undershoot for saccadic performance in the "adjusted contrast experiment" assuming no influence from the current post-saccadic error, independently of the performed session, $F(1, 21) = 14.50$, $p = 0.003$. The main effect of session and the interaction term experiment × session did not reach significance ($p = 0.175$ and $p = 0.728$, respectively).

We additionally analyzed the temporal tuning of the serial dependency by calculating the influence of Trial_{n-2}, Trial_{n-3}, Trial_{n-4}, and Trial_{n-10} (Figure 6). We performed the same 2 × 3 ANOVA with the factors experiment (constant contrast, adjusted contrast) and session ($T2_{n-1}$ small/$T1_n$ small, $T2_{n-1}$ large/$T1_n$ small, $T2_{n-1}$ small/T1n large) for each *n*-back structure. Only the interaction term for the influence of Trial_{n-3} was significant, $F(2,42) = 4.03$, $p = 0.025$; all other $p > 0.195$). Bonferroni-corrected post hoc *t*-tests indicated no difference among the groups (all $p > 0.067$).

Last, we determined if differences in the number of corrective saccades between sessions and experiments could be observed. For the "constant contrast" experiment, we identified 34.86% corrective saccades for the $T2_{n-1}$ small/$T1_n$ small session over the whole experiment (mean amplitude, 0.49° ± 0.10°), 35.23% corrective saccades for the $T2_{n-1}$ large/$T1_n$ small session (mean amplitude, 0.46° ± 0.10°), and 32.82% corrective saccades for the $T2_{n-1}$ small/$T1_n$ large session (mean amplitude, 0.49° ± 0.10°). For the "adjusted contrast" experiment, the percentage of identified corrective saccades and their mean amplitudes were descriptively similar ($T2_{n-1}$ small/$T1_n$ small, 36.80% and 0.70° ± 0.08°; $T2_{n-1}$ large/$T1_n$ small, 35.59% and 0.58° ± 0.08°; $T2_{n-1}$ small/$T1_n$ large, 36.85% and 0.63° ± 0.08°, respectively). A 2 × 3 ANOVA with the factors experiment (constant contrast, adjusted contrast) and uncertainty ($T2_{n-1}$ small/$T1_n$ small, $T2_{n-1}$ large/$T1_n$ small, $T2_{n-1}$ small/$T1_n$ large) was calculated, revealing no significant differences among the amplitudes of the corrective saccades (all $p > 0.203$).

Discussion

In this study, we investigated how pre- and post-saccadic saccade target uncertainty influences serial dependencies in saccade amplitudes. If a saccade target is displaced during saccade execution, amplitudes of succeeding saccades will be adaptively modified to minimize the post-saccadic error (Bahcall & Kowler, 2000; Pomè, Tyralla, & Zimmermann, 2023; Tyralla et al., 2023; Zimmermann & Lappe, 2010). We manipulated the saccade target appearance by using Gaussian blobs as targets for which the spatial constants were either small (implying a high spatial certainty) or large (implying a low spatial certainty). When both the pre-saccadic and the displaced, post-saccadic target had a small spatial constant, we observed serial dependencies with strengths comparable to those of a previous report (Cont & Zimmermann, 2021). In our main experiment, either the pre-saccadic or the post-saccadic target had a high spatial constant. We compared two experiments: If the pre-saccadic target had a high spatial constant, the post-saccadic target had a small spatial constant and vice versa. We additionally varied the contrast of the saccade.

We found that if the pre-saccadic target had a small and the post-saccadic target had a high spatial constant, serial dependencies were indistinguishable from the session in which both targets had a small spatial constant. However, if the pre-saccadic target had a high spatial constant and the post-saccadic target had a low spatial constant, the strength of serial dependencies differed drastically between the two contrast experiments. If targets had a constant contrast then serial dependencies were weak, whereas if targets had an adjusted contrast they were much stronger.

Manipulating the spatial constant of the pre-saccadic target can affect saccadic landings in several ways. On the one hand, if the pre-saccadic target has a high spatial constant, saccade landing might become more variable, thus washing out influences of the previous post-saccadic error. On the other hand, saccade landing might rely more on the previous error because the current pre-saccadic target is more difficult to localize. In that case, serial dependencies might either become relevant in the perceptual target localization or remain in the sensorimotor domain and the strength of their influence depends on the visuospatial uncertainty of the target. In both of these cases, serial dependencies should become stronger for targets with a higher spatial constant. Both of these explanations are incompatible with the observed data, as we did not find that landing was more variable for targets with a high spatial constant, nor did saccade landings take the error more into account than for a target with a small spatial constant. It is therefore unlikely that the uncertainty of the pre-saccadic target explains our data. The absence of saccade target uncertainty effects on saccade adaptation are consistent with a previous report. Souto et al. (2016) found little correlation between target uncertainty and saccade adaptation rates.

The findings lead us to conclude that it is rather the stimulus visibility and the spatial extent of pre-saccadic target T1 that determines how much the post-saccadic error (i.e., target T2) is taken into consideration. Put simply, a large saccade target allows many correct landing positions. The displaced target T2 will thus not induce adaptive changes as strong as it would have for a spatially focused saccade target T1. The post-saccadic evaluation of the landing error will be more tolerant for high contrast and large saccade targets. The tolerance built up only when pre-saccadic target T1 was large. When it was small and post-saccadic target T2 became large, no change in serial dependency strength was observed. This asymmetry demonstrates that the trans-saccadic change in the target size cannot be responsible per se for the weaker serial dependencies. One could argue that congruency between the pre- and post-saccadic target is a requirement for adaptive amplitude changes; however, we argue that a minimum target contrast is required to compare the spatial extent of pre-saccadic target T1 to the location of post-saccadic target T2. Our data revealed that the spatial extent of pre-saccadic target T1 served as an anchor in the evaluation of the post-saccadic error. Previous research established that the driving signal of saccade adaptation is the prediction error, consisting of the difference between the observed retinal and the predicted post-saccadic error (Bahcall & Kowler, 1999; Bahcall & Kowler, 2000; Collins & Wallman, 2012; Wong & Shelhamer, 2011). Heins et al. (2023) found that presenting a pre-saccadic target is not necessary to induce saccadic adaptation. Saccade amplitudes changed adaptively through the mere presence of a post-saccadic error. However, subjects could predict where the target would appear and thus also could predict the post-saccadic error. Our data show that the prediction of post-saccadic error takes into account the features of the pre-saccadic target. A large and salient target induces a spatially more distributed prediction of the saccade target than a focused target.

In conclusion, our study shows that features of a pre-saccadic target determine how strong post-saccadic errors induce adaptive amplitude changes.

Keywords: saccadic adaptation, serial dependency, uncertainty, gaussian blobs

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Serial dependencies and overt attention shifts

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Abstract

Uncertain visual input is serially dependent on stimulation from the recent past. We can attend to stimuli either endogenously based on an internal decision or exogenously, triggered by an external event. Here, we wondered whether serial dependencies are selective for the attentional mode which we draw to stimuli. We studied overt attention shifts, i.e. saccades and recorded either motor error correction or visual orientation judgements. In Experiment 1, we assessed sensorimotor serial dependencies, focusing on how the post-saccadic error influences subsequent saccade amplitudes. In Experiment 2, we evaluated visual serial dependencies by measuring orientation judgments, contingent on the type of saccade performed. In separate sessions, participants performed either only voluntary saccades, only delayed saccades, or both saccade types alternated within a session. Our results revealed that sensorimotor serial dependencies were selective for the saccade type performed. When in the preceding trial voluntary saccades have been performed, serial dependencies were much stronger if in the current trial voluntary instead of delayed saccades were executed. In contrast, visual serial dependencies were not influenced by the type of saccade performed. Our findings reveal that shifts in exogenous and endogenous attention differentially impact sensorimotor serial dependencies, while visual serial dependencies remain unaffected.

Keywords: saccadic adaptation, serial dependency, orientation judgement, overt attention

Introduction

Perceptual processing must constantly compensate for noisy sensory input. One method to do so has been discovered in the last decade and consists in a reliance of current stimulus interpretation on the recent past. Fischer & Whitney (2014) described that when observers had to report the orientation of stimuli presented in the visual periphery, their estimate was biased by the orientation of the stimulus presented in the previous trial. In other words, the interpretation of noisy sensory input is serially dependent on similar stimulation from the recent past. Most serial dependencies that have been reported are positive, such that the current stimulus appears a bit more similar to the previous one (Liberman et al., 2016; Taubert et al., 2016; Tyralla & Zimmermann, 2024). Since then, serial dependencies have been found in various domains such as visual orientation (Fischer & Whitney, 2014; Fritsche & de Lange, 2019; Rafiei et al., 2021), shape (Manassi et al., 2019, Manassi et al., 2021; Collins, 2022), color (Bays et al., 2009; Barbosa & Compte, 2020), numerosity (Cicchini et al., 2014; Fornaciai & Park, 2018), visual stability (Manassi & Whitney, 2022) or saccadic eye movements (Cont & Zimmermann, 2021). Serial dependencies are believed to be the signature of a mechanism that stabilizes perception (Manassi & Whitney, 2024; Cicchini et al., 2024). If our environment is successfully integrated into a stable perception, these authors argue, object features will result in smooth and continuous perception. We have recently reported sensorimotor serial dependencies that exist between motor errors and perceptual estimates (Cont & Zimmermann, 2021). When subjects were required to perform a saccade, an artificial error was created by displacing the saccade target during movement execution. The amplitude of the immediately following saccade was shaped by this post-saccadic error. If subjects were asked after having experienced a post-saccadic error in the previous trial to visually localize a target in space, their estimate in the current trial was likewise biased by the preceding one.

However, serial dependencies operate within certain limits. Perception would be tremendously impaired if every object biases every other. Serial dependencies are temporally and spatially tuned such that only objects close in space and time affect each other (Fischer & Whitney, 2014; Manassi et al., 2018). Several studies suggested that visual serial dependencies affect the current stimulus only if the previously encountered stimulus had been attended (Fischer and Whitney, 2014, Rafiei et al., 2021; Fornaciai and Park, 2018; Liberman et al., 2016, Bae and Luck, 2020). Fischer and Whitney (2014) presented eight oriented targets and a cue indicating which of the targets has to be judged. They find significant serial dependencies only if the cue validity was 100%. In another approach, Bae and Luck (2020) could demonstrate that not only the target but also its specific feature had to be attended to produce serial dependencies. They found serial dependencies for motion only if subjects had to report the direction of motion but not if they reported the color of the same stimulus in the previous trial. However, other studies did not find any effects of attention on serial dependencies (Fornaciai & Park, 2018; Goettker & Stewart, 2022). In a meta-analysis, Manassi et al. (2023) found that devoting fewer attentional resources to the previous stimulus will result in reduced serial dependencies.

Allocation of attention can be divided into two modes. Endogenous attention is drawn deliberately to objects of interest. Exogenous attention is triggered by a sudden event in the external world which leads to an automatic attention shift to its location (Carrasco and Barbot, 2015). Deployment of exogenous attention is transient, builds up for ~100-120 ms and decays fast. Endogenous attention by contrast takes longer to build up (~300 ms) and can be uphold as demanded. Attention shifts improve the processing of visual contrast and spatial resolution. The effects of exogeneous and endogenous attention shifts differ. For spatial resolution, exogenous attention improves spatial resolution in the visual periphery at the cost of central information. Endogenous attention can improve perception simultaneously, at peripheral and central locations. A recent study investigated the impact of exogenous and endogenous covert attention shifts on the sensory tuning of orientation. Both modulate sensory tuning by changing its gain with exogenous attention having stronger orientation gain enhancement (Fernández, Okun & Carrasco, 2022).

Attention can also be divided to the way it is drawn to objects of interest. We can attend either covertly to objects or events while keeping our eyes still or overtly by performing an eye movement. While the differences between exogenous and endogenous attention have mostly been studied in covert attention shifts, they can also be observed in overt attention shifts. When a sudden visual event appears, we perform a delayed saccade to it automatically. However, we can also execute voluntary saccades moving the eye to a target selected by an internal decision. Shortly before a saccade is executed attention shifts mandatorily to the saccade target location (Deubel & Schneider, 1996; Hoffman & Subramaniam, 1995; Kowler et al., 1995; Shepherd et al., 1986; Van der Stigchel & Theeuwes, 2007). Overt and covert attention shifts are assumed to be coupled (Awh, Armstrong, & Moore, 2006; Corbetta, 1998; Smith & Schenk, 2012).

In the current study we asked whether exogenous and endogenous overt attention shifts would differentially affect serial dependencies. If the separate effects of exogenous and endogenous covert attention shift on neural orientation tuning take place likewise in overt attention shifts, the magnitude of serial dependencies might vary. For sensorimotor serial dependencies, results from saccade adaptation suggest differences for overt exogenous and endogenous attention shifts. In saccade adaptation, the amplitude adjustment in response to the previous post-saccadic error increases across trials until it reaches a steady-state level (Pélisson 2010, McLaughlin 1967, Hopp & Fuchs 2004). Different saccade types have been tested in saccade adaptation experiments. The adaptation transfer between delayed and voluntary saccade types has been tested (Erkelens & Hulleman, 1993; Deubel, 1995; Collins & Dore-Mazars, 2006; Alahyane et al., 2007). These studies revealed that the adaptation of voluntary saccade substantially transfers to delayed saccades whereas delayed saccade adaptation does not transfer to the same extent to voluntary saccades. It is yet unclear why the transfer is different. On the one hand, amplitude adjustments might occur at programming stages which only partly overlap for both saccade types. On the other hand, the presentation duration of the saccade targets which differ for delayed and voluntary saccades might produce the asymmetric adaptation transfer. Consistent with this view, studies have shown that adaptation of voluntary saccades affects the localization of stationary and flashed visual targets whereas the adaptation of delayed saccades affects the localization of flashed targets only (Zimmermann & Lappe, 2009; Schnier, Zimmermann & Lappe, 2010).

In the current study we sought to compare how overt exogenous and endogenous attention shifts affect sensorimotor and visual serial dependencies.

Methods

Participants

23 subjects (mean age 21.13 years, *SD* = 2.49 years; 19 women) participated in Experiment 1. 23 subjects (mean age 22.71 years, *SD* = 5.22 years; 18 women) participated in Experiment 2. Participants were German native speaker, reported normal vision or wore lenses during the experiment, and indicated no psychiatric or neurological diseases. Participants were recruited at the Heinrich-Heine University Düsseldorf. They either received course credits or 10 euros per hour for participation.

Setup

Stimuli were presented on a CRT screen (Diamond Pro 2070, 12.9 inches, resolution: 800x600 pixel, refresh rate: 120 Hz). In both experiments, subjects were placed 57cm away from the screen in a dark room. We used a homogeneously grey background (0.09 cd m^2). Participants placed their head in a chin rest to prevent head movements. Eye movements were recorded by a desktop-mounted eye-tracker (EyeLink 1000 Plus, 1000 Hz sampling rate). Participants performed the task binocularly but only the left eye was recorded. A standard 9 points calibration routine was conducted. For recording participants' responses, a standard keyboard and mouse were used.

Experimental procedure

Two experiments were conducted. In Experiment 1, saccade sensorimotor serial dependencies and in Experiment 2, visual serial dependencies were investigated. Each experiment contained three sessions. In the first session of each experiment, voluntary saccades were performed, in the second delayed saccades and in the third, voluntary and delayed saccades alternated (e.g. trial_{n-2} voluntary, trial_{n-1} delayed, trial_{n} voluntary, etc.) across trials.

The sequence of sessions within each experiment was fixed and all subjects performed the three sessions in the described order. Since we aimed to match saccade latencies in the voluntary and delayed saccade sessions, we measured first voluntary saccades in order to use the average latencies from these sessions for the timing of the go-signal in the delayed saccade sessions. Delayed and voluntary saccade sessions had 400 trials each (duration of 20 min each). The third session, in which voluntary and delayed saccades alternated, had 800 trials (duration of 40 min; starting with a voluntary saccade trial).

Figure 1

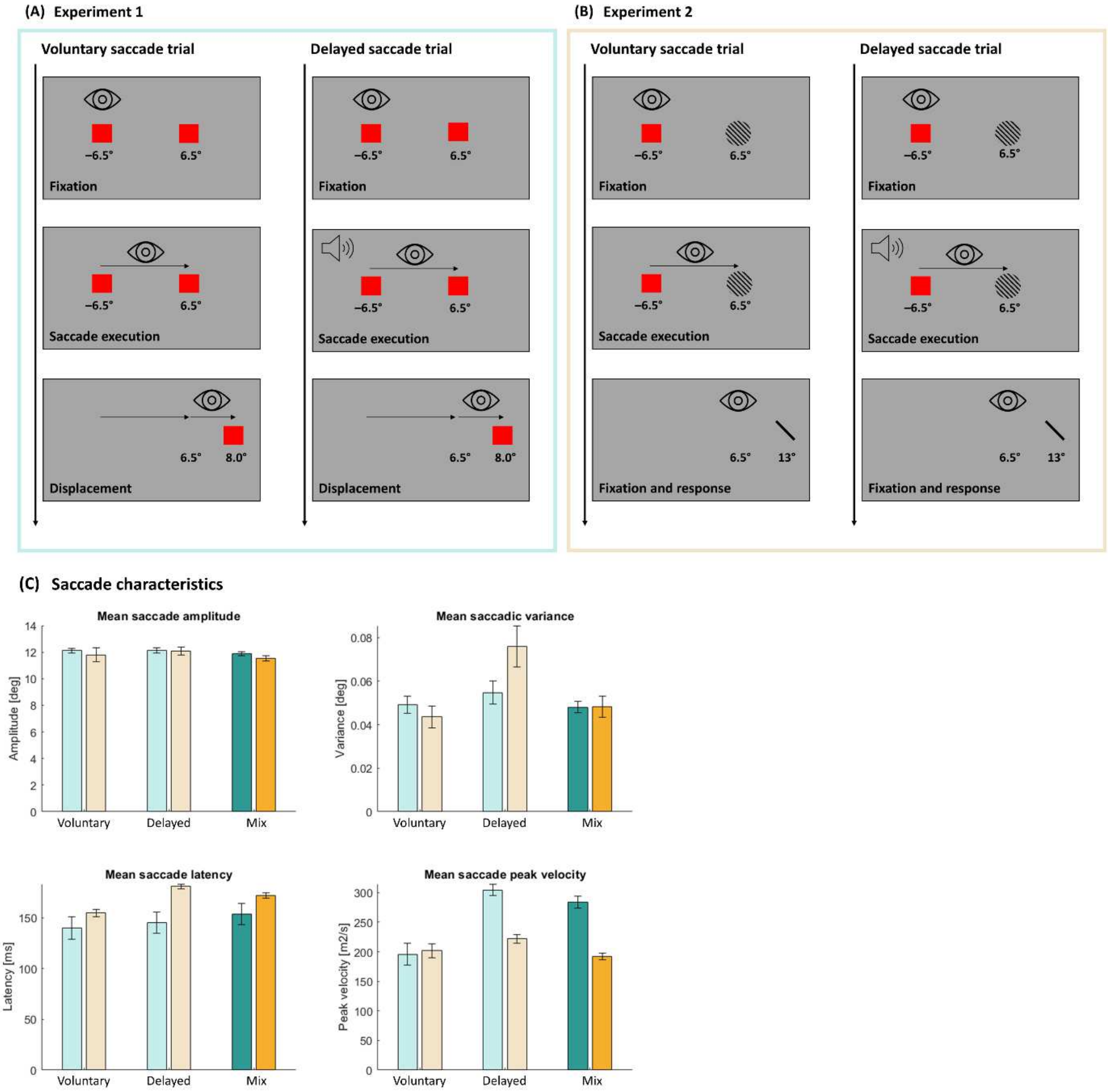


Figure 1. (A) Schematic description of the procedure for voluntary and delayed saccade trials (Experiment 1). Subjects performed a saccade towards the target, either voluntarily (left) or delayed (right). During saccade execution, the target was displaced to one out of six possible positions. In delayed saccades trials the saccade execution was indicated by an acoustical cue.
(B) Schematic description of the procedure for the orientation judgement task (Experiment 2). The structure was similar to (A). Instead, subjects should perform a saccade toward a Gabor patch. After saccade execution (either voluntarily or delayed) subjects should keep fixation and reproduce the perceived orientation by rotating a response bar.
(C) Average saccade characteristics for saccadic amplitude, variance, latency and peak velocity were specified for Experiment 1 (blue), and Experiment 2 (yellow).

Lighter colors indicate the condition with the same saccade type, darker colors the mixed condition. Error bars represent the standard error of the mean.

Experiment 1

Voluntary saccade session

Figure 1A schematically shows the trial structure. A trial began with the presentation of a fixation square (red, 0.55° x 0.55°) that was shown 6.5° to the left side of the screen and the saccade target T1 (red, 0.55° x 0.55°) that was shown 6.5° to the right of the screen center. Subjects were instructed to perform a saccade towards saccade target T1 at their own pace. Eye movements were recorded online and as soon as the stimulus program detected an eye velocity higher than 30° s^{-1} in five consecutive eye-tracking samples, the target was displaced. One target displacement size was randomly selected out of six possible (-2.5°, -1.5°, -0.5°, 0.5°, 1.5°, 2.5°). The second target disappeared and a new trial started 1200 after saccade completion.

Delayed saccade session

A trial began with the presentation of a fixation square (red, 0.55° x 0.55°) that was shown 6.5° to the left side of screen center and a saccade target T1 (red, 0.55° x 0.55°) that was shown 6.5° to the right side of screen center. Subjects were instructed to perform a saccade as soon as they heard a sinus sound cue. We measured the mean saccade latency for each subject individually in their voluntary saccade session to match the average saccade latency (± *SD*) for the delayed saccade trials. The rest of the trial was identical as in voluntary saccade sessions.

Experiment 2

Voluntary saccade session

A trial began with the presentation of a fixation square (red, 0.55° x 0.55°, 6.5° to the left of the screen center) and a Gabor patch (T1) that was shown 6.5° to the right of the screen center (Fig. 1B). The Gabor patch had a spatial frequency of 0.3 cycles per degree and a Gaussian contrast envelope of 1.5° standard deviation. The orientation of the Gabor patch randomly varied between five possible orientations (25°, 35°, 45°, 55°, 65°). Subjects were instructed to perform a voluntary saccade towards T1 and to keep fixation after saccade landing. As soon as saccade landing was detected (eye velocity smaller than 30° s^{-1} in five consecutive samples) the Gabor patch disappeared and a response bar (width of 0.80°) occurred simultaneously in their periphery. The spatial distance between the saccade landing position and the response bar location was adjusted to correspond with the size of the saccade amplitude. This adjustment ensured that T1 (while fixating on the left) and the response bar (while fixating on T1 location) maintained the same retinal position. The orientation of the response bar was randomly determined for each trial. Participants were instructed to align the orientation of the response bar with their perceived orientation of the Gabor patch. Using a standard computer mouse, participants could rotate the response bar either clockwise or counterclockwise. They confirmed their response by pressing the space bar.

Delayed saccade session

Delayed saccades trials were designed as in the delayed saccades session of Experiment 1. Instead of red target squares we used Gabor patches as targets with the same characteristics and task described for voluntary saccade trials for Experiment 2.

Data analyses

All saccades with amplitudes larger than half the required distance were included in the analysis. In delayed saccade trials, we excluded trials in which subjects performed anticipatory saccades, which started before the sinus sound cue was played. For Experiment 2 we additionally excluded trials in which participants did not fixate the Gabor patch location after saccade execution, i.e. in which gaze positions exceeded a radius of 2.5° around the Gabor patch. On average, ~95% of trials went into analysis.

For Experiment 1, we computed the post-saccadic error for each trial as the difference between the actual target position of T1 (6.5°) and the saccade landing position. For Experiment 2, we computed the error between the Gabor patch orientation and the reproduced orientation.

In order to analyze serial dependencies in Experiment 1 and in Experiment 2, we calculated linear regressions between errors in the previous and the current trial for each subject in each session. Bonferroni-corrected students t-tests against zero on the slopes were conducted to investigate serial dependence effects. We conducted a 2x2 ANOVA with the within-subject factor ‘previous trial’ (voluntary, delayed) and the within-subject factor ‘current trial’ (voluntary, delayed) to investigate differences in the strength of trial-by-trial influences.

Results

Experiment 1

Figure 2A shows saccadic amplitudes for a representative subject in Experiment 1. Subjects were instructed to perform a horizontal saccade of 13° (indicated by the dashed line in all panels). In all sessions, the subject undershot the target systematically. In the left panel, saccadic amplitudes for only voluntary saccades are presented, resulting in a mean saccadic amplitude of 11.34° (*SD* = 0.03°). In the middle panel, only delayed saccades were performed (mean amplitude: 11.06°, *SD* = 0.03°). In the right panel, the subject performed voluntary and delayed saccades alternatingly, starting with voluntary saccades (mean amplitude: 10.70°, *SD* = 0.03°).

Figure 2

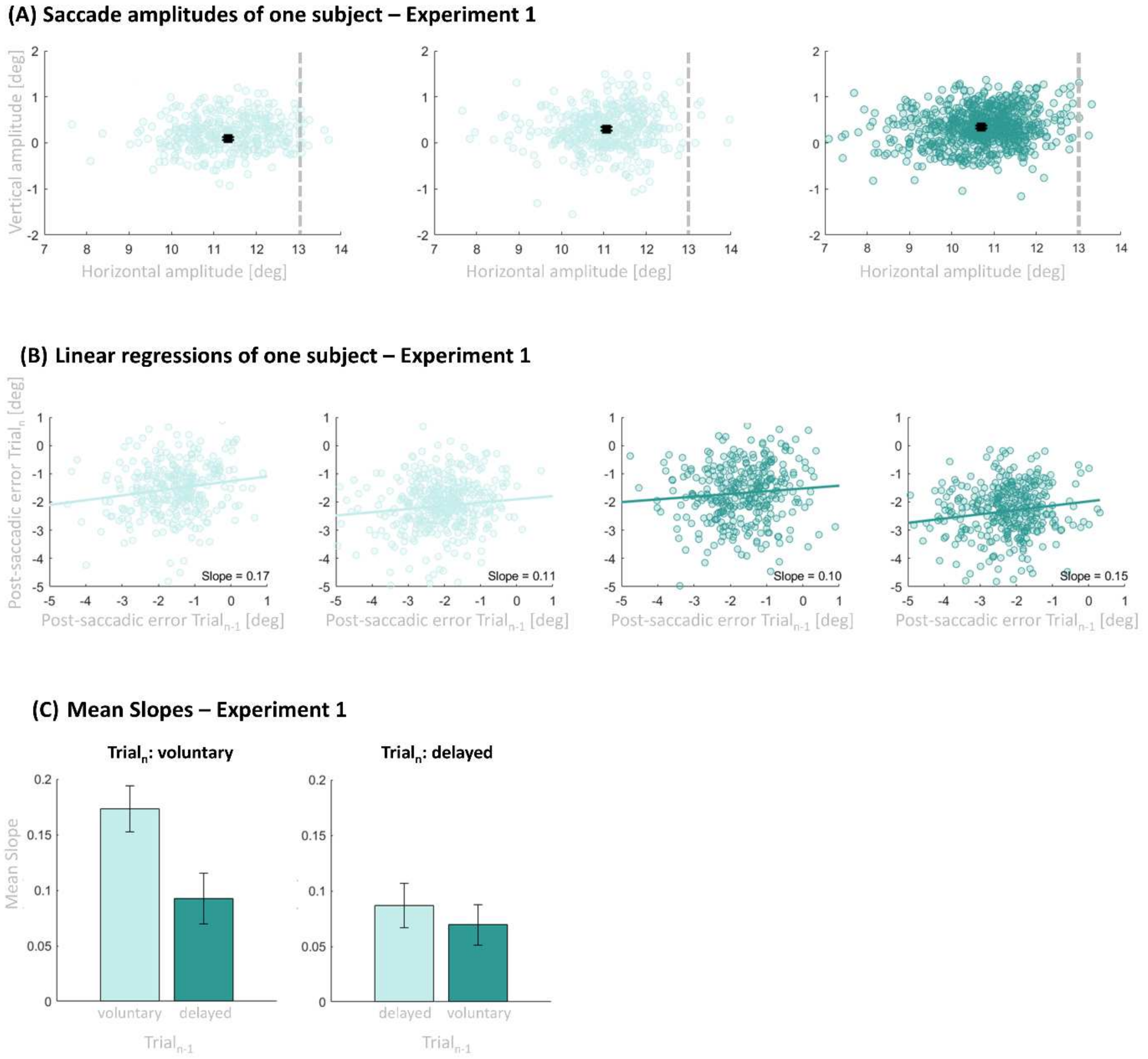


Figure 2. (A) Saccadic amplitudes of one example subject for each session in Experiment 1. Dashed line represents the optimal amplitude to reach the target. The black square represents the mean saccadic amplitude. Error bars represent standard error of the mean.

(B) Presentation of the post-saccadic error in trial$_n$ as a function of the post-saccadic error of trial$_{n-1}$ of one example subject for each session. Positive errors are interpreted as saccades overshooting the target while negative numbers represent a saccadic undershoot. The positive slope (solid line) reveals that larger post-saccadic errors in the previous trial led to larger post-saccadic errors in the current trial.

(C) Mean slopes for the linear regression between the predictor ‘previous trial’ (voluntary or delayed saccades) and the criterium ‘current trial’ (voluntary or delayed saccades). Only when currently a voluntary saccade is performed, the last behavior is taken into consideration if it was a delayed saccade. Error bars represent the standard error of the means.

Serial dependencies and overt attention shifts

We calculated the post-saccadic error between the actual target position and the saccadic amplitude. In Figure 2B, one example subject for each session is presented to visualize the magnitude of saccade-by-saccade influences. Negative numbers indicate an undershooting of saccadic amplitude, while positive numbers indicate an overshooting of the target. To investigate serial dependencies of the post-saccadic error from the previous trial (trial_{n-1}) to the current trial (trial_{n}), we fitted a linear regression model for each subject in every session separately. In this experiment, horizontal saccades were performed, either only voluntary (first panel), only delayed (second panel), voluntary and delayed (third panel) or delayed and voluntary (fourth panel) horizontal saccades are performed. We used the slopes of the linear fits to quantify the magnitude of sensorimotor serial dependencies. Positive slopes indicate a positive serial dependency between post-saccadic errors: larger post-saccadic errors in the trial_{n} led to larger post-saccadic errors in trial_{n-1}.

Overall, we found serial dependencies for horizontal saccades, independently of the performed saccade type combination, as Bonferroni corrected t-tests against zero for the mean slopes indicate (Fig. 2C; trial_{n-1}: voluntary, trial_{n}: voluntary: $t(22) = 8.63$, $p < .001$; trial_{n-1}: delayed, trial_{n}: delayed: $t(22) = 4.41$; $p < .001$; trial_{n-1}: voluntary, trial_{n}: delayed: $t(22) = 3.86$; $p < .001$; trial_{n-1}: delayed, trial_{n}: voluntary: $t(22) = 4.10$; $p < .001$).

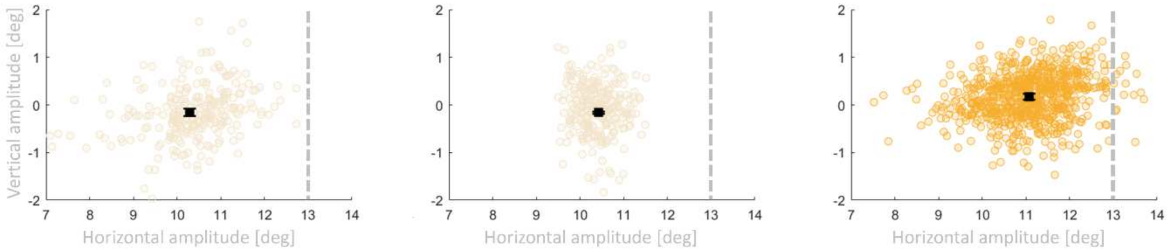
Additionally, we were interested if the serial dependence strength differs if we currently perceive a voluntary or delayed saccade trial, dependent of the previously performed saccade trial (voluntary, delayed). A 2x2 ANOVA with the factor ‘previous trial’ (voluntary, delayed) and the within-subject factor ‘current trial’ (voluntary, delayed) for horizontal saccades indicate a significant main effect of ‘previous trial’ ($F(1,22) = 4.75$, $p = .040$) and ‘current trial’ ($F(1,22) = 9.46$, $p = .006$) in the serial dependence strength, as well a significant interaction effect, $F(1,22) = 4.42$, $p = .047$. Bonferroni corrected post-hoc tests indicate stronger serial dependencies for trials in which only voluntary saccades were performed, compared to only delayed saccades ($t = 3.76$, $p = .003$), voluntary followed by delayed saccades ($t = 3.57$, $p = .006$) and delayed followed by voluntary saccades ($t = 2.95$, $p = .033$). On the contrary, when subjects perform a delayed saccade, the previously performed post-saccadic error does not lead to significantly different serial dependence strengths compared to all other sessions.

Experiment 2

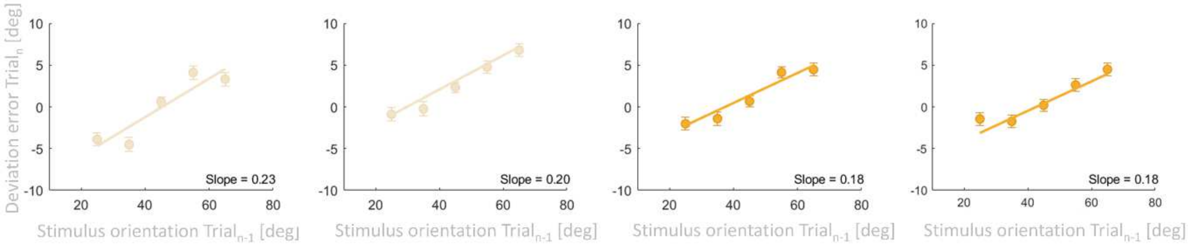
Figure 3A shows saccadic amplitudes for a representative subject in Experiment 2. Subjects were instructed to perform a horizontal saccade of 13° (indicated by the dashed line in all panels) before judging the orientation of the target stimulus. In the left panel, saccadic amplitudes for only voluntary saccades are presented, resulting in a mean saccadic amplitude of 10.29° (*SD* = 0.08°). In the middle panel, only delayed saccades were performed (mean amplitude: 10.42°, *SD* = 0.02°). In the right panel, the subject performed voluntary and delayed saccades alternatingly, starting with voluntary saccades (mean amplitude: 11.07°, *SD*: 0.06°).

Figure 3

(A) Saccade amplitudes of one subject – Experiment 2



(B) Linear regressions of one subject – Experiment 2



(C) Mean Slopes – Experiment 2

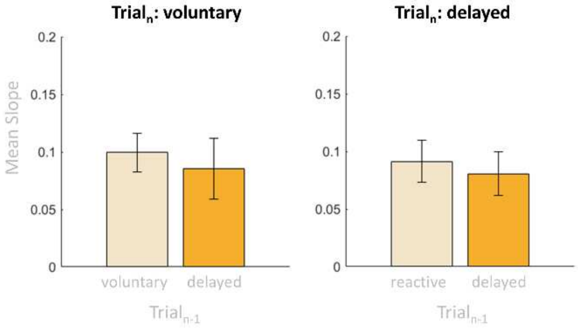


Figure 3. (A) Saccadic amplitudes of one example subject for each session. The dashed line represents the center of the saccade target, i.e. the Gabor patch. The black square represents the mean saccadic amplitude. Error bars represent standard error of the mean.
(B) Presentation of the deviation error in trial$_n$ as a function of target orientation in trial$_{n-1}$ of one example subject for each session. Positive errors are interpreted as judgements overestimating while negative numbers represent an underestimation. The positive slope (solid line) reveals that larger orientations in the previous trial led to larger deviation errors in the current trial.
(C) Mean slopes for the linear regression between the predictor 'previous trial' (voluntary or delayed saccades) and the criterium 'current trial' (voluntary or delayed saccades) for both experiments. No differences in the serial dependency magnitude were found. Error bars represent the standard error of the means.

We calculated the deviation error between the perceived and reproduced orientation.

In Figure 3B, one example subject for each session is presented to visualize the magnitude of saccade-by-saccade influences for Experiment 2. To investigate serial dependencies of the presented orientation of the previous trial (trial_{n-1}) to the deviation error of the current trial (trial_{n}), we fitted a linear regression model for each subject in every session separately. Orientation judgments were required after either only voluntary saccades (first panel), only delayed saccades (second panel), voluntary and delayed saccades (third panel) or delayed and voluntary saccades (fourth panel).

We found serial dependence influences for all four sessions (Fig. 3C), independently of the performed saccade type combination, as t-tests against zero for the mean slopes indicate (Fig. 4C; trial_{n-1}: voluntary, trial_{n}: voluntary: $t(22) = 6.03$, $p < .001$; trial_{n-1}: delayed, trial_{n}: delayed: $t(22) = 5.13$; $p < .001$; trial_{n-1}: voluntary, trial_{n}: delayed: $t(22) = 4.35$; $p < .001$; trial_{n-1}: delayed, trial_{n}: voluntary: $t(22) = 3.28$, $p = .003$).

A 2x2 ANOVA with the factor ‘previous trial’ (voluntary, delayed) and the within-subject factor ‘current trial’ (voluntary, delayed) indicated no differences in serial dependency strengths (main effect ‘previous trial’: $F(1,22) = 0.11$, $p = .917$); main effect ‘current trial’: $F(1,22) = 0.17$, $p = .608$; interaction effect ‘previous trial’ * ‘current trial’, $F(1,22) = 0.47$, $p = .498$). Independently of the performed saccade type in the current and previous trial, orientation judgment errors influence equally strongly from trial to trial.

To further explore the absence of results in Experiment 2, we conducted a median split. The literature indicates that attention shifts differ between undershooting and overshooting saccades. Specifically, with undershooting saccades, the spatial attention focus overlaps less with the visual stimulus, potentially leading to more uncertain stimulus processing. Higher uncertainty might increase serial dependency magnitude, as subject might rely on past sensory processing in compensation. We split the data into post-saccadic errors in each trial according to the saccadic amplitude size in the current trial ($mdn_{\text{voluntary}} = 12.02°$, $mdn_{\text{delayed}} = 10.65°$, $mdn_{\text{voluntary-delayed}} = 11.21°$, $mdn_{\text{delayed-voluntary}} = 11.21°$). Independent t-tests between the two groups for each saccade type (voluntary, delayed, voluntary-delayed or delayed-voluntary) indicate only a significant difference in slopes of voluntary saccades, more clearly higher trial-by-trial influences for undershooting saccades compared to overshooting saccades, $t(22) = 2.06$, $p = .050$ (trial_{n-1}: delayed, trial_{n}:

delayed: *t*(22) = 1.02; *p* = .318; trial_{n-1}: voluntary, trial_{n}: delayed: *t*(22) = 0.48; *p* = .635; trial_{n-1}: delayed, trial_{n}: voluntary: *t*(22) = 0.37, *p* = .717; see also Figure 4).

Figure 4

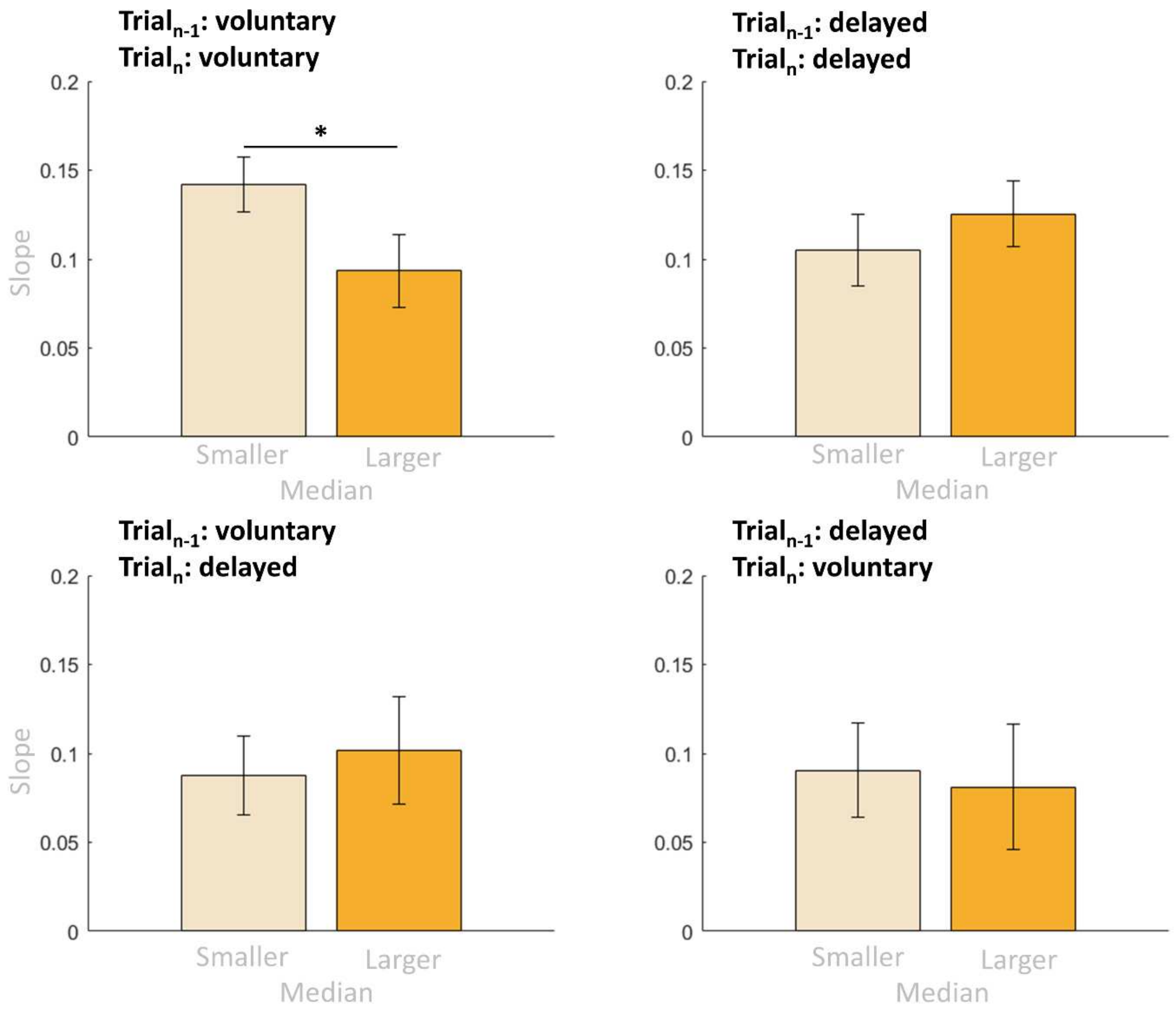


Figure 4. Mean slopes for the linear regression between the predictor ‘previous trial’ (voluntary or delayed) and the criterium ‘current trial’ (voluntary or delayed), separated for post-saccadic error trials with a saccadic amplitude smaller (light yellow) or larger (dark yellow) in the current trial. We only found a significant difference in the session in which subjects were instructed to only perform voluntary saccades. Error bars represent the standard error of the means.

We performed the same analysis for the intercepts of the fits (see Figure 5) to investigate the deviation error in the current trial, implying no influence of the previous trial. Independent t-tests on the intercepts indicate only a significant difference for the voluntary saccade type, *t*(22) = 4.85, *p* < .001 (trial_{n-1}: delayed, trial_{n}: delayed: *t*(22) = 1.51; *p* = .143; trial_{n-1}: voluntary, trial_{n}: delayed: *t*(22) = 1.17; *p*

= .254; trial$_{n-1}$: delayed, trial$_n$: voluntary: $t(22) = 0.61$, $p = .549$; see also Figure 5). We find higher trial-by-trial influences for undershooting saccades compared to overshooting saccades.

Figure 5

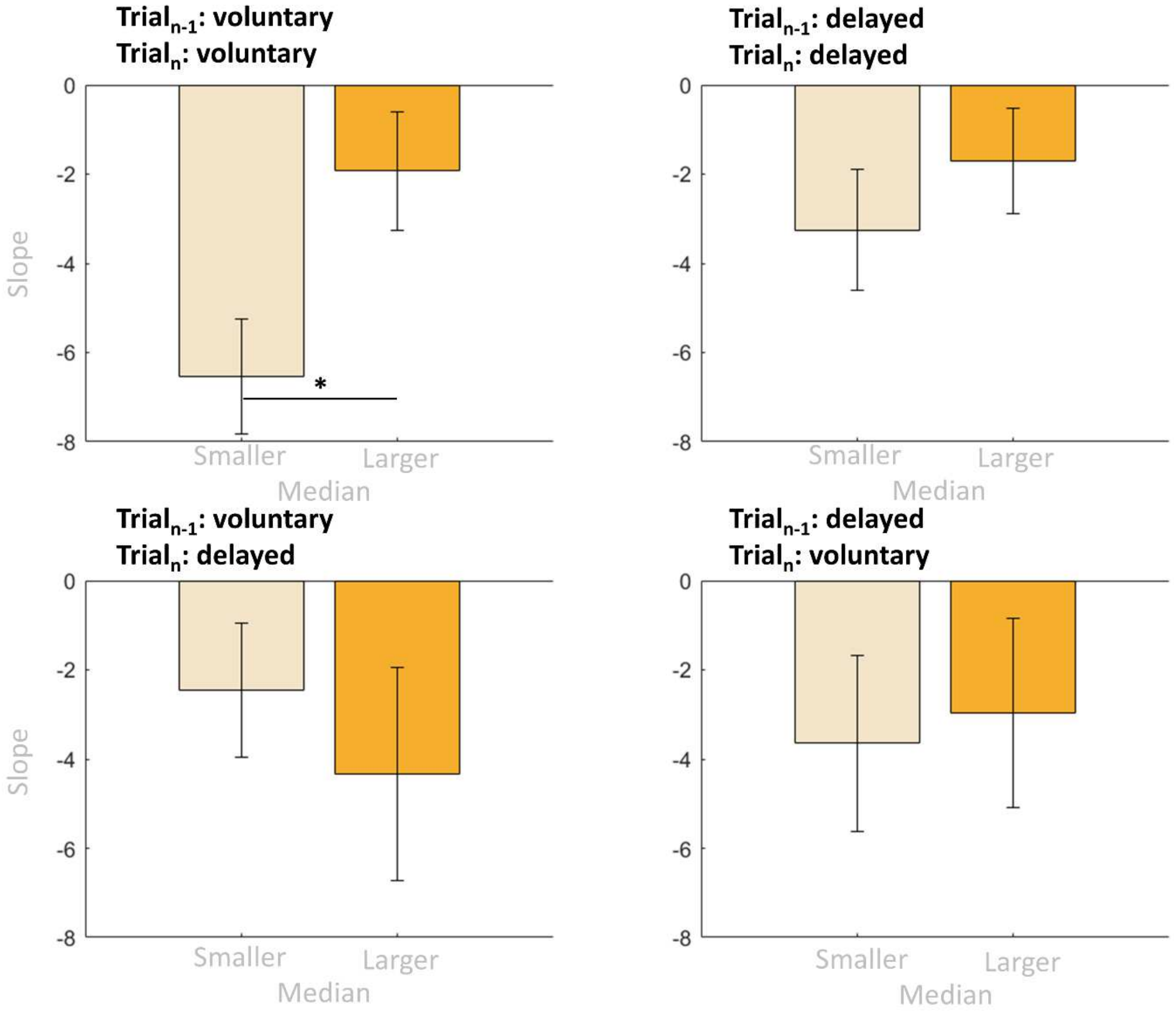


Figure 5. Mean intercepts for the linear regression between the predictor ‘previous trial’ (voluntary or delayed) and the criterium ‘current trial’ (voluntary or delayed), separated for post-saccadic error trials with a saccadic amplitude smaller (light yellow) or larger (dark yellow) in the current trial. We only found a significant difference in the session in which subjects were instructed to only perform voluntary saccades. Error bars represent the standard error of the means.

Discussion

In this study we compared the influence of delayed and voluntary saccade performance on serial dependencies in saccade landings and in visual orientation judgements. We found that serial dependencies in saccade landings were selective for the saccade type. If voluntary saccades were performed in the previous and in the current trial, the magnitude of amplitude adjustments was significantly higher than if voluntary saccades were followed by delayed saccades. However, if delayed saccades were performed in the previous trial, amplitude adjustments were equally strong irrespective if delayed or voluntary saccades were executed in the following trial.

We reasoned that the asymmetric transfer could either be related to the programming of saccades (voluntary / delayed) or to the way visual attention is drawn to the saccade target (endogenous / exogenous). In the latter case, asymmetric transfer should also be observable for visual features if attended to either endogenously or exogenously. To this end we also measured serial dependency strength on visual orientation judgements for oriented targets to which subjects performed either delayed or voluntary saccades. Serial dependencies were equally strong, irrespective of the saccade types that had been performed to the targets. In summary, in our study we found serial dependencies in saccade amplitude shifts are selective for the saccade type, while serial dependencies in orientation judgements are independent of it.

The asymmetric transfer of serial dependencies in motor errors and amplitude adjustments between delayed and voluntary saccades might reflect the different programming stages for both saccade types (Deubel, 1995). If the post-saccadic error would have been processed differently due to the different covert attentional deployment, one would have expected stronger serial dependencies in one of the two saccade types. However serial dependencies were equally strong between delayed and voluntary saccades in blocks in which only one saccade type was performed.

Our data replicate the asymmetric transfer of motor error information that has been reported previously in the saccade adaptation literature (Erkelens and Hulleman, 1993; Deubel, 1995; Collins & Dore-Mazars, 2006; Alahyane et al., 2007). Our results are consistent with the idea that the differences in motor error transfer between voluntary and delayed saccades are to be found at the saccade programming stages. In an early model idea, it was suggested that voluntary saccade adaptation might reside in frontal areas and delayed saccade adaptation in the superior colliculus (Deubel, 1995). Given that the frontal areas are higher up in the hierarchy, the asymmetric transfer of adaptation would be explainable. If adaptation occurred in the frontal areas, delayed saccades, being programmed more downstream would remain uninformed of it. In the opposite case, voluntary saccade would be affected by adaptation in the colliculus, though which voluntary saccade planning signals would pass. However, neural activation corresponding to saccade adaptation does not attest to such an easy picture.

Electrophysiological studies (Métais et al., 2022; Gerardin et al., 2012; Guillaume et al., 2018) and PET studies (Desmurget et al., 1998, 2000) have highlighted the pivotal importance of the cerebellum for saccade adaptation. The cerebellum detects and processes the post-saccadic error (Herzfeld et al., 2018) and might also be responsible for amplitude adjustments. However, two patient studies found that lesions in thalamic nuclei that transport information from the cerebellum to cerebellar areas diminish saccade adaptation magnitude. A functional brain imaging in humans investigated neural activation corresponding to delayed and voluntary saccades. They found activation in middle-temporal, temporo-parietal and frontal areas for delayed saccade adaptation. Voluntary saccade adaptation included the same areas and in addition parietal areas. The authors argued that this dissociation matches the dorsal/ventral specialization of parieto-frontal streams relative to covert shifts of visual attention (Corbetta and Shulman, 2002). Guillaume et al, (2018) pointed out that neural activation in the classical saccade adaptation paradigm might result either from adaptation or from saccade error processing. Since usually an adaptation task is compared to a control task in which no intra-saccadic target displacement is applied, cortical activation might reflect the error but not the adaptation. By displacing the target only during saccade execution and then clamping the target close to saccade landing, Guillaume et al, 2018 avoided that confound. Under this condition they found activation of parietal and frontal areas involved in the adaptation of delayed saccades. The involvement of frontal areas in delayed saccade adaptation that was not attributable to mere error processing was also confirmed in an fMRI localizer study (Métais et al., 2022).

In visual orientation judgments, serial dependencies were equally strong irrespective of whether delayed saccades, voluntary saccades or an alternation of delayed and voluntary saccades were performed. The interpretation of motor-type specificity is in line with the absence of any difference between delayed and voluntary saccades on sensory serial dependencies. Serial dependencies in vision have been argued to stabilize perception by smoothening sensory input toward previous experiences.

Serial dependencies and overt attention shifts

Since serial dependencies are concerned about the interpretation of the external world, they should generalize about whether objects were attended endogenously or exogenously. It is unclear whether results observed in experiments involving covert attention shifts can be used to interpret findings from paradigms involving overt attention shifts. There is no guarantee that overt and covert attention shifts are always coupled. By contrast, Casteau & Smith (2020) provided evidence that only exogenous overt attention is coupled to eye movement programming. Endogenous overt attention however appeared to be independent of it as it could be directed to regions of the visual field that would be unreachable with eye movements. The independence of endogenous overt attention from eye movement programming might also explain the asymmetric transfer of adaptation.
In conclusion, the current study demonstrated that exogenous and endogenous attention shifts differentially affect sensorimotor serial dependencies but not visual serial dependencies.

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