Trajectory curvature in saccade sequences: spatiotopic influences vs residual motor activity.

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Abbreviated Title:

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Abstract:

When decisions drive saccadic eye movements, traces of the decision process can be inferred from the movement trajectories. For example, saccades can curve away from distractor stimuli, which was thought to reflect cortical inhibition biasing activity in the Superior Colliculus. Recent neurophysiological work does not support this theory, and two recent models have replaced top-down inhibition with lateral interactions in the Superior Colliculus or neural fatigue in the brainstem Saccadic Burst Generator. All current models operate in retinotopic coordinates and are based on single saccade paradigms. In order to extend these models to sequences of saccades, we assessed whether and how saccade curvature depends on previously fixated locations and the direction of previous saccades. With a two-saccade paradigm, we first demonstrated that second saccades curved away from the initial fixation stimulus. Furthermore, by varying the time from fixation offset and the intersaccadic duration, we distinguished the extent of curvature originating from the spatiotopic representation of the previous fixation location or residual motor activity of the previous saccade. Results suggest that both factors drive curvature, and we discuss how these effects could be implemented in current models. In particular, we propose that the collicular retinotopic maps receive an excitatory spatiotopic update from the Lateral Interparial region (LIP).

New & Noteworthy:

Saccades curve away from locations of previous fixation

Varying stimulus timing demonstrates effects of both 1) spatiotopic representation and 2) motor residual activity from previous saccades.

Spatiotopic effect can be explained if current models are augmented with an excitatory top-down spatiotopic signal.

1 Introduction

Most actions are made in sequence and typically involve the selection of one target, at the expense of irrelevant information. Response trajectories are known to reflect the dynamics of this decision process. For instance, the curvature of arm movements can reveal distractor interference (Howard and Tipper 1997; Tipper et al. 1997; Welsh et al. 1999; Chieffi et al. 2001; Chang and Abrams 2004; Welsh and Elliott 2004) and indecision or preference reversal in multi-alternative tasks (Freeman and Ambady 2010; Koop and Johnson 2011, 2013). Saccadic eye movements—although traditionally considered ballistic—may curve towards a distractor item if the target selection has not yet been fully resolved so that a distractor-related activity is still present in the oculomotor areas at saccade onset (McPeek et al. 2003; McPeek 2006). Moreover, saccades may curve away from distractor items and this is correlated with lower neural discharge at the distractor location in the Superior Colliculus (SC) compared to when the distractor is not present (McPeek et al. 2003; see their Figure 5).This phenomenon was initially thought to reflect the inhibition of distracting information (Howard and Tipper 1997; Tipper et al. 2001; McSorley et al. 2004). Consistent with this explanation, transient deactivation of a locus in SC of monkeys can cause saccade curvature away from the corresponding locus in space (Aizawa and Wurtz 1998; Quaia and Optican 1998), and in humans, early saccades were observed to curve toward the distractor, while late saccades curved away from the distractor, reflecting the putative time-course of top-down inhibition (McSorley 2006; Walker et al. 2006; Zoest et al. 2012).

However recent neurophysiological findings challenge this account (White et al. 2012). In this study, monkeys were required to perform a simple saccadic task

whilst ignoring any distractor. In trials when the distractor appeared before the target and for which saccades curve away from the distractor, White et al. (2012) expected to observe the trace of top-down inhibition at the distractor loci while the monkey was waiting for the target to appear. Contrary to these expectations, no trace of inhibition was observed during that interval in the SC. Note that this surprising finding does not contradict the earlier observations of McPeek et al. (2003; 2006), in which less activity at distractor location was reported during the saccade-related discharge. White et al. (2012) did report a similar result*after* target onset. However, there seems to be no clear anatomical candidate to send precise and spatially-tuned inhibition to the SC. Because of that and the lack of computational model that implement it, some authors have argued that top-down inhibition is essentially a "deus ex machina" which explains the deviation away using an unexplained mechanism (Kruijne et al. 2014).

There are currently two computational models that account for curvature away from a non-target signal without top-down inhibition. Wang and colleagues proposed that the curvature originates from local lateral interactions in the intermediate layer of the SC (SCi) (Wang et al. 2012; Wang and Theeuwes 2014). Alternatively, Kruijne and colleagues proposed an explanation based on a short term depression in the neurons driving the eye muscles—downstream from Superior Colliculus (Kruijne et al. 2014). These models will be described in more detail in the General Discussion. For now, we note two key features that are also shared with the top-down inhibition theory. First these models operate entirely in retinotopic coordinates; hence, they currently do not account for spatiotopic influences (i.e. signals that remain in world coordinates). Secondly these models were built to explain single-saccade paradigms, and currently do not account for any deviation influence arising from previous saccades. Our study aims to address the presence of both influences in a two-saccade para-

digm in order to direct potential extensions of the current models to account for sequences of saccades.

Studies of free viewing or visual search have shown that, in sequences of saccades, previously fixated locations may influence saccadic behavior in a spatiotopic frame and in an automatic way (Klein and MacInnes 1999; Sogo and Takeda 2006; Smith and Henderson 2011, 2011; Bays and Husain 2012). One obvious example is Inhibition of Return (Posner and Cohen 1984; Sumner 2006), where it can take longer to initiate saccades directed back to a previously fixated location compared to other directions (Klein and MacInnes 1999; Hooge and Frens 2000; Hooge et al. 2005; Ludwig et al. 2009; Farrell et al. 2010). However, it is currently unclear whether and in what way IoR and saccade curvature are related. Godijn and Theeuwes (2004) suggested that saccadic curvature and (covert) IoR are based on different mechanisms. Importantly, another set of studies, using single-saccade paradigms, have suggested that saccades tend to curve *away* from memorized stimuli either in retinotopic space (Theeuwes et al. 2005) or in object-centered space (Boon et al. 2014). Furthermore, curvature away was found from the representation of the distractor location in previous trials (Van der Stigchel and Theeuwes 2006). This work highlights that past stimuli can influence the trajectory of the current saccade and that this influence is not necessarily coded in retinotopic space. That naturally paves the way for exploring the effect of memory traces in sequences of saccades.

In this regard, the study of saccade trajectories during visual search is relevant (Sogo and Takeda 2006). These authors demonstrated that saccades tend to curve away from the spatiotopic representation of previous fixation zones and suggest an effect of the 3 last fixation zones. However, these results could support either spatiotopic representations of previous stimuli, or motor residual activity from the direction of previous saccades. Indeed, it has been suggested

that saccades can allow for residual activity to persist in the motor map after their completion—particularly, that motor residual activity would facilitate successive saccades in the same direction (Klein and MacInnes 1999; Anderson et al. 2008; Smith and Henderson 2009, 2011; Wang et al. 2011). In other words, in Sogo and Takeda (2006), the current saccade might curve away from the previous fixation because the vector of the previous saccade was, by definition, pointing away from that previous fixation, and this vector remains partially active or facilitated.

A more direct test for the effect of automatic spatiotopic representations on saccade curvature was performed recently by Jonikaitis and Belopolsky (2014). Participants executed two saccades: the first rightward or leftward while the second was upward or downward. Before the initiation of the first saccade, a distractor briefly occurred to the left or to the right of the vector of the second saccade, so that the first saccade dissociates the retinotopic and spatiotopic locations of that distractor. Curvature in the second saccade appeared to depend on the spatiotopic location—they deviate leftward for the rightward distractor and vice versa—and thus may challenge purely retinotopic views of saccade trajectory curvatures. However, there is still room for a retinotopic explanation of Jonikaitis and Belopolsky's data. First, both models can produce larger deviation with larger inter-stimulus distances (more detailed in Discussion). Second, if there is some residual motor activity caused by the first saccade, this would induce a deviation in the direction of the first saccade (see **Figure 2**B). Consider how these two factors might interact, with illustration of a “right-then-up” trial. A distractor to the right of the second saccade vector must appear in a more eccentric location from the initial fixation point than a distractor to the left of the second saccade vector. Retinotopically, both distractors are rightward, predicting leftward curvature, but the most eccentric stimulus can produce stronger curvature in the models. In parallel, the assumption of residual

motor activity from the first saccade would add an equal tendency of rightward curvature to both situations. It is plausible that for a leftward distractor (which has a weak influence), the residual motor activity would be dominant, leading to curvature to the right while, for a rightward distractor (which has a strong influence), the residual motor activity would *not* prevail, resulting in curvature to the left. Thus, Jonikaitis and Bolopolsky (2014)'s data could be explained by a particular combination of these retinotopic effects.

In order to extend the work of Jonikaitis and Bolopolsky (2014) and Sogo et al. (2006) and test without ambiguity the influence of spatiotopic representations and motor residual activity, we developed a simple two-saccade paradigm without any distractor. First, we established that the second saccade in our sequence curves away from the location of the initial fixation stimulus, consistent with either of these mechanisms. Second, we distinguished these mechanisms through varying the time of the second saccade onset from 1) the fixation offset and 2) the first saccade offset.

2 Method

2.1 Participants

Fourteen observers (25-30 years old, nine male) with normal or corrected vision, participated in this experiment, which was performed with approval from the ethics committee of Cardiff University School of Psychology. All but one (the first author) were naïve to the purpose of the experiment and received payment for their time.

2.2 Procedure and Stimuli

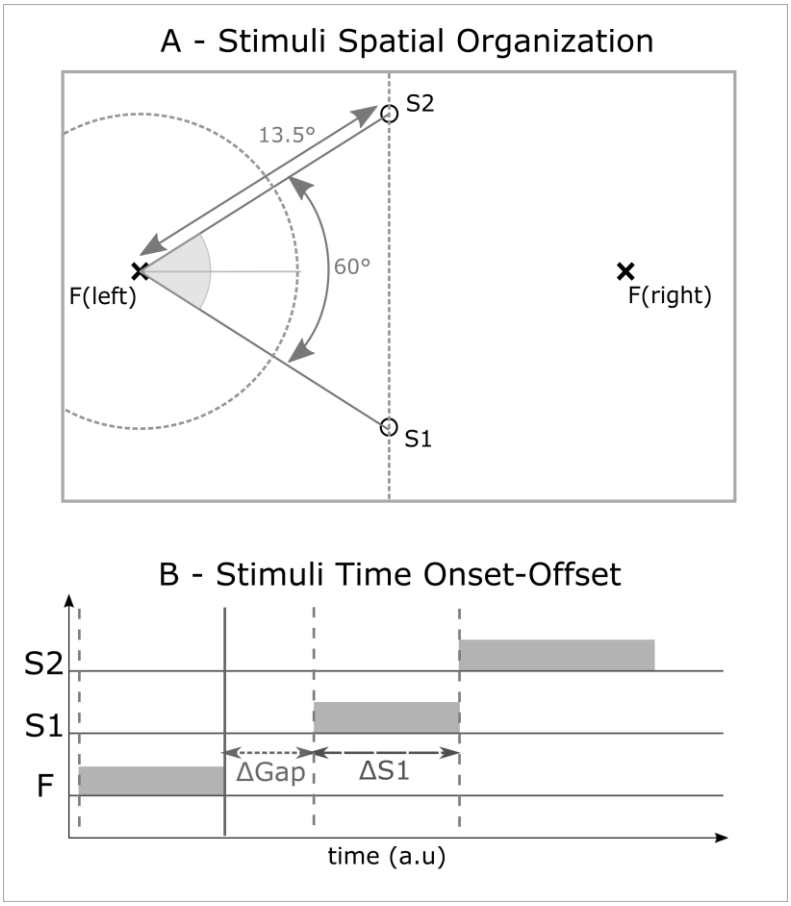

Figure 1: Description of the Stimulus Presentation*. The expressions F, S_1 and S_2 refer to the Fixation Cross, stimulus 1 and stimulus 2, respectively. The expression ΔGap refers to the duration of the gap between F and S_1 while ΔS1 refers to the duration of S1 presentation. In A, only one of the Fixation stimuli — F(left) or F(right) — is shown during a trial. The lines in gray and dashed gray are used to highlight the relative positions between stimuli and were not presented to the participant.*

There were three types of trials: control trials, single stimulus trial, and double stimulus trials, which will be described below. The control trials were present in case we needed a reference to compute the curvature of saccades. It turned out we did not need such a reference, so these trials are not considered in our analyses and report. The single stimulus trials were used to prevent the participant anticipating a second saccade, and are also not analyzed. A participant would complete two experimental sessions of approximately 1 hour, separated by at least one night. Each session consisted of setting the chair and chin-rest for the participant to sit comfortably; a 13-point calibration of the Eyelink 2000 Eye tracker; 160 control trials; 640 trials mixing randomly single-stimulus and double-stimuli trials. A break was suggested to the participant every 200 trials, and re-calibration was conducted every 400 trials.

Figure 1A and B summarize the spatial and temporal configuration of the stimuli. For single and double stimulus trials, the participant was required to fixate a "+" fixation cross (*F* in **Figure 1**) of radius 0.2° on the screen. The fixation cross could appear either on the left or on the right of the screen, along the horizontal axis. The participant pressed the space bar to confirm fixation after which the fixation cross disappeared at a random time drawn from a uniform distribution *U(*500 ms, 1100 ms*)*. Following an optional gap target S1 was presented: a circular stimulus of radius 0.4°. It could appear either on the top or the bottom of the screen, along the vertical axis. In the double stimuli trials, the presentation of S1 was followed by the presentation of S_2 which was the vertical mirror image of S1 with an angular distance of 60° (i.e., using the Fixation as origin, if S1 is at -30° of directional angle, S2 will be at 30°). S_1 and S_2 were al-

ways at 13.5° of eccentricity from fixation on both single and double step trials. In the control trials, the participants were simply making saccades from S1 to S2 locations and vice versa.

As justified in the next section, we manipulated the Gap and S1 durations in a 2x2 design (short/long S1 and short/long Gap). For short S1 trials, S1 duration was randomly taken from a uniform distribution between 250 ms and 450 ms, while for long S1 trials it was taken between 550 ms and 750 ms, so that duration could not be anticipated even when the short duration had passed. For short Gap trials, the Gap duration was randomly selected from a uniform distribution between 0 ms to 200 ms while for long Gap trials, the Gap duration was picked between 300 ms to 500 ms. Note that the change in duration between short and long conditions is the same for Gap duration and S1 duration (300 ms). Each condition had an equal number of trials and these were randomly inter-mixed, independently for each participant.

All code for running the experiment, the data and analysis scripts can be found on the Open Science Framework at *https://osf.io/t96t2*.

2.3 Hypotheses: Predicted effects of spatiotopic representations or residual retinotopic motor activity.

Our pilot studies made us confident that the second saccade would observably curve away from the previously fixated stimulus (as will be demonstrated in Results below). However, such curvature could be equally explained by a spatiotopic representation of the previous fixation, or residual motor activity from the first saccade (**Figure 2**A and B). Our experiment was designed to discriminate between these mechanisms by separately adjusting S1 and Gap durations in a 2x2 design.

Importantly, we assumed that the curvature of the saccade is proportional to the sum of the effect of both mechanisms. **Figure 2**C illustrates this point for the case where the effect of the previous fixation (F) and the effect of the residual activity (M) both decrease with time.

Figure 2C shows that the effect of motor residual is affected by the time between Saccade 2 and Saccade 1, while the effect of the previous fixation depends on the time between Saccade 2 and Fixation offset. On the one hand, increasing the Gap duration prolongs the time between Saccade 2 and Fixation offset while keeping the intersaccadic interval (between Saccade 1 and Saccade 2) unchanged (we will test the extent to which this assumption holds below). In other words, Gap duration can be used to test for an effect of the previous fixation (F) only. On the other hand, increasing S1 duration extends both the intersaccadic interval and the time between Saccade 2 and Fixation offset, which affects both the effect of the previous fixation (F) and motor residual activity (M). In other words, S1 duration *cannot* be used on its own to test an effect of residual motor activity (M).

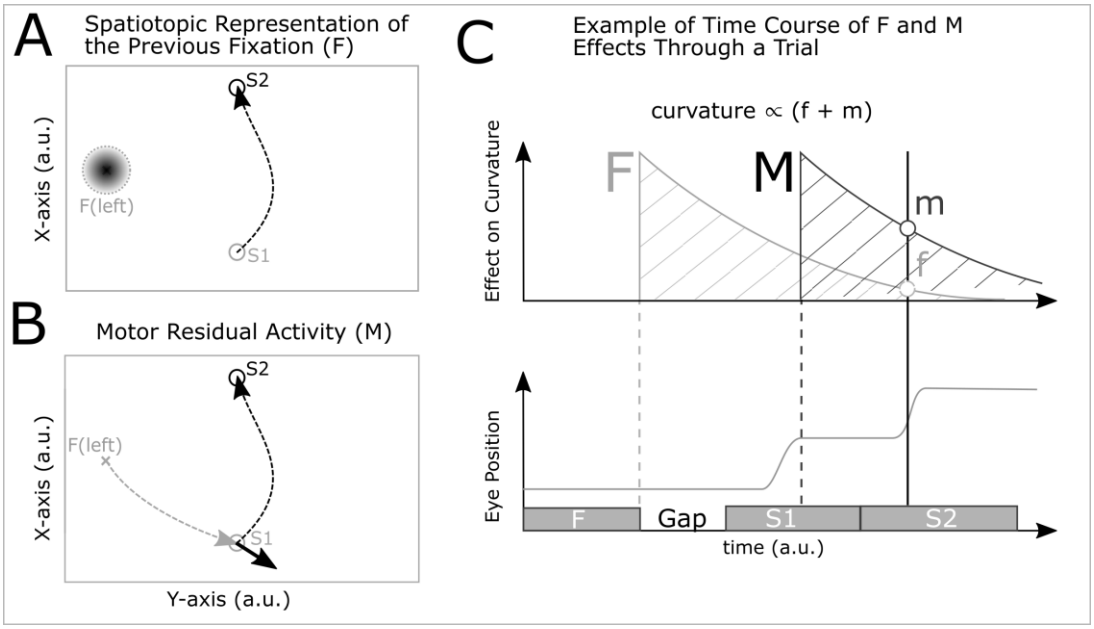

Figure 2: Predicted Effect of the Spatiotopic Representation of the Previous Fixation (F) and of the Motor Residual Activity from Saccade 1 (M) on Saccade 2's curvature. *Although both mechanisms are expected to curve the second saccade (dashed black line, in A and B) away from the previously fixated location, their time courses can be used to distinguish between them (C).* ***In A****, the saccade curvature would be caused by the memorized representation of F(left) (depicted as a black Gaussian gradient) while* ***in B****, the saccade curvature would be caused by a residual trace of the Saccade 1 vector (thick black arrow; the dotted gray curve is Saccade 1) during the execution of Saccade 2 (dotted black line).* ***In C****, we highlight that the time course of each mechanism is attached to a different event in the trial. The time course of the effect of F (bright gray curve) is linked to the Fixation offset (bright gray dashed vertical line). The time course of the effect of M (dark gray curve) is linked to Saccade 1 offset (dark dashed vertical line). Finally, the curvature of Saccade 2 depends on the sum of the effect of F and M (white dots f and m) at the time of Saccade 2 onset (thick black vertical line). In* ***Figure 3****, we will see that varying Gap and S1 duration can allow us to distinguish between the two mechanisms.*

This can be solved by choosing carefully a 2x2 design with short/long S1 durations and short/long Gap durations. **Figure 3** illustrates, for each condition, the intersaccadic intervals, the time since Fixation offset and how the time course of the effect of both motor residual activity (M) and previous fixation (F) would

affect the curvature of Saccade 2 (last row). We chose the durations of S1 and Gap so that the combinations "*long Gap / short S1"* and *"short Gap / long S1"* both give a similar time between Saccade 2 and Fixation offset (we will assess the extent to which this assumption holds below). Thus, in these conditions, mainly the intersaccadic interval is changed, allowing us to test for an effect of motor residual activity (see dark gray lines in last row, column 1, Hypothesis 1). An effect of Fixation only (see light gray line in last row, column 2, Hypothesis 2) would lead to an effect of Gap and S1 duration, but no difference between the conditions "*long Gap / short S1"* and *"short Gap / long S1".* Finally, an effect of both Fixation and motor residual activity would lead to an effect of Gap and S1 duration and a difference between the conditions "*long Gap / short S1"* and "*short Gap / long S1"* (column 3, Hypothesis 3). Importantly, similar effects were predicted with linear decays and increase functions while the effect sizes varied with the parameters of the functions (more figures and source code accessible online).

It is noteworthy that we do not assume any direction concerning the time course of the effects and our paradigm is tailored to inform us on their direction. In **Figure 3**, if the motor residual activity increases with time, then the related trend line (dark gray line in last row) will have a positive slope. Similarly, if the effect of Fixation increases with time, then the related trend lines (light gray line in last row) will have a positive slope.

Importantly, if the effect of Fixation and of the motor residual activity progresses in the same direction over time, an alternative way to check for an effect of motor residual activity is to test whether the effect of S1 duration is greater than the effect of Gap duration (rather than equal, see **Figure 3**, column 3, last row). That is due to the fact that a change of S1 duration affects both the effects of Fixation and motor residual activity (as seen with **Figure 2**).

To summarize, our paradigm can discriminate between three hypotheses in addition to the null hypothesis. **Hypothesis 1:** only the residual motor activity of the previous saccade has an effect. **Hypothesis 2:** only the spatiotopic representation of the previous fixation has an effect. **Hypothesis 3:** both the spatiotopic representation and residual motor activity have an effect. It can also differentiate between an increasing and a decreasing time course of each effect.

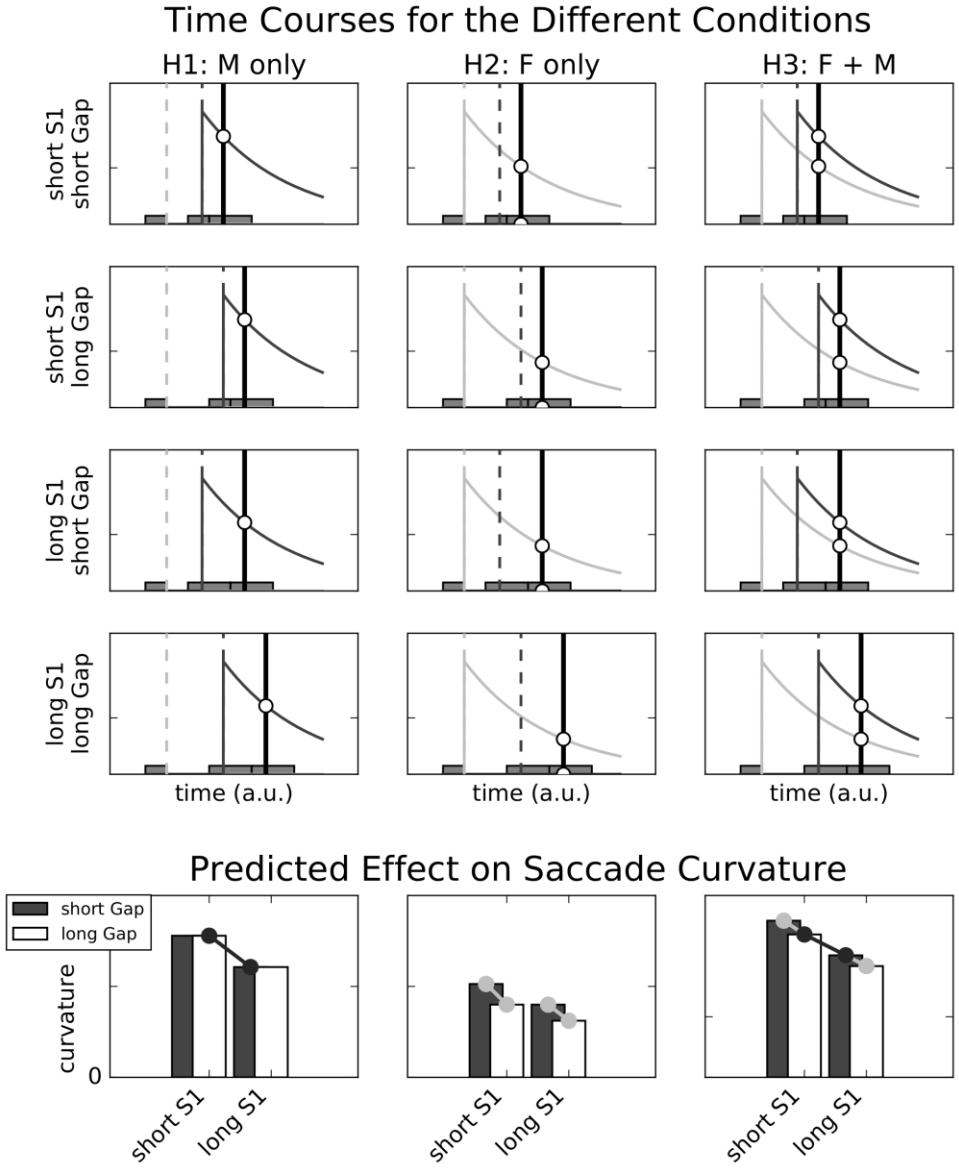

Figure 3: How our Paradigm Distinguishes the Effects of Motor Residual Activity (M) and of the Spatiotopic Representation of the Previous Fixation (F). *The paradigm design can differentiate between an effect of F and M, and also between increasing and decreasing time courses.* ***Row 1-4:*** *Each row represents a condition of our paradigm while Columns 1 consider a time dependent effect of M with no effect of F and Columns 2 consider a time dependent effect of F with no effect of M. Column 3 considers an effect of both F and M. The subplots used a similar representation as seen in* ***Figure 2****C. The effect of M and F are represented, respectively by dark and bright gray curves (exponential based in this example). The small gray boxes at the bottom represent the stimuli timing. The bright dashed line, the dark dashed line and the solid thick line represents, respec-*

tively the Fixation offset, the Saccade 1 offset and the Saccade 2 onset. The white dot is particularly important as it represents the effect of M and F at Saccade 2 onset. ***Row 5*** *summarizes the height of the white dot in row 1-4 (i.e. the effect of M and F on Saccade 2's curvature at Saccade 2 onset) for each condition. A positive number denotes a curvature away from previous fixation. It is important to note that the trend in condition shortS1/longGap and longS1/shortGap (depicted with two dots linked by a black line) is a good marker of an effect of M. This marker of M will not be affected if there is an effect of F in any direction (i.e. if we sum the bars in Column 1 and 2 with the bars of Columns 3 or 4). Similarly, an effect of Gap duration (depicted with two dots linked by bright line) is a good marker of an effect of F. Finally, if there is an effect of both M and F that goes in the same direction (e.g. decreasing), the effect size of S1 duration should be greater than the effect size of Gap duration.*

2.4 Data Analysis

A saccade was marked for analysis if the acceleration was greater than 6,000 $°.s^{-2}$, the absolute velocity was larger to $10°.s^{-1}$ and the amplitude was larger than 5.4°. A trial was rejected if: no saccade was made, or two saccades were made to reach a stimulus, the reaction time or intersaccadic time was shorter than 80 ms, a saccade duration was longer than 150 ms, or a saccade contained eye positions outside the screen or missing data.

In our experimental design, the selection of one hypothesis (see previous section 2.3) over another may be based on the *absence* of an effect (i.e. a null effect). The Bayesian framework provides one way to assess the graded evidence in favor or against the influence of some experimental factor (Wagenmakers 2007; Rouder et al. 2009; Morey and Rouder 2011). Thus, we employed the Bayes Factor framework for analysis of our data (Rouder et al. 2012; specifically the R package BayesFactor; Rouder and Morey 2012). Furthermore, Bayes Factors are very useful in order to test models against each other and/or select the best model as they penalize complexity (Raftery 1995).

The analysis proceeded in three steps. First, we demonstrate that the second saccades curved away from the spatiotopic location of the Fixation stimulus (replicating pilot experiments that showed this on a small sample of partici-

pants). We simply selected, based on the Bayes Factor (BF), the best model that explains the initial deviation (see **Figure 4** for the precise measure) among models combining effects of Participant and Fixation side. That analysis used the trial-by-trial initial deviations of the participants (~125 data points per participant per condition).

In a second step, we checked that the assumptions we made on the consistency of saccade latencies and durations across conditions were met. Importantly, we needed to make sure that: 1) the time onset of Saccade 2 since the Fixation offset is similar between the conditions shortGap/longS1 and longGap/shortS1; 2) the intersaccadic time is similar between shortGap and longGap conditions. We used within-subject Bayesian 2x2 ANOVAs to check these requirements.

In a third step, we tested the hypotheses outlined in the previous section to discriminate the effect of motor residual activity from the effect of the spatiotopic representation of the previous fixation. For simplicity and better readability of the results, we collapsed the data so that we obtained the mean difference in initial deviation between the conditions Fixation left and Fixation right (abbreviated to IDD_{LR}) for each participant and each condition (i.e. Gap/S1 durations). To test an effect of the Fixation, we ran a Bayesian top-down analysis that assesses the importance of Gap and S1 duration in explaining our data. Specifically, a full model that considers all the variables and interactions is tested against models that omit each of the independent variables (ΔGap, ΔS1), random variables (Participant), and their interactions (see Figure 7 and ***Table 1***). Thus, the full model we used was the following general linear model:

IDD_{LR} ~ *S1.Duration + Gap.Duration + Participant + S1.Duration:Gap.Duration + S1.Duration:Participant + Gap.Duration:Participant + S1.Duration:Gap.Duration:Participant.*

Then, to assess an effect of the motor residual activity of the previous saccade, we tested the effect direction between shortS1/longGap and longS1/shortGap and whether the effect size of S1 duration is greater than the effect size of Gap duration.

We matched the BFs with the interpretation tags of Raftery (1995; see also Kass and Raftery 1995). These tags are written in italics. For readers preferring null hypothesis significance tests, these can be found on the OSF repository and support the same conclusion.

3 Results

The average rejection rate of trials was 27 % (the rejection rules can be found in section 2.4. We rejected in total 3 participants based on their proportion of rejected trials (greater than 40%; we aimed to get at least 50 data points in each cell of the design to allow for robust estimates of measures of central tendency of latency, duration, and curvature), concluding that the gap was too disruptive to their performance (anticipatory saccades) or that the eye-tracker was not recording properly (missing data).

3.1 Saccade curvature away from the previous fixation point

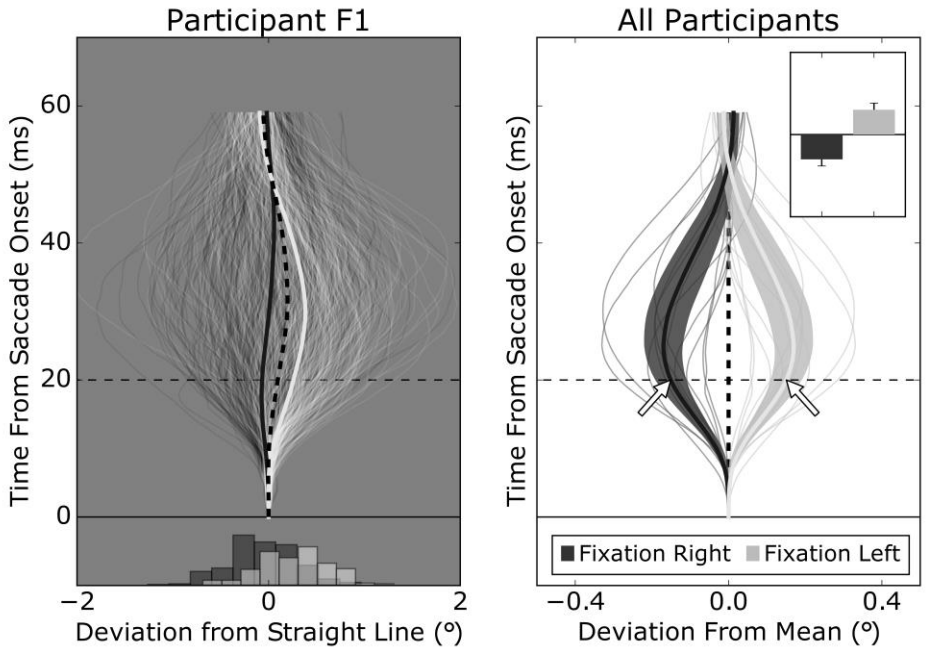

Figure 4: Effect of fixation side on the second saccade curvature. *The dark solid curves and bars are associated with the condition where the Fixation was on the right, while the brighter ones are associated with the left condition.* ***Left Panel:*** *the plot is made from the data of one participant. The thin curves represent the distance from the straight line (i.e. deviation) of the second saccade over time for each trial, per condition. The thick and solid curves represent the average deviation across trials, per condition. The thick dashed line is the mean deviation across both left and right conditions. Negative values are on the left of the straight line while positive values correspond to the right. The* ***initial deviation*** *reported in this paper corresponds to the deviation measured at 20 ms from the saccade onset (indicated by the horizontal dash line). From the histograms of the initial deviation (bottom), it can be observed that the saccade in the right condition (dark bars) are deviating more leftward than the bright curves (bright bars).* ***Right Panel:*** *the solid dark and solid bright curves represent the average deviation from the participant mean across all participants, when, respectively, the Fixation was presented on the right and on the left. The vertical thick dashed lines in the left and right panels represent the same thing; that is the participant average across left and right conditions.*

Figure 4 reveals that the second saccade clearly curves away from the initial fixation position at the participant level (left subplot) and at the participant average level (right subplot). The inset of the right subplot shows the mean saccade deviation at 20 ms from saccade onset, averaged over the participants,

with 95% confidence intervals. Clearly, the deviations are significantly more rightward when the fixation is on the left (brighter bars) and more leftward when the fixation is on the right (darker bars). These impressions of the data were confirmed by the Bayes Factor analysis—the model that includes Fixation side and Participant was unambiguously better than the model with Participant only (BF > 1000). The model with an interaction between Participant and Fixation side was classed as the best model (BF > 1000 against the main effect model) suggesting inter-individual differences in the effect of Fixation side.

3.2 Intersaccadic intervals and second saccade latency

It is worth recalling that a good data set for testing our hypotheses should show:

1. An effect of S1 Duration but no effect of Gap Duration on the intersaccadic interval,
2. A similar distribution of the time interval between Fixation offset and Saccade 2 onset when comparing *"long S1 / short Gap"* with *"short S1 / long Gap"* conditions.

The data broadly met those requirements. **Figure 5**A shows the latency of the second saccade relative to the first saccade offset. A Bayesian 2x2 within-subject ANOVA on the intersaccadic intervals, revealed an effect of Gap Duration (BF >1000 against a Gap Duration omission). However, this effect is very small compared to the effect of S1 Duration— i.e., 9 times smaller (267 ms against 31 ms on average). **Figure 5**B shows the latency of the second saccade relative to fixation offset. Again, although a Bayesian t-test reveals a difference in the time from Fixation Offset when comparing *"short Gap / long S1"* with *"long Gap / short S1"* (BF > 1000 against null slope), this difference is 10 times

smaller than the main effects of S1 Duration and Gap Duration (301 ms for Gap Duration, 272 ms for S1 Duration against 30 ms for the analyzed slope).

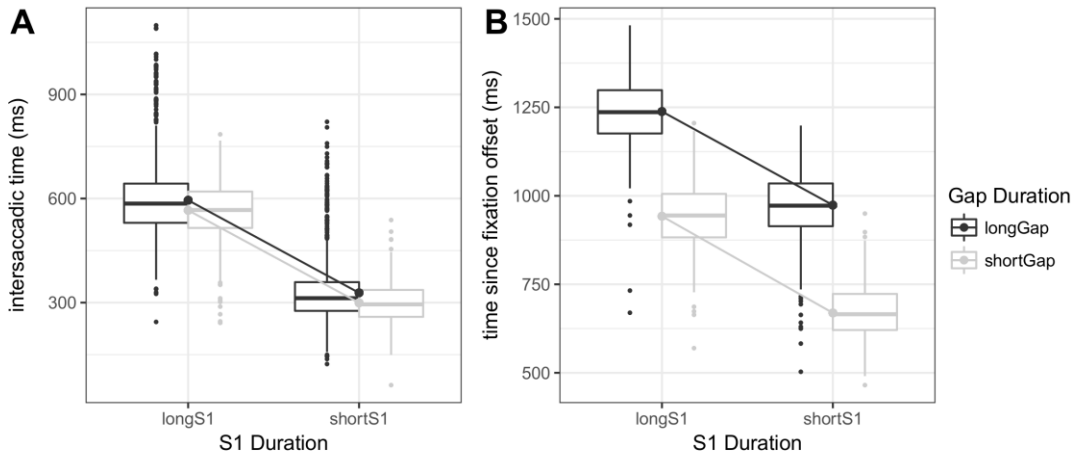

Figure 5: Interaction Boxplots for the Inter-saccadic time between Saccade 1 and Saccade 2 and for the time interval between Saccade 2 onset and Fixation offset. *Note that a within-subject correction (Cousineau 2005) was applied to the data to illustrate that the analysis treated the participant as a random effect. In both A and B, the lower and upper hinges correspond to the first and third quartiles. The lower and upper whisker extend from the hinge to the lowest/highest value within 1.5 times the inter-quartile range, so that the trials beyond these whiskers—plotted as points—can be considered as outliers of a normal distribution. The lines are connecting the mean of the distributions.*

3.3 Testing the Origin of the Fixation Side Effect

Figure 6 presents a summary of the data that can be compared directly to the predictions presented in **Figure 3**. At first glance, there seems to be an effect of Gap and S1 duration, which suggests an effect of the previous fixation, while the conditions short S1/long Gap and long S1/short Gap look different, which suggests an effect of the motor residual activity of the previous fixation. The general pattern of results support a decreasing time course of both effects.

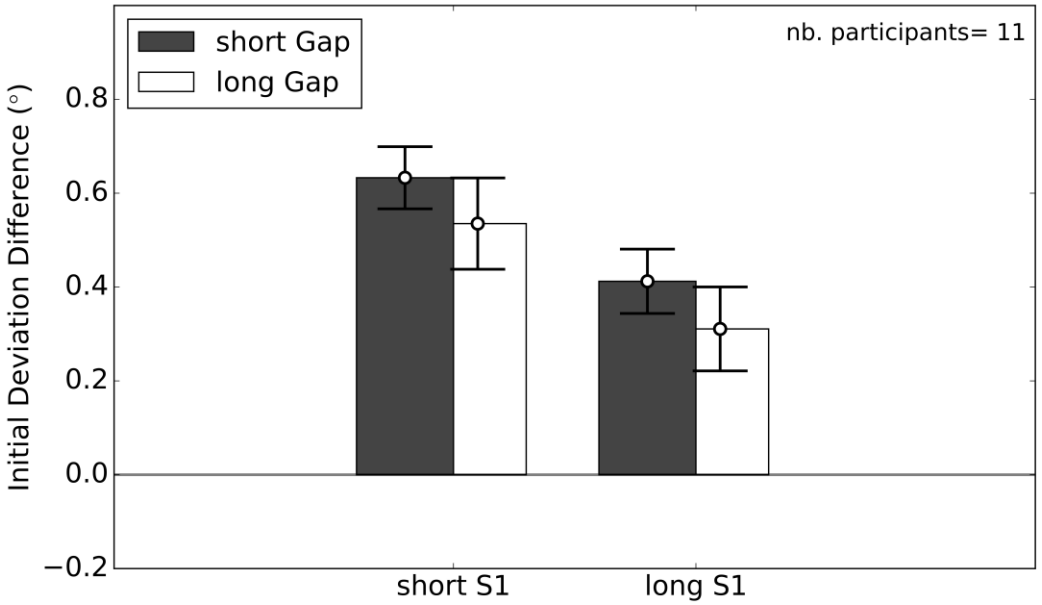

Figure 6: Summary of the Data Analyzed. *Error bars display the within-subject 95% confidence intervals. Note that IDD_{LR} stands for the difference in initial deviation between the conditions Fixation Left and Fixation Right.*

Table 1 shows the results of the Bayesian Top-down analysis. The polarity tag *in favor* means that to omit the variable is detrimental to the full model— i.e. the evidence is *in favor* of an effect of the variable. Matching the BFs with the interpretation tags of Raftery (1995), we can see that there is *positive* evidence in favor of an effect of both Gap and S1 durations. The model is also improved by including some differences between participants in the effect of S1 duration. The best model reported by the analysis is the following:

$IDD_{LR} \sim S1.Duration + Gap.Duration + Participant + Participant:S1.Duration$

Where IDD_{LR} stands for the difference in initial deviation between the conditions Fixation Left and Fixation Right. Thus, our analysis, by suggesting an effect of both Gap and S1 duration, is supportive of an effect of the spatiotopic

representation of the previous fixation (see **Figure 3**, last row). To test the direction of the effect of Gap (longGap – shortGap), we ran a one-sided paired t-test on the distributions for longGap and short Gap conditions. When tested against the null, the BF of the effect of Gap being positive is 0.06 (+-0.1%) while the BF of being negative is of 20.7 (+-0%). Overall, the BF of being negative against being positive is very strong (combined BF = 20.7/0.06 = 321). We read the combined BF as very strong evidence of an asymmetry favoring negative values; that is supportive of a decrease of the Fixation effect over time.

Table 1: *Bayes factor top-down analysis on Initial Difference in Deviation (Left-Right).*

	Effect of Omission	BF or 1/BF		Polarity	Interpretation Tag
[1]	ΔGap:ΔS1:Participant	1.02	±5.26%	none	weak
[2]	ΔGap:Participant	3.88	±4.26%	against	positive
[3]	ΔS1:Participant	>1000	±4.65%	in favor	very strong
[4]	ΔGap:ΔS1	2.37	±5.96%	against	weak
[5]	Participant	>1000	±5.19%	in favor	very strong
[6]	ΔGap	5.1	±6.07%	in favor	positive
[7]	ΔS1	4	±4.46%	in favor	positive

Note. We inversed (1/BF) the BFs less than 1 for easier reading. We add a Polarity column that tells if the evidence is against or in favor of an effect of the omitted variable. BF against the full model: IDD_{LR} *~ ΔS1 + ΔGap + Participant + ΔS1:ΔGap + ΔS1:Participant + ΔGap:Participant + ΔS1:ΔGap:Participant. Where* IDD_{LR} *stands for the difference in initial deviation between the conditions Fixation Left and Fixation Right.*

Now that we have strong evidence for an effect of the spatiotopic representation of the Fixation, we need to discriminate between Hypothesis 2 (Effect of Fixation only) and Hypothesis 3 (Effect of Fixation and motor residual activity).

As explained in section 2.3, more tests are needed to assess the effect of the motor residual activity of the previous saccade. One way is to compare the

longS1/shortGap and shortS1/longGap conditions (see **Figure 3**, last row, dark gray lines), so we ran a paired one-sided t-test on their distributions. When tested against the null, the BF of (longS1/shortGap - shortS1/longGap < 0) is 1.26 while the BF of (longS1/shortGap - shortS1/longGap > 0) was 0.14. In other words, our data does not provide enough evidence to distinguish between no effect and decreasing effect of motor residual activity over time (i.e. the time since fixation being controlled). However, the data contains positive evidence against an increasing effect. That asymmetry between the two t-test leads the combined BF testing for the effect being negative rather than positive to be 1.26/0.14 = 9, which is positive evidence in support of a decreasing effect. Hence, although we would need more data to settle unambiguously whether there is a decreasing effect, the asymmetry between the two t-test is an encouraging result.

As there is some evidence that the fixation effect and the motor residual effect go in the same direction over time (or, at least, not in opposite directions), we expect the effect size of S1 to be greater than the effect size of Gap if a motor residual activity is indeed present (see section 2.3). We computed the distribution of non-standardized effect sizes for S1 (i.e. short S1 – long S1) and for Gap (i.e. short Gap – long Gap) and we ran a one-sided paired t-test on them. We are here mostly interested in (S1 effect > Gap effect) against the null (S1 effect = Gap effect), for which the BF is 2.89. That represents weak evidence in favor of an effect of motor residual activity.

Finally, **Figure 7** illustrates the difference in effect size by sampling these effects from the posterior distribution of the best model. When comparing the two subplots, the effect of S1 duration appears to be greater, but also more variable than the effect of Gap duration. Recall that, under Hypothesis 3, S1 duration effect would be the sum of the effect of Fixation and motor residual activity, while Gap duration effect only depends on the effect of Fixation. This sum of

two effects would lead to a greater effect and greater variance for S1 duration. In other words, the posterior distribution is such as expected under Hypothesis 3.

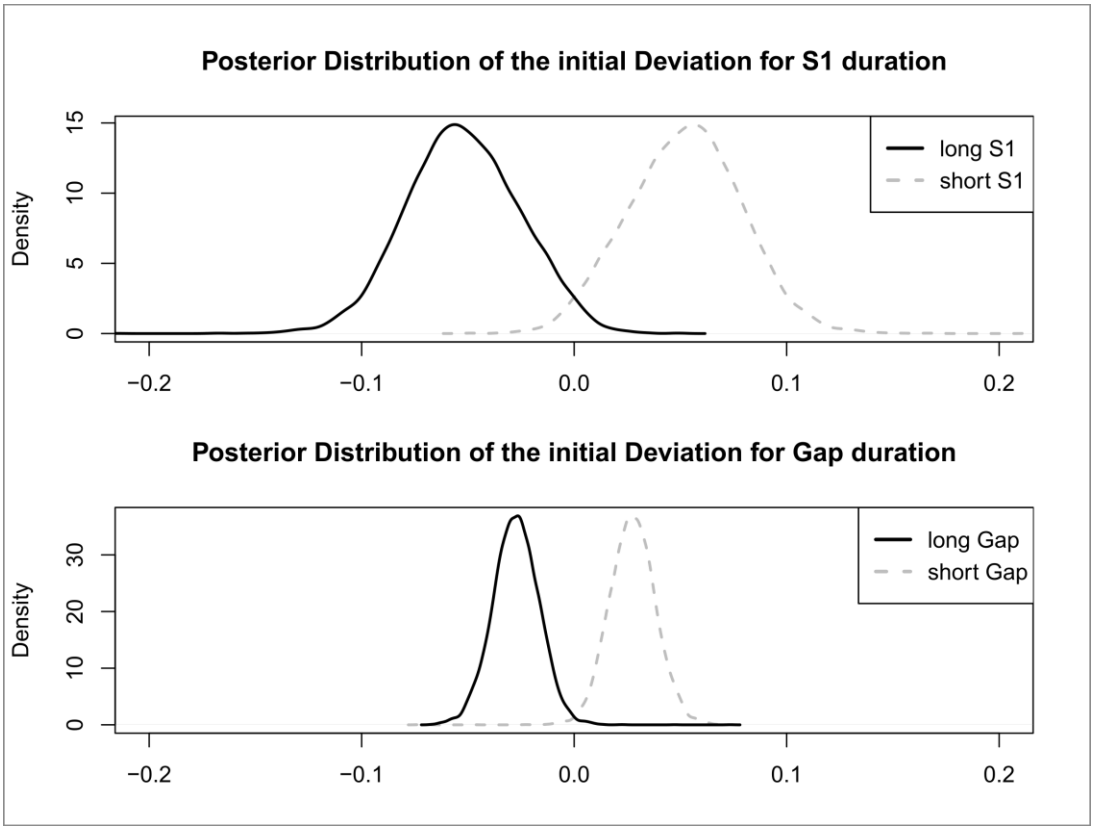

Figure 7: Estimation of the non-standardized effect size of Gap and S1 duration on IDD_{LR} *(i.e. the difference in initial deviation between Left and Right Fixation conditions). We plotted the distribution of the non-standardized effect size of S1 and Gap duration from sampling 10,000 points from the posterior distribution of the best model (see main text). Two observations can be made: 1) both S1 and Gap duration have a negative effect on IDD_{LR} (i.e. as we increase Gap or S1 duration, the distribution shift leftward), and 2) the effect of Gap duration on IDD_{LR} seems smaller than the effect of S1 duration.* ***Top:*** *Kernel density bandwidth of 3.816e-03.* ***Bottom:*** *kernel density bandwidth of 1.533e-03.*

To conclude, the data provide some support for **Hypothesis 3** over **Hypothesis 2** while rejecting **Hypothesis 1**. In other words, the curvature away that we observed is caused by both a spatiotopic representation of the previously fixated

location and a motor residual activity from the previous saccade. Furthermore, the effect of the previous fixation and of the motor residual activity decreases with time in the interval under consideration here.

4 Discussion

Analyzing trajectory curvature during a sequence of saccades allowed us to answer whether there is a need to extend recent computational models of saccade curvatures that are based on retinotopic brain regions (Kruijne et al. 2014; Wang and Theeuwes 2014). These models that were built to explain trajectory curvatures in single-saccade paradigm and thus could not predict influence of 1) the spatiotopic representation of previous stimuli and/or 2) previous saccades on the current saccade trajectory that may happen during sequence of saccades. Using a two-saccade paradigm, we demonstrated an influence of both these factors and suggested that their influence decreases with time. Such a decreasing time course is expected for a residual motor signal, but it might be surprising for a memorized, spatiotopic representation. Indeed, previous studies that tested the spatiotopic representation of peripheral stimuli at a shorter time scale than ours reported increasing curvature with time (Jonikaitis and Belopolsky 2014). However our results are in agreement with work that tested the representation of previous fixations—as in our experiment—at a similar time scale as ours (Sogo and Takeda 2006; see their Figure 8). In the next sections, we will discuss how the current models of saccade curvature can be updated in order to explain our results.

4.1 Prediction of Kruijne et al. (2014)'s model

The model of Kruijne et al. (2014) is based on fatigue (resembling Short Term Depression, a decrease in the neuronal sensitivity following sustained input) occurring in the brainstem. They assume one neural population per saccadic direction (left, right, up, down) and a fatigue mechanism in the Long-Lead-Burst neurons (LLBNs). The LLBNs are known not to be inhibited by the omnipause neurons between saccades (Scudder et al. 2002)). In addition a visually evoked signal on the SC can activate the LLBNs (Rodgers et al. 2006). Conse-

quently, the idea of Kruijne et al. (2014) is that a distractor would activate the LLBNs and fatigue specifically the neurons coding for a saccade to the distractor. That fatigue would modify the trajectory of the next saccade: a distractor placed on the right of the target would fatigue the right LLBNs: the imbalance would cause a curvature to the left for the next saccade. As the SC connections to LLBNs are stronger for eccentric positions, the fatigue caused to the LLBNs would increase with distractor eccentricity, resulting in a stronger curvature (in line with Van der Stigchel et al., 2007). With the same logic, the model assumes that a long presentation of the distractor would also increase the fatigue of the LLBNs. Their theory is rather appealing in the way in which it explains the major phenomena that top-down inhibition control was given credit for.

In our experiment, however, such a fatigue mechanism driven by visual stimuli would predict either no curvature or a curvature *toward* the previous fixation point depending on the time scale of the fatigue. For instance, as stimulus S1 is foveal shortly before the second saccade, a short-term fatigue would affect equally all four LLBN populations, leading to no curvature. Alternatively,, in trials where S1 appears toward the right, for instance, a long-term fatigue from S1 could still affect the right LLBNs during the second saccade: the second saccade should curve toward the left, towards the previous fixation. In any case, these predictions are opposite to what we observed.

4.2 Prediction of Wang et al. (2012, 2014)'s model

The model of Wang et al. (2012; 2014) is based on hypothetical spatial interactions and winner-take-all selection occurring between stimuli on the Superior Colliculus (SC) map. These spatial interactions assumed that the SC is reducible to a Dynamic Neural Field with a Mexican hat kernel. The Mexican hat (MH) kernel defines three interaction zones centered around the stimulus input locus: a circular attraction zone, a ring repelling zone and a no-interaction zone

(Amari 1977). Because of these, the locus of a peak of activity on the SC map can deviate from the locus of its related stimulus input. Furthermore, it is the locus of one of these peaks that will determine the saccadic vector through a winner-take-all selection. With this simple attraction/repulsion mechanism between stimulus representations, Wang et al. (2012; 2014) successfully explained the relationship between initial deviations in saccade trajectory and distractor-target separation observed in the previous literature, notably based on McSorley et al. (2009)'s data and on a meta-analysis across 12 data sets. Furthermore, considering that a fixated stimulus also evoked a MH activation of the SC, they predicted and demonstrated experimentally that the timing of the fixation stimulus can affect the trajectory of saccades curving away from a distractor (Wang and Theeuwes 2014). This influence is explained by a Fixation-Target repelling effect interacting with a Target-Distractor repelling effect while the timing of the fixation stimulus varies the strength of the former effect.

This demonstration of their theory is elegant, however, to place the Mexican hat kernel and the fixation representation specifically in the SC without external updates prevents their model in its *current* state from explaining our results. With retinotopic inputs, both S1 and the Fixation stimulus would participate in shaping a MH profile centered on the rostral pole (i.e. fixation zone) of the SC (note that S1 is in the fixation zone after saccade 1). This MH profile would vary in strength according to Gap and S1 durations, and would result in different deviation of S2's representation from the rostral pole. This predicts slight changes (< 0.2° in Wang and Theeuwes 2014) in the amplitude of Saccade 2, but no changes in curvature.

4.3 Proposed model updates

We believe that our work does not disqualify the main mechanisms of the recent models, however, it calls to augment them with additional mechanisms.

The large dependence of saccadic curvature on the time since the previous saccade, is likely to partly originate from a saccade-related residual activity in the Superior Colliculus, as assumed by the work of other authors (Soetens et al. 1985; Anderson et al. 2008; Wang et al. 2011). The model of Kruijne et al. (2014) and Wang et al. (2012, 2014) did not consider motor residual activity from previous saccades because they were both developed to explain results from single-saccade paradigms. Concerning Kruijne et al. (2014), it might be difficult to reconcile the inhibitory effect of a fatigue mechanism with the excitatory effect of a motor residual activity. For instance, motor residual activity in the SC could cause fatigue in the LLBNs and lead to the reverse effect of what we observed— i.e. a deviation toward the initial Fixation stimulus. One solution would be to treat saccade-evoked activation of LLBNs differently from stimuli-evoked activation of the LLBNs. This could translate to the different types of neurons in the SC, respectively the motor-related and visual-related neurons. In a revised version of the model, the former would produce residual activity without fatigue in the LLBNs, whilst the latter would produce fatigue in the LLBNs by the time the critical saccade occurs.

In the model of Wang et al. (2012, 2014), the motor residual activity should not conflict with the current mechanisms. Neural field models—such as in Kruijne et al. and Wang et al. —generate automatically decaying residual activity after input offset because of the decay time constant (10-50 ms) they use. In fact, that kind of residual activity was used to explain several behavioral data sets on overt Inhibition of Return (IoR, Wang et al. 2011). Nevertheless, if motor residual activity is subject to Mexican Hat spatial interactions, there will be a similar problem as in the model of Kruijne et al. (2014). While the participant is fixating S1 and preparing to move to S2, the residual activity of Saccade 1 will push the activity related to S2 toward the initial Fixation point and lead to deviation *toward* the initial Fixation point. To avoid this, the addition of motor re-

sidual activity needs to be independent from spatial interactions, and may, for instance, take place in the LLBNs or another layer of the SC.

Our experiment also provides evidence for a curvature away from the spatiotopic representation of a previous fixation stimulus. A second revision of the models could then add either a satellite structure, which would send spatiotopic signals to the SC/LLBN, or a feedback mechanism, which would automatically shift the SC's signal when a saccade occurred (find more discussion in the next section). It is important to note here that the spatiotopic signal would project on the SC/LLBN with *excitatory* connections. That may at first seem contradictive with the top-down inhibition theory, but it is not. Indeed, in both the models of Wang et al. (2012, 2014) and Kruijne et al. (2014), the curvature away is explained by local suppression (i.e., lateral inhibition or neural fatigue) generated indirectly by an excitatory signal (i.e. a visual stimulus). In short, only an *excitatory* signal can activate the inhibitory mechanism that causes the curvature away in these models. To have fixation-related inputs from satellite bodies would echo evidence that there are several mechanisms of fixation-related inhibition, including cortical mechanisms (Sumner et al. 2006).

4.4 An Excitatory Spatiotopic Signal from the Lateral Intraparetial Area

One possible source for a top-down spatiotopic excitatory signal is the Posterior Parietal Cortex (PPC) that connects to the SC mainly through the Lateral Intraparietal area (Paré and Wurtz 1997). Using a double-step paradigm, Heide et al. (1995) have shown that patients with damage to the PPC are impaired in executing their second saccade when the second target is extinguished before the first saccade is initiated. In that situation, the second target has to be memorized and its retinal representation on the SC needs to be shifted in accordance with the first saccade vector (that is the spatiotopic update). Interestingly, patients with damage to the dorsolateral prefrontal cortex (DPFC) or to the

Frontal Eye Field (FEF) did not show such impairment (see also (Rivaud et al. 1994; Schiller and Chou 1998). Finally, predictive remapping of a target has been shown to occur in LIP (as well as the FEF), so that neurons respond to a target that will be in their receptive field after a saccade is completed (Goldberg and Bruce 1990; Goldberg et al. 1990; Duhamel et al. 1992; Umeno and Goldberg 1997; Kusunoki and Goldberg 2003). Neurophysiological work has demonstrated that such predictive activations also occur in specific cells of the SCi, i.e., the quasivisual cells (Mays and Sparks 1980; Walker et al. 1995). These findings support the possibility of a spatiotopic excitatory update of the SCi: notably the LIP/FEF would be projecting preferentially to the quasivisual neurons that, in turn, would reflect the activity of the LIP/FEF.

4.5 Conclusion

We conclude that both residual activity from previous saccades and spatiotopic representation of previously fixated stimuli can influence the trajectory of the current saccade. This influence is translated into a trajectory curvature away from the previously fixated stimulus. These findings call for current retinotopic models of curvature to update and take into account spatiotopic representations and the motor history. We suggest that the Lateral Intraparietal area would be a good candidate to provide excitatory spatiotopic signal to the SC.

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