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Discussion forum

Towards assessing extra-retinal uncertainty: A reply to M. Lisi (2020)

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1. Discussion forum

Saccades allow for sampling the environment for visual information, using the part of the retina with the highest receptor density, the fovea. The same saccades also cause frequent disruptions to visual processing, yet remarkably these disruptions mostly go unnoticed to human observers. It is generally thought that the visual system can account for these disruptions using extra-retinal signals, such as an efference copy from the oculomotor system or a proprioceptive signal containing information about eye position (Sommer & Wurtz, 2008). Several converging research lines have indicated that the posterior parietal cortex (PPC) plays an important role in accounting for these disruptions (Wurtz, 2008). One particularly fruitful line of research has focused on eye tracking studies in patients with lesions to the PPC (Duhamel, Goldberg, FitzGibbon, Sirigu, & Grafman, 1992; Fabius, Nijboer, Fracasso, & Stigchel, 2020; Heide, Blankenburg, Zimmermann, & Kömpf, 1995; Rath-Wilson & Guitton, 2015; Russell et al., 2010; Vuilleumier et al., 2007).

In our recent study (Fabius et al., 2020), patients with PPC lesions and healthy controls performed the two versions of the intra-saccadic displacement (ISD) task. In this task, extra-retinal information must be used to accurately discriminate the direction of displacement that a saccade target made during the saccade. From previous studies, it is known that the discrimination ability is surprisingly low, but can be improved by introducing a small temporal gap (with a blank screen) between the displacement and saccade offset (Deubel, Bridgeman, & Schneider, 1998; Deubel, Schneider, & Bridgeman, 1996). The main hypothesis for this improvement is that without the blank, the visual system assumes

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that the target had remained stable across the saccade, mostly disregarding extra-retinal signals. Instead, with the blank, that assumption of stability is broken and the available extra-retinal signals are used to perform the task (Mathôt & Theeuwes, 2011). The hypothesis in our study was that if the improvement in discrimination performance with a blank is related to the availability of extra-retinal signals, and the PPC is crucially involved in monitoring these extra-retinal signals, discrimination performance should not improve from the blank after a lesion to the PPC (in contrast to the healthy controls). Our results showed that although the improvement was smaller in patients than in controls, the improvement was still substantial. We concluded, therefore, that there are still some extra-signals available after a PPC lesion. Given that different forms of extra-retinal signals exist (i.e., efference copy and proprioceptive signals), we argued that the PPC monitors the most reliable one and that after a lesion, performance in the blank condition can only be improved (relative to the standard ISD condition) using the non-impaired extra-retinal signal, which does the job but with a lower fidelity.

Our conclusions are in line with data from another task that has been assessed in patients with PPC lesions to measure the availability of extra-retinal signals (Duhamel et al., 1992; Heide et al., 1995; Rath-Wilson & Guitton, 2015): double-step saccades (Hallett & Lightstone, 1976). In double step saccades, extra-retinal information must be used to guide the execution of a second saccade, because the target coordinates had been presented before the execution for a first saccade. If no extra-retinal, and only retinal coordinates were used, the second saccade would systematically be executed in the wrong direction. Although originally, it had been assumed that a lesion to the PPC abolished the use of extra-retinal signals because patients did not seem to execute any correct saccade to the second saccade target (Duhamel et al., 1992; Heide et al., 1995), these findings have been nuanced by demonstrating that these patients can reach the target but with multiple smaller saccades (Rath-Wilson & Guitton, 2015).

In a commentary on our study, Lisi (2020) provided an interesting alternative interpretation of our results and the results of the double-step saccade studies. He posits the hypothesis that these results can potentially be explained with a simple model that he and his colleagues developed (Lisi, Solomon, & Morgan, 2019). The model provides a probabilistic account of the systematic undershoot of prosaccades (typically ~10% of the required amplitude). Based on empirical data, the model assumes that there is inevitable variance in saccade landing points, most likely due to noise in sensory encoding of the saccade target (van Beers, 2007). Therefore, there will inevitably be some saccades that will require a second (corrective) saccade to reach the target. However, from previous observations it is known that not all corrective saccades are equal, in terms of latency. Corrective saccades are faster when they are in the same direction as the preceding saccade than when they are in the opposite direction (Ohl, Brandt, & Kliegl, 2011). This observation inspired the authors to model the 'cost' of saccade errors with an asymmetric cost function. In their model, the cost was operationalized as the latency of the corrective saccade. To reach a target with the lowest overall cost, the saccade amplitude gain should be adapted to the overall variability in saccade endpoint errors. If there is virtually no variability in endpoint errors, and all saccades to a target land approximately on the same location with each repetition, the gain should be close to 1. But if there is a lot of variability, meaning that there are many instances in which the eyes do not land on the target, it is beneficial to land at a location from which a corrective saccade can be executed in the same direction as the first saccade, i.e., a low saccade amplitude gain. Inverting this logic, Lisi and colleagues estimated the shape (or degree of asymmetry) of the cost function by examining the relationship between the variability in saccade endpoint errors and the average amplitude gain. To change the variability of saccade endpoints, they manipulated positional uncertainty of the saccade target with different methods (e.g., embedding the saccade target in varying levels of noise). Indeed, their paradigm successfully manipulated saccade amplitude variability: amplitude variability increased with increased positional uncertainty. In turn, with increased amplitude variability, amplitude gain decreased. This negative relationship was well explained by the asymmetrical cost function, the asymmetry of which could be estimated per observer. Moreover, the degree of asymmetry of the cost function correlated with the estimated "cost" (i.e., difference latency of backward *vs* forward corrective saccades) across participants.

From a theoretical perspective, the asymmetric cost function is an elegant model because it captures several aspects of saccadic behavior with only a single free parameter: the degree of asymmetry in the cost of forward versus backward corrective saccades. Applying the model to extra-retinal signals and PPC lesions, Lisi (2020) argued that the lesion in the patients of our study could have decreased the precision of extra-retinal information, irrespective of whether there are different types of extra-retinal signals involved. The hypothesis is that a reduced precision of extra-retinal signals will lead to a lower precision in the ISD task. Such a lower performance should be most pronounced when performance relies most the extra-retinal signals, i.e., in the blank condition. We agree that this is an intriguing and plausible hypothesis. We explored the implementation of the model for both the ISD task and double-step saccades. In doing so, we encountered several unknown relationships between oculomotor behavior and updating of visual information that could provide fruitful directions for future studies.

1.1. *Double-step saccades*

We will first discuss double-step saccade because they are conceptually closer to the paradigm employed by Lisi and colleagues (2019) than the ISD task. The hypothesis that should be tested using the model is whether extra-retinal information has become noisier after a lesion to the PPC. So, rather than applying the asymmetrical cost function to amplitude gain of the primary saccade, it should be applied to the *second* saccade of the double-step saccade paradigm. If the extra-retinal information is noisier after a PPC lesion, the second saccades should have more variable endpoint errors, and – according to the model – also a lower average gain. Although this sounds fairly straightforward, we are unsure whether it is clear what the reference is when we speak of

'more variable' or 'lower average gain' If this reference is the group of control participants, we are implicitly assuming that the shape of the cost function is unaffected by the lesion, or even more strict, the same for all participants, patients and controls alike. This assumption is likely unwarranted.

To illustrate this point, we analyzed saccades from a small unpublished dataset we had available in our archives. In this experiment, healthy participants made saccades to targets that either appeared on the left or right side, or on only one side of the fixation point, to manipulate positional uncertainty. In the former case, two motor plans need to be prepared before each trial, in contrast with the latter case, where a single motor plan is sufficient. We expected higher position uncertainty in blocks where the landing fixation point could appear unpredictably on the left or right side of fixation compared to blocks with a unique (predictable) landing fixation point on one side. All participants completed trials in both blocks. Looking at each condition separately, there is no negative relation between amplitude gain and amplitude variability *between* participants (Fig. 1, dashed lines). But, on average, there is a negative slope between amplitude gain and amplitude variability *within* participants between the different conditions (Fig. 1, solid line). In the terminology of the model, this implies that the exact shape of the cost function is different across participants, but on average it treats overshoots as more expensive than undershoots.

Assuming that the double-step data indeed shows a lower amplitude gain and higher second saccade endpoint variability in patients, this leaves us with two possible interpretations: either the extra-retinal information is noisier, or the cost-function is more asymmetrical. Dissociating

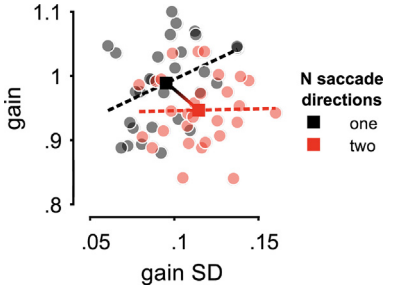

Fig. 1 – Illustration of the relationship between the variability in saccade endpoints (gain SD) and average saccade amplitude (gain) between and within participants. In this experiment, participants made saccades to a target that would always appear on the same side of the initial fixation point (one saccade direction; black), or it could appear on either side (two saccade direction; red). Increasing the number of saccade directions increases the positional uncertainty of the saccade target. The different number of saccade directions were presented in different blocks. According to the asymmetrical cost function model, amplitude gain should be negatively correlated with endpoint variability, no such negative correlation can be observed within each condition across participants (dashed lines). However, there is a negative relationship between amplitude gain and endpoint variability within participants (solid line).

between these two possibilities requires at least two different conditions with different endpoint variabilities *within* each participant. We are not aware of any existing studies that have assessed extra-retinal signals this way. Perhaps a manipulation similar to Lisi et al.'s (2019) could be used with different positional uncertainties, but instead of manipulating the uncertainty of the first saccade target, the uncertainty of the second target should be manipulated. It is probably wise to assess the feasibility of such an experiment in a sample of healthy controls first. If there is a manipulation that proves to robustly change the endpoint variability within participants, it would be very interesting to test patients with PPC lesions with the same paradigm. The question would then be: is the decrease in amplitude gain with an increase endpoint variability larger after a lesion to the PPC than in control subjects? In this way, the group comparison is a comparison of the size of the within-subject differences, similar to our analysis of the difference between performance in the standard and blank conditions of the ISD task (Fabius et al., 2020). In sum, we agree that the asymmetrical cost function can yield specific predictions for performance in the double-step saccade task, although it should be verified that variability of the second saccade endpoints can be manipulated within the same participants.

1.2. *Intra-saccadic displacement task (ISD)*

Next, we will explore how the asymmetric cost function could potentially be applied to data of our ISD task (Fabius et al., 2020). There are several 'translation' issues that need to be resolved to go from a saccade task to the ISD task. First, because the ISD task is a perceptual task, the 'cost' is different from the cost in the saccade task. In the context of ISD, the cost could be the number of incorrect responses. Or, more generally and probabilistically, the cost could be defined as the probability of erroneously ascribing retinal displacements due to a saccade to an external sensory event, resulting in 'hallucinatory' jumps – side note, this interpretation of the cost of extra-retinal processing has also been used in the interpretation of psychosis (Bansal, Bray, Schwartz, & Joiner, 2018; Rösler et al., 2015). Second, to apply the asymmetric cost function to our data we should translate 'amplitude gain' and 'endpoint variability' to the displacement task. Gain variability could be translated to be the inverse of the slope of the psychometric function (this is also done by Lisi et al. in their supplementary 'perceptual' experiment) and the perceptual null location can serve as a measure of gain. Is there a systematic relationship between the perceptual null location and the slope, like the relationship between amplitude gain and endpoint variability? How are they related to different levels of reliability of the extra-retinal signals? If the reliability of extra-retinal signals can be deduced from the oculomotor performance in the double-step saccade task, it would also be interesting to examine the relationship between performance on the ISD task and concurrent oculomotor performance.

Currently, many of the relationships between ISD performance and oculomotor parameters are unknown, but there are several findings that started to fill in the gaps. The existing literature suggests a systematic 'backward' bias of the

perceptual null location, that scales with saccade amplitude and a negative correlation between saccade amplitude and the slope of the psychometric function, i.e., flatter curves for larger saccade amplitudes (Bansal, Jayet Bray, Peterson, & Joiner, 2015; Bridgeman, Hendry, & Stark, 1975; Jayet Bray, Bansal, & Joiner, 2015; Joosten & Collins, 2018; Niemeier, Crawford, & Tweed, 2003). Although a 'forward' bias has also been reported (Deubel et al., 1996). Furthermore, variability in saccade endpoints seem to correlate with the slope in the standard displacement task, but not with the slopes of the displacement task with a blank (Niemeier et al., 2003; Wexler & Collins, 2014). Possibly, the slope and variability only correlate in the standard ISD task and not in the blank condition, because the saccade endpoint variability reflects the precision of sensory encoding (van Beers, 2007), whereas performance in the blank condition better reflects the precision of the extra-retinal information. The perceptual null location of neither task correlates with the saccade endpoint variability (Bansal et al., 2015; Collins, Rolfs, Deubel, & Cavanagh, 2009). A more formal description of the relationship between oculomotor performance and ISD performance is given by the model of Niemeier et al. (2003). In their model, retinal and extra-retinal information are integrated in a Bayesian framework. Their model predicted both ISD and oculomotor performance, and the relationship between the two for the standard ISD task, but not for the blank condition. Moreover, they accounted for between-participant difference by examining both displacements parallel and orthogonal to the saccade direction. As has been observed in different studies, orthogonal displacement to the saccade direction are more readily detected than displacements parallel (Jayet Bray et al., 2015). The ratio between the sensitivity for orthogonal and parallel displacements correlated with the ratio between the major and minor axis of saccade endpoint variability (Niemeier et al., 2003).

2. Conclusion

Together, the known relationships between ISD performance and oculomotor performance provide good starting points for the implementation of a probabilistic account of PPC lesions on ISD performance. However, as mentioned before, not all relationships are known yet. Because there are too many unknowns, we cannot, at this point, make explicit predictions what an increase in variability of extra-retinal signals would result in, with respect to ISD performance. Nonetheless, we believe that Lisi's hypothesis is intriguing and plausible, and we think explicit models of the ISD task in combination of models of oculomotor behavior would improve our understanding of trans-saccadic perception and could yield concrete hypotheses that can be tested directly in both population of healthy controls and patients with PPC lesions.

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