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Saccadic inhibition can cause the remote distractor effect, but the remote distractor effect may not be a useful concept

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Abstract

We have suggested that the Remote Distractor Effect (RDE), the elevation of average saccadic reaction time (SRT) induced by a task-irrelevant distractor, is a statistical consequence of a characteristic reshaping of the SRT distribution known as Saccadic Inhibition (SI) (Buoncore & McIntosh, 2008). In a recent paper, Walker and Benson (2013) argue against this idea, and claim that the RDE and SI are partly dissociable. Here, we examine this claim, taking the opportunity to clarify potential ambiguities about how SI affects average SRT, and how the presence of SI can be inferred from SRT distributions. We highlight what we consider to be the most interesting aspects of Walker and Benson's data, and suggest that a more flexible and nuanced view of SI can account for them. In considering the relation between SI and the RDE, we conclude that the RDE may no longer be a useful concept for eye movement researchers.

Article

The effect of task-irrelevant distractors on the timing of saccades can be visualised and measured in different ways. A simple one is to compare a measure of central tendency of saccadic reaction time (SRT) between trials with and without a distractor. The elevation of average SRT that can often be observed by doing so is known as the Remote Distractor Effect (RDE) (Walker, Kentridge & Findlay, 1995; Walker, Deubel, Schneider & Findlay, 1997). More detailed analyses have shown that irrelevant visual onsets provoke a complex but consistent reshaping of the SRT distribution, such that saccades are less likely to be launched around 60-125 ms following the change, with a subsequent 'rebound' phase of elevated saccade frequency, reflecting delayed launching of the saccades inhibited. The rebound phase can extend for a further ~100 ms. This characteristic distributional change is known as Saccadic Inhibition (SI) (Reingold & Stampe, 1999; 2000; 2002). We have suggested, on empirical grounds, that SI underlies the RDE (Buonocore & McIntosh, 2008); in other words, the so-called RDE may simply reflect the overall impact of SI on average SRT.

In a recent paper in the *Journal of Vision*, Walker and Benson (2013) set out to test the idea that the RDE and SI reflect the same inhibitory phenomenon. They studied the effect of distractors, presented at different locations and times relative to a predictable target, on average SRT (to reveal the RDE) and on the shape of the SRT distribution (to reveal SI), reasoning that, if the RDE and SI

are the same, they should show similar patterns of modulation across conditions. Unfortunately, this relies on the mistaken assumption of a simple translation of SI into average SRT. In truth, the complexity of the SI signature means that its effects on average SRT are far from transparent, even if optimal methods are used. For instance, we have shown that a clear SI dip is not always accompanied by a significant RDE (Buonocore & McIntosh, 2008; 2012). This can be partly a matter of power: if a weak SI affects only a few saccades, the overall impact on SRT may be too small to detect within a typical experiment. However, it is also related to the insensitivity of average SRT measures to the distributional changes wrought by SI.

Figure 1 illustrates this, using simulated SRT distributions for saccades to predictable lateralised targets, with SI induced by distractors presented at various times following target onset. The baseline SRT distribution is a normal distribution with a plausible mean and standard deviation. For each distractor condition, we modified the baseline distribution using a simple mathematical simulation of SI, for which we specified fixed values or distributions for: (1) the neural delay for visual information to reach the inhibitory centres, which defines the lower temporal limit for the distractor to influence saccade launching; (2) the duration of the inhibition, which defines the upper temporal limit for the distractor to cause a saccade to be inhibited; (3) the proportion of saccades that will be inhibited during the window between the lower and upper temporal limit; (4) the delay that inhibited saccades undergo before launching. See the footnote for fuller details.

Figure 1A shows that the raw SRT distribution differs quite dramatically for different distractor timings, because the SI signature affects different portions of the baseline distribution (cf. Buonocore & McIntosh, 2008). In order to 'recover' the SI profile, we must undo this interaction, subtracting the distribution for the baseline condition from that for each distractor condition (Figure 1B), and scaling the difference by the baseline (Figure 1C) (Bompas & Sumner, 2011). Figure 1C shows that the SI profile thereby recovered is constant across distractor conditions (because we explicitly simulated it as being constant), but is shifted in time according to the distractor delay. Figure 1D shows the impact of this constant but shifted profile on average SRT, using the median as the measure of central tendency. First, the impact of each distractor upon median SRT follows a typical pattern described for the RDE, with measured distraction being large for distractors within 50 ms after the target and falling off steeply for distractor delays above 80 ms (Buonocore & McIntosh, 2008; Walker et al., 1995). The basic pattern is replicated in the new data reported by

Walker and Benson (Experiments 2 and 3), who likewise used the median as their measure of central tendency. Figure 1A helps to illustrate why such extreme variation in the RDE can arise from a constant SI profile with different distractor timings. Median SRT will be elevated mainly when the distractor impacts upon the left side of the SRT distribution, but will not be altered at all if the SI effect is confined within the right side of the distribution. The mean might provide a more stable global measure of distraction; but no measure of central tendency will appropriately describe the bimodal SRT distributions that distractors can sometimes induce, which is why a full distributional analysis should be preferred (Buonocore & McIntosh, 2008; 2012: 2013).

Therefore, we certainly agree that the RDE and SI are partially dissociable, because the RDE can vary independently of SI magnitude, and it is even possible to observe SI without any RDE at all. However, this dissociation, though important to appreciate, is not interesting at a theoretical level, because it just reflects the inadequacy of median SRT as an index of the distributional changes wrought by SI. The more interesting direction of dissociation claimed by Walker and Benson is the opposite one: that the RDE can arise without SI. This conclusion is entirely plausible, but we think that it is premature, and that Walker and Benson provide no compelling evidence for it. There are a few limitations of their methods which should be noted, before moving on to some more inescapable limitations of SI analysis itself.

Walker and Benson first seek evidence of SI by looking for a 'notched dip' in the SRT distributions for distractor conditions. But, as already discussed, because of the complex interaction of SI with the baseline SRT distribution, one should not expect a distractor to induce bimodality (a 'notched dip'), unless inhibition impacts around the centre of the SRT distribution. Given different distractor timings, the distribution may be quite differently affected. For instance, an earlier distractor may tend to push the distribution rightward, and skew it positively. In general, the absence of a 'notched dip' in the SRT distribution does not exclude the presence of SI.

Walker and Benson next subtract the baseline distribution and look for the characteristic dip, 60-125 ms after distractor onset, in the difference histogram. However, although simple subtraction of the baseline can help show SI (Buonocore & McIntosh, 2008), the optimal method is to scale the difference by the baseline frequency at each point in time, thus normalising the differences (Bompas & Sumner, 2011). Even using this proportional method, the estimate of the dip can be unstable if it impacts upon a part of the baseline distribution with a low frequency of saccades. Studies of SI

during reactive saccadic tasks have often taken account of the baseline distribution by adjusting distractor timing per-participant so that the point of maximum SI impacts around the peak of the baseline distribution, to give a robust dip. This is a powerful strategy when studying the influence of non-temporal aspects of the distractor, such as contrast (Bompas & Sumner, 2011), size or location (Buonocore & McIntosh, 2011; Edelman & Xu, 2009); the only situation in which it is not viable is if the timing of the distractor is itself the independent variable of interest (e.g. Buonocore & McIntosh, 2008). Walker and Benson did not adapt distractor timings to their participants, but used fixed distractor delays even when studying the influence of distractor location alone (Experiment 1). The use of fixed delays and unscaled difference histograms could compromise the stability of Walker and Benson's measures of SI, so we should not expect their estimates of dip timing to be precise.

Even so, it is reassuring that Walker and Benson did report distributional changes at least broadly consistent with SI in all of the conditions in which we would expect to see it (+30, +60 and +90 ms distractor delays). The conditions in which SI was not so obvious, and on which Walker and Benson place emphasis, were those in which the distractor was onset simultaneously with the target, or preceded it. Here, rather than showing a 'notched dip', the SRT distributions for the distractor conditions showed a rightward shift with an increase in positive skew. It is *conceivable* that this pattern represents a distinct mechanism of distractor effect, but we think that it is more parsimonious to explain it within an SI framework.

We begin by considering an inescapable limitation of the standard SI analysis itself. Specifically, one can only record a 'dip' in saccadic frequency within a normally active portion of the SRT distribution; inhibition occurring earlier than the earliest expected saccades cannot be observed directly. For instance, inhibition caused by a simultaneous distractor would begin around ~60 ms, coinciding with the very earliest possible saccades. Suppression of early saccades would delay the onset of saccadic behaviour, manifesting as a rightward shift of the distribution. The distractor distribution would continue below baseline until the end of the dip (~125 ms), with a subsequent 'rebound' phase of (up to) a further ~100 ms, during which saccade frequency would exceed the baseline as the inhibited saccades undergo delayed launching. This increase in the population of longer-latency saccades would cause positive skew, precisely as Walker and Benson observed with simultaneous distractors (see their Figure 3). Walker and Benson are unclear on this

point, however, because they "*regard SI to be a short-lasting effect, starting at about 60 ms after a visual onset and lasting for around 60 ms*" (p. 18), and they do not think that such a transitory effect could account for a sustained increase of longer-latency responses. This apparently confuses the transitory 'dip' for the complete SI profile, which includes the sustained recovery phase. The effects of simultaneous distractors that Walker and Benson report are in fact fully consistent with the predictions of SI.

Walker and Benson's more unexpected, and most intriguing result derives from the condition in which the distractor preceded the target by 60 ms. This advance distractor, when presented contralateral to the target, induced changes quite similar to those caused by a simultaneous distractor, with a robust RDE associated with a rightward shift of the early distribution and a slight increase in long-latency saccades. Walker and Benson again argue that these distributional changes extend too far in time to be caused by SI. At face-value, there should indeed be little scope for target-elicited responses to be affected by the SI dip, which is generally understood to be from around 60-125 ms after a distractor. For a distractor presented 60 ms before the target, the inhibitory influence should be ending around the time of the arrival of the target signal at the ocuolomotor centres of the intermediate Superior Colliculus, so interference should be minimal. One might simply conclude, as Walker and Benson have done, that SI cannot be the sole mechanism of distraction; but we believe that it is possible to accommodate the effects of advance distractors within an SI framework, if some unstated assumptions are recognised and relaxed.

It must be remembered that the inhibitory mechanism(s) giving rise to SI are still debated, and that SI is simply a descriptive label for a characteristic behavioural effect. That effect is typically elicited and visualised using certain methods, which colour our view of the phenomenon itself. First, given that the SI dip is usually visualised as a failure to launch expected saccades in a specific time window, it is easy to assume that SI acts only by cancelling the execution of mature saccade programs (i.e. saccades that have been prepared, and are poised for launching). We ourselves held this view when first studying SI (Bunocore & McIntosh, 2008). However, the power of advance distractors to reshape SRT distributions suggests to us that SI can cause comparable delays at earlier stages of the saccade generation process. Late distractors tend to interfere with saccade launching, causing the familiar dip profile, but earlier distractors may interfere with prior stages of target selection, causing rightward shift and positive skew.

Second, the relative consistency of the classic SI profile may encourage the assumption that the parameters of SI are known and fixed, where in fact they are subject to some uncertainty and may be malleable. A key area of uncertainty is over the duration of inhibitory activity. The SI dip typically extends from 60-125 ms after the distractor, so it is tempting to infer that inhibitory activity lasts for ~65 ms only. However, this inference is unlikely to be valid, because the SI profile is shaped not only by the inhibition of saccades, but also by the superimposed recovery of inhibited saccades. It is not yet known what delay is imposed on individual saccades, but the default assumption would be that it follows a Gaussian distribution (in Figure 1, we modelled SI using a delay distribution with a mean of 62 and a SD of 25 ms). If the lower range of the delay distribution is any less than the true duration of inhibitory activity, then the dip will be artificially truncated at its right-hand edge by a rising sub-population of recovered saccades, and the duration of the dip will underestimate the duration of inhibitory activity. The true duration of inhibitory activity is thus probably greater than 65 ms, so the true inhibitory reach of a distractor will extend for more than 125 ms after its onset, giving ample scope for advance distractors to affect the SRT distribution.

A further consideration is that the parameters of SI are not rigidly fixed, but depend on task and stimulus factors (e.g. Bompas & Sumner, 2011; Buonocore & McIntosh, 2011; 2012; Edelman & Xu, 2009). We will again focus on the duration of inhibitory activity, as estimated from the width of the SI dip. In the standard SI paradigm, the dip is elicited by a distractor in the presence of a visible target. The bottom-up signal from this visible target is likely to aid the suppression of distractor-related activity; consistent with this, Edelman and Xu (2009) found that the SI dip was longer-lasting when the distractor competed with a remembered target rather than a visible one. Advance distractors are an interesting further case, because they laterally inhibit activation at an expected target location (Olivier, Dorris & Munoz, 1999) but, initially at least, will do so in the absence of a visible target. Advance distractors may thus have a more persistent inhibitory action than concurrent or delayed distractors, giving more scope for them to exert a sustained influence on subsequent SRTs, as Walker and Benson observed. We do not entirely rule out other mechanisms of inhibition. It is possible, for instance, that resisting the impulse to saccade to an advance distractor requires the participant to increase activation of the fixate system, which could dampen subsequent responses to the target. However, there is no strong reason to invoke other mechanisms, because it may be quite possible to account for all of the changes observed in terms of SI. In sum, across

Walker and Benson's three experiments, only one condition (the -60 ms contralateral distractor condition of Experiment 2) presents any challenge to an SI account, and this may be met by a more flexible and realistic view of the character of SI.

SI can certainly induce an elevation of average SRT, classically called the RDE, but it remains to be seen whether it is the sole cause. If it is, then the RDE simply reduces to the more informative concept of SI; if not, then the RDE is a non-specific umbrella term covering multiple inhibitory effects. Moreover, its original description, as a latency elevation caused by distractors that are close in time to but spatially remote from the target (Walker et al, 1995, 1997), is belied by recent evidence. Walker and Benson (Experiment 2) themselves obtain the RDE with very early (-60 ms) and very late (+60 ms) distractors, as well as with ipsilateral distractors close to the target. Others have confirmed that ipsilateral distractors can inhibit saccades (Edelman & Xu, 2009; McSorley, McCloy & Lyne, 2012; Walker et al., 1997), and we have recently shown that these inhibitory effects can be even stronger than those of remote distractors (Buoncore & McIntosh, 2012). Given this non-specific character, and considering that average SRT is an unstable and relatively uninformative global measure of distraction, the RDE may not be a useful concept for eye movement researchers. The temporal effects of distractors on saccadic behaviour are better studied via distributional analyses capable of revealing SI and, importantly, of uncovering changes that would be incompatible with SI (such as a monotonic shift of the whole distribution).

Finally, it must be emphasised that SI is only a descriptive term for a characteristic behavioural effect; it does not yet imply specific underlying mechanisms, let alone a unitary one. Different mechanisms might potentially account for different aspects of SI and its modulation by task. Beyond this complexity is a much wider complexity, because temporal inhibition of saccades is just one aspect of a diverse, intertwined set of distractor effects, which can include facilitation as well as inhibition (Corneil & Munoz, 1996; Dorris, Olivier & Munoz, 2007; Trappenberg, Dorris, Munoz & Klein, 2001), and spatial as well as temporal consequences for saccade execution (Edelman & Xu, 2009; Guillaume, 2012; McSorley et al., 2012; Walker et al., 1997). These diverse effects may ultimately reduce to a core set of principles governing target selection in the saccadic

system. The elucidation of these core principles will require the confluence of human behavioural work, neurophysiological investigations, and computational modelling.

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Figure legend

Figure 1. (A) Mean of 100 simulated percentage frequency histogram of SRTs for baseline (target only) condition (grey shaded area) and distractor conditions (coloured lines). Each colour corresponds to a different/target distractor SOA, from simultaneous onset to 150 ms delay in steps of 25 ms. (B) Mean of 100 simulated difference histograms (*absolute SI profile*) for each distractor condition representing the bin-by-bin subtraction of baseline histogram from frequency distractor histogram. Data are aligned to target onset and displayed up to 300 ms after distractor onset. (C) Mean of 100 simulated proportional difference histograms (*proportional SI profile*) for each target/distractor SOA representing the subtraction histogram divided the baseline histogram. As in Panel B, data are aligned to target onset and displayed up to 300 ms after distractor onset. (D) Means of the median SRTs for each simulated distractor SOA (solid line) and target only baseline (dotted line). For comparison, empirical data are plotted from Buonocore and McIntosh (2008).

Footnote: simulation method

All distributions were generated in Matlab. For both no-distractor and distractor conditions, 10000 SRTs were drawn randomly from a distribution in the Pearson system with mean equal to 183 ms, standard deviation equal to 52.83 ms, skewness equal to 0.78, and kurtosis equal to 3.89. These parameters were chosen to simulate the baseline distribution obtained empirically by Buonocore and McIntosh (2008). Distributions (10000 values) for "neural delay", "SI duration", and "saccadic delay" were generated by drawing values randomly from Gaussian distributions with means of 70, 70 and 62 ms and standard deviations of 10, 25 and 25 ms respectively. In the distractor condition, the simulated SRTs were modulated following a simple set of rules. For each simulated saccade, an inhibitory window was defined with a "lower limit" (the current SOA plus a value extracted from the "neural delay" distribution) and an "upper limit" (the current "lower limit" plus a value extracted from the "SI duration" distribution). Each SRT falling within the inhibitory window had an associated value ranging from 0 to 1 taken from a discrete uniform distribution. If this value was smaller than the magnitude of inhibition (i.e. the probability to be inhibited, see below), a value extracted from the "saccadic delay" distribution was added to the current SRT. Otherwise the current SRT remained unchanged. In the simultaneous condition the upper limit was fixed at 155 ms after target onset. The probability of inhibiting a saccade was fixed at 0.28 (28% of the maximum inhibition). From the resulting set of simulated SRTs, percentage frequency histograms were created (bin width 4 ms) and lightly smoothed using a Gaussian kernel with 16 ms window and 2 ms SD and then interpolated to obtain 1 ms precision. The absolute change for each point in time was computed by subtracting the baseline condition from the distractor condition (cf. Buonocore & McIntosh 2008). The proportional change for each point in time in the distractor distribution relative to the no-distractor distribution was computed from the formula: (no-distractor - distractor)/no-distractor (cf. Bompas & Sumner 2011). To simulate the effect of SOA, the SOA was incremented by 25 ms each loop, starting from a value of zero, for a total of seven iterations. Each simulation was run 100 times. At the end of the simulation the output of each condition was averaged across runs.

Figure 1.