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The Attentional Blink is Related to the Microsaccade Rate Signature

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Abstract

The reduced detectability of a target T2 following discrimination of a preceding target T1 in the attentional blink (AB) paradigm is classically interpreted as a consequence of reduced attention to T2 due to attentional allocation to T1. Here, we investigated whether AB was related to changes in microsaccade rate (MSR). We found a pronounced MSR signature following T1 onset, characterized by MSR suppression from 200 to 328 ms and enhancement from 380 to 568 ms. Across participants, the magnitude of the MSR suppression correlated with the AB effect such that low T2 detectability corresponded to *reduced* MSR. However, in the same task, T1 error trials coincided with the *presence* of microsaccades. We discuss this apparent paradox in terms of known neurophysiological correlates of MS whereby cortical excitability is suppressed both during the microsaccade and MSR suppression, in accordance to poor T1 performance with microsaccade *occurrence* and poor T2 performance with microsaccade *absence*. Our data suggest a novel low-level mechanism contributing to AB characterized by reduced MSR, thought to cause suppressed visual cortex excitability. This opens the question of whether attention mediates T2 performance suppression independently from MSR, and if not, how attention interacts with MSR to produce the T2 performance suppression.

Key words: human, individual differences, psychophysics, rapid serial visual presentation, saccadic suppression

Introduction

In the attentional blink (AB) paradigm, participants are required to respond to two items embedded in a rapid serial visual presentation (RSVP) sequence (Raymond et al. 1992). If the second target stimulus is presented within the specific time window of around 150 to 250 ms after the first target stimulus, detection and discrimination of the second target is impaired. While a host of theories have been presented to account for this effect (for review see Dux and Marois 2009, 2010), consistent themes have been that correctly identifying the targets requires defined processing resources for encoding, working memory, response selection, and above all attention, which are required to enhance the target representation and suppress distracter representations. In the AB paradigm, processing of the first target (T1) is thought to render these resources temporarily unavailable, thereby impairing the identification of the second target (T2) for a brief period. As microsaccades (MSs) have been proposed to contribute to both attention and perceptual performance (Hafed and Clark 2002; Engbert and Kliegl 2003; Galfano et al. 2004; Rolfs et al. 2005; Rolfs 2009; Laubrock et al. 2010; Pastukhov and Braun 2010; Pastukhov et al. 2013; Yuval-Greenberg et al. 2014; Hafed et al. 2015; Meyberg et al. 2015; Tian et al. 2016; Lowet, Gips et al. 2018), we asked what role they would play in the AB effect.

MSs are small involuntary eye movements that take place during attempted fixation of eye position, including during natural vision (for review see Martinez-Conde et al. 2013). Although during stable viewing conditions MSs typically occur at a relatively stable rate (Engbert and Kliegl 2003; Otero-Millan et al. 2008; Bosman et al. 2009; Otero-Millan et al. 2013), microsaccade rate (MSR) can be influenced by visual input (Engbert and Kliegl 2003; Otero-Millan et al. 2008; Cui et al. 2009; Otero-Millan et al. 2013; Mccamy et al. 2014) and by cognitive factors (Gowen et al. 2005; Betta and Turatto 2006; Di Stasi et al. 2013; Fried et al. 2014; Siegenthaler et al. 2014; Gao et al. 2015) by which, notably, sustained attention has been associated with a reduction of MSR (Gao et al. 2015; Pastukhov and Braun 2010; Siegenthaler et al. 2014; Xue et al. 2017; for review see Rolfs 2009). Such influences can lead to rapid changes in MSR: The onset of a visual stimulus leads to a rapid reduction of MSs lasting between 100 and 500 ms, followed by a rebound and overshoot occurring 200–600 ms after stimulus onset (Betta and Turatto 2006; Rolfs et al. 2005, 2008; Hafed and Ignashchenkova 2013; Fried et al. 2014; Scholes et al. 2015; Xue et al. 2017). The same "microsaccade rate signature" has been observed in response to oddballs in a stream of visual stimuli (Valsecchi et al. 2007; Valsecchi and Turatto 2009; Pastukhov et al. 2013), by the onset of endogenous or exogenous attentional cues (Laubrock et al. 2005; Rolfs et al. 2005; Gowen et al. 2007; Xue et al. 2017) and at the onset of working memory retention cues (Dalmaso et al. 2017).

Fixational eye movements, including drifts and MSs support active vision (Rucci and Desbordes 2003; Rucci et al. 2007; Ko et al. 2010; Chen and Hafed 2013; Kagan and Hafed 2013; Poletti et al. 2013) and are crucial for maintaining perception during attempted fixation, as a perfectly stable image on the retina quickly fades (Costela et al. 2013, 2017; Martinez-Conde et al. 2006; McCamy et al. 2012, 2013, 2014; Riggs et al. 1953; for reviews see Rolfs 2009; Martinez-Conde et al. 2004, but see also Collewijn and Kowler 2008; Poletti and Rucci 2010 and subsequent rebuttal McCamy et al. 2012). However, perception is also compromised around the moment of the eye movement (Zuber and Stark 1966; Hass and Horwitz 2011; Xue et al. 2017; Scholes et al. 2018) and it may be adaptive to suppress MSs in demanding visual tasks (Bridgeman and Palca 1980; Pastukhov and Braun 2010; Xue et al. 2017). The double role of MSs, necessary to prevent fading yet deleterious at the moment of the movement, implies that vision will be impacted whenever MSR is too high or too low.

Following the idea that during MS execution visual processing is suppressed and that MS are generally suppressed during sustained attention, we hypothesized that in the AB paradigm, MSs would be suppressed prior to and during T1 presentation. Such a strategy would maximize T1 performance by avoiding MS occurrence at the moment of T1 presentation. We further anticipated a MSR signature following T1 presentation. If the time course of the MSR signature were relatively slow (e.g., Dalmaso et al. 2017; Pastukhov et al. 2013; Rolfs et al. 2008; Xue et al. 2017 for attentional cue onset), the T2 performance deficit could coincide with MSR suppression. However, if the time course were relatively fast (e.g., Hafed and Ignashchenkova 2013; Valsecchi et al. 2007; Valsecchi and Turatto 2009; Xue et al. 2017 for stimulus onset), the T2 performance deficit might coincide with MSR rebound. In the latter case, T2 presentation might have a high chance of remaining unnoticed due to co-occurring MSs. At the outset of our work, we had hypothesized that the presence of MSs would underlie both T1 and T2 errors.

We found that MSR was indeed modulated in the AB paradigm. In line with our original hypothesis, we found that T1 performance was poor during *higher* MSR. However, contrary to our hypothesis, we found that the period of poor T2 detection was associated with a marked *reduction* of MSR. Moreover, the amount of suppression was positively linked to the magnitude of the AB effect, whereby participants who showed the greatest behavioral deficit also showed the largest reduction in MSR. These data suggest a paradox that performance was hindered by the presence of MSs in the T1 task but by the absence of MSs in the T2 task.

Eye movements, including MSs, have been found to be accompanied by a rapid modulation in neuronal activity. While modulation is typically small compared with modulation in response to a stimulus onset, and there is diversity in the reported form of neuronal modulation (see Martinez-Conde et al. 2013 for review), we (Lowet et al. 2016) and others have observed a rapid suppression of neuronal activity at around the time of the movement (Leopold and Logothetis 1998; Hass and Horwitz 2011; Meirovithz et al. 2012; McFarland et al. 2015) followed by a brief period of enhanced activity (Leopold and Logothetis 1998; Kagan et al. 2008; Bosman et al. 2009; Hass and Horwitz 2011; Meirovithz et al. 2012; McFarland et al. 2015; Troncoso et al. 2015). In line with the T1-triggered MSR signature, we here argue that neuronal suppression around the eye movement may explain the current behavioral data around T1, while the brevity of neuronal enhancement after the movement, turning to neuronal suppression during temporary MSR reduction, may explain behavioral data around T2.

We have previously demonstrated how MSs are linked with rapid suppression followed by brief enhancement of spike rates, gamma power, and gamma frequency in macaque V1 during passive fixation (Lowet et al. 2016; Gips et al. 2017). We have also replicated these effects in computational modeling (Lowet, Gips et al. 2018), in which PING (Pyramidal-InterNeuron Gamma) network input was modulated by a double-exponential kernel adjusted to match recordings in LGN (Reppas et al. 2002; Martinez-Conde et al. 2009). To illustrate our interpretation of the current data, we selected MSs from the monkey data that were followed by prolonged periods of stable eye position, matching the duration of MSR suppression observed in the attentional blink experiment. Gamma power and spike rates were as suppressed during prolonged MSR suppression as during the MS itself, suggesting a mechanism by which perception can be weakened by both the occurrence and the absence of MSs.

The electrophysiological correlates of MS demonstrates the "built in paradox" of the visual system (Martinez-Conde et al. 2006), in which cortical excitability and perception are reduced by both MS execution and suppression, because of a similar reduction in cortical excitability. Our data provide the first evidence that T1 and T2 performance in the AB paradigm strongly depend on MS rates and may indicate that worse T1 performance during MS presence and worse T2 performance during MS absence both may be explained by a similar reduction of cortical excitability. Our results suggest a previously unknown low-level contribution, whereby reduced cortical excitability due to reduced MSR contributes to reduced T2 detection. As the MSR reduction is part of the T1-triggered MSR signature, which is at least in part an automatic response, our data indicate that the contribution of attention to the AB, and the mechanism by which this occurs, needs to be re-evaluated.

Materials and Methods

Participants

We recruited 32 participants (27 female; mean age 21.9, SD 2.8). All had normal or corrected-to-normal visual acuity and were naive to the purpose of the experiment. After giving full information about all procedures and the right to withdraw participation at any time, informed written and verbal consent was obtained according to the Helsinki Declaration. All procedures were approved by the local Ethical Committee of the Faculty of Psychology and Neuroscience. For their participation in the study participants received either monetary reward or credits to fulfill course requirements.

Stimuli, Apparatus, and Task

Data collection took place in a dimly lit room while participants were sitting in a chair with their head supported by a chin and head rest to keep eye-screen distance constant at 57 cm. Visual stimuli were displayed on a 19-in Samsung SyncMaster 940BF LCD monitor (60 Hz refresh rate, 1280 × 1024 resolution). Stimulus presentation and response recording was performed by Cortex v.5.9.6 (NIH freeware for psychophysical and neurophysiological experimentation).

Participants were shown an RSVP stream of dark gray capital letters (luminance: 11.73 cd/m^2) on a light gray background (luminance: 39.59 cd/m^2). Their task was to identify the only red letter in the stream which could be either a "T' or an "L" (referred to as T1), and to indicate whether a black "X" (referred to as T2) was presented anywhere after T1. T2 presentation, when it occurred, was randomized between the first and seventh letter presentation after T1. The RSVP letter stream consisted of 27 letters. In the results section, individual letters will be referred to by their position in the stream (1–27). The gray distracter letters were randomly chosen from the alphabet excluding the letters T, L, X, and Q. Immediate repetition of a letter was also excluded. T1 was presented at random between positions 10 and 14 inclusive, and consequently T2 could appear anywhere between positions 11 and 21. Each letter was presented for 33 ms with an inter-stimulus interval of 100 ms. Letters were presented in the center of the screen and were 0.3° large (Fig. 1A). There were nine different experimental conditions: T2 could either be presented 133, 266, 400, 533, 666, 800, or 933 ms after T1 (referred to as Lags 1–7), only T2 could be omitted, or both T1 and T2 could be omitted. Each condition occurred with the same likelihood, so that 22% of all trials were without T2. Trials began with a 305 ms fixation period resulting in a total duration of each trial of 3896 ms.

Each condition was presented 40 times, subdivided into 4 blocks. After each trial the response options for T1 ("T,", "L," and "no letter") and T2 ("no X" and "X") were displayed on the screen and participants could indicate their response using a standard computer keyboard with the left, right, and down arrow key that corresponded spatially to the display of possible answers (see Fig. 1B). Feedback was provided by highlighting the correctly given answer in green and removing incorrect options or, in case of an incorrect answer, highlighting the given response in red and showing the correct answer in black.

The experiment consisted of four blocks each with 10 trials per condition, thus 90 trials per block and 360 trials in total. Blocks were separated by a short break. Participants were required to maintain fixation within a 3° by 3° window. If the gaze shifted beyond that window at any time during the trial, the trial was immediately aborted, and a new trial would start.

Aborted trials were repeated later in the block. Before starting the experiment, participants completed 18 training trials (two per condition) to familiarize themselves to the task.

Eye movements were recorded monocularly using the subject's dominant eye with a desktop-mounted Eyelink 1000 eye-tracker (SR Research Ltd., 500 Hz or 1000 Hz sampling frequency, <0.01° RMS spatial resolution, eye-movement data were down-sampled to 250 Hz and recorded in Cortex). Eye position was calibrated using a nine-point fixation procedure. The calibration was regarded successful if the validation procedure resulted in an average gaze-position error of less than 0.5° and a maximum error of less than 1°. If necessary, calibration was repeated between blocks.

Data and Analyses

Unless otherwise stated, we only included trials for analysis in which T1 was correctly identified. Behavioral performance in the T2 identification task was calculated as proportion correct among trials where the T1 was correctly identified. MSs were identified using the algorithm by Engbert and Kliegl (2003) in MATLAB (MathWorks, Natick, MA, U.S.A). Here the time series of eye positions was transformed into velocities calculated over a moving window of seven samples. For MS detection, we used relative velocity threshold of 5 times the standard deviation of the velocity distribution and a minimum saccade duration of 6 samples. MS times were defined by the onset time of the eye movement.

To verify the validity of the detected MSs, we examined whether the eye movements classified as MSs satisfied the main-sequence criterion (Zuber et al. 1965), which requires a monotonic relationship between saccadic peak velocity and amplitude and verifies the ballistic nature of saccade execution. Figure 1C shows a consistent monotonic relationship reflected by high correlation coefficients. MSs were characteristically small, whereby the majority of gaze shifts were of less than one degree (Fig. 1D).

T1 onset time was randomized between 5 possible positions over trials (see Stimuli, Apparatus, and Task and Fig. 2A). To account for the variation in MSR observed at the different T1 onset times, we subtracted the trial-start locked MS rate from the rate observed around T1 on each given trial, leaving only the variation in MS rate unique to T1 presentation. To facilitate this operation, the binary time series of MS onset times was convolved with a Gaussian kernel with a full width at half maximum of 30 ms to produce an estimate of instantaneous MS rate per individual trial. Thus, for each trial, we subtracted the average trial-start locked rate, calculated for all trials, except the current trial of interest, from the T1-locked MS rate on that trial. MS rates are presented as MS rate corrected by the trial-start locked average, unless otherwise stated.

Statistical Testing

We were interested to test how presentation of the T1 influenced MS rate on a moment-to-moment basis and whether this temporal variation in T1-locked MS rate correlated with the detection rate. These tests were made using sliding window analysis. Correction for multiple comparisons (the number of window positions) was achieved using a cluster-based permutation method as implemented in the Fieldtrip Matlab toolbox (Oostenveld et al. 2011) and described in detail by Maris and Oostenveld (2007). To test for effects on MSR, we calculated the differences between subjects' mean MS rate profile around T1
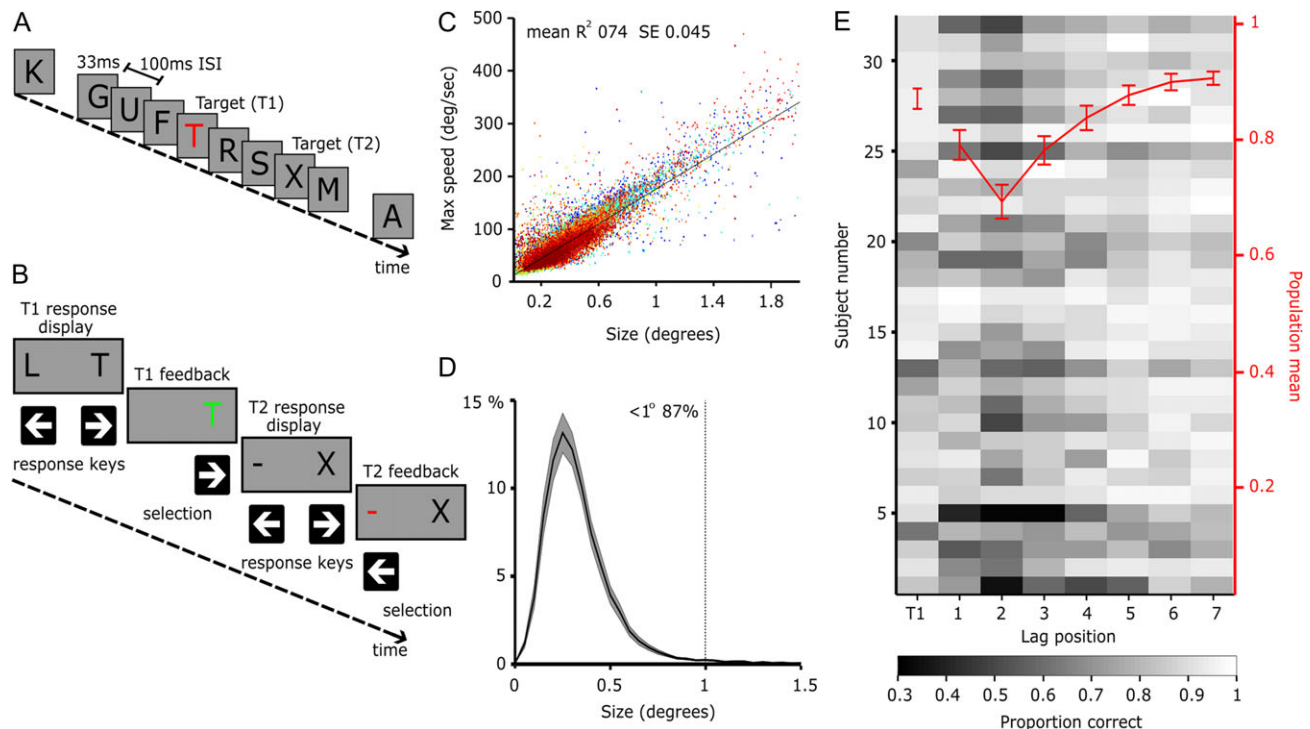
Figure 1. Task, MS, and performance quantification. (A) Each trial consisted of a stream of 27 letters. Gray letters were distracters, the red letter was target 1 (T1) and the black X was target 2 (T2). Each letter was shown for 33 ms, with an inter-stimulus interval (ISI) of 100 ms. Letters fitted into a 0.3° x 0.3° area in the experimental display, centered at fixation. (B) Example of response displays, showing response keys and feedback display for T1 and T2. Response displays were shown centrally on the screen at the end of the letter stream. Participants were not required to fixate in the response period. The T1 task required discrimination of T1 ("L" or "T") by pressing the corresponding arrow after which a correct response turned the selected letter green (or red in case of an incorrect response). The T2 task required indicating the presence of the target "X" or its absence (indicated by a—on the response screen) by the corresponding key, with a correct response indicated in green. In the case of incorrect responses, the correct response was shown in black and the given response indicated in red. (C) Quantification of MS statistics confirming the "microsaccade main-sequence" criterion: a monotonic relationship between MS size and peak velocity. Each dot corresponds to an individual MS, colors correspond to different participants. Correlations were high in all participants, with average R-squared value of 0.74. (D) Histogram of MS size, averaged over participants, line width shows standard error. Over all subjects, 87% of MS were of less than 1 degree. (E) T1 and T2 task performance. Shaded background corresponds to individual participant performance (participant number on left y-axis). Overlaid red line corresponds to population average (error bars ± SEM, values on the rightward axis).

onset and their mean MS rate profile following randomly selected distracters. For each time point, we calculated the significance of the paired *t*-test T-value using the Monte-Carlo permutation distribution after 1000 random resamplings and cluster-based correction for multiple comparisons.

To test the relationship between subjects' MSR time course and behavioral performance, we calculated the correlation between MS rate and performance at the T1 and T2 tasks. We calculated the significance of the correlation using the Monte-Carlo permutation distribution after 1000 random resamplings and cluster-based correction for multiple comparisons. For both sliding window analyses, we used a 133 ms window length, matching the stimulus onset asynchrony in the task, to calculate mean MS rate and moved the window in 25 ms steps from 500 ms before T1 onset to 1 second after T1 onset.

Electrophysiology

To illustrate our interpretation of the behavioral data, we made qualitative comparisons between the human behavioral data and macaque V1 electrophysiology collected in other experiments. All macaque data were collected in line with EU Directive 2010/63/EU for animal experiments and with the approval of local ethics committee (Radboud University Dier Experimenten Commissie). A portion of this data has been previously presented (Lowet et al. 2016; Gips et al. 2017) and

detailed methods of the electrophysiological recordings have been presented elsewhere (Roberts et al. 2013; Lowet et al. 2016). Briefly, recordings of spikes and LFP were made using linear arrays of 16 electrodes (Plexon U probes, 150 micro inter-contact spacing) inserted perpendicular to V1 cortical surface. Eye movements were monitored via an infrared camera (Thomas recording, 250 Hz). The animal's task was to maintain fixation while a luminance modulated static square wave grating (5 degree diameter, 2 cycles per degree, presentation time randomized between 800 and 2000 ms) was presented over the receptive fields of the recorded neurons. Here, we present data from using 20% contrast in one animal and 53 recording sessions. Multi-unit activity and frequency domain LFP data were averaged over all 16 electrodes along the recording probe. Hence, we present data from 848 electrodes from 1956 individual trials. Time–frequency analysis was performed using wavelet decomposition, as described in Cohen (2014).

Results

Task Performance

Figure 1E shows the suppression of T2 detection performance at different time intervals following the identification of T1 in the AB paradigm as implemented in our study. We saw in the average data (red solid lines) that the suppression of T2 detection was especially prominent at the second lag period (266 ms
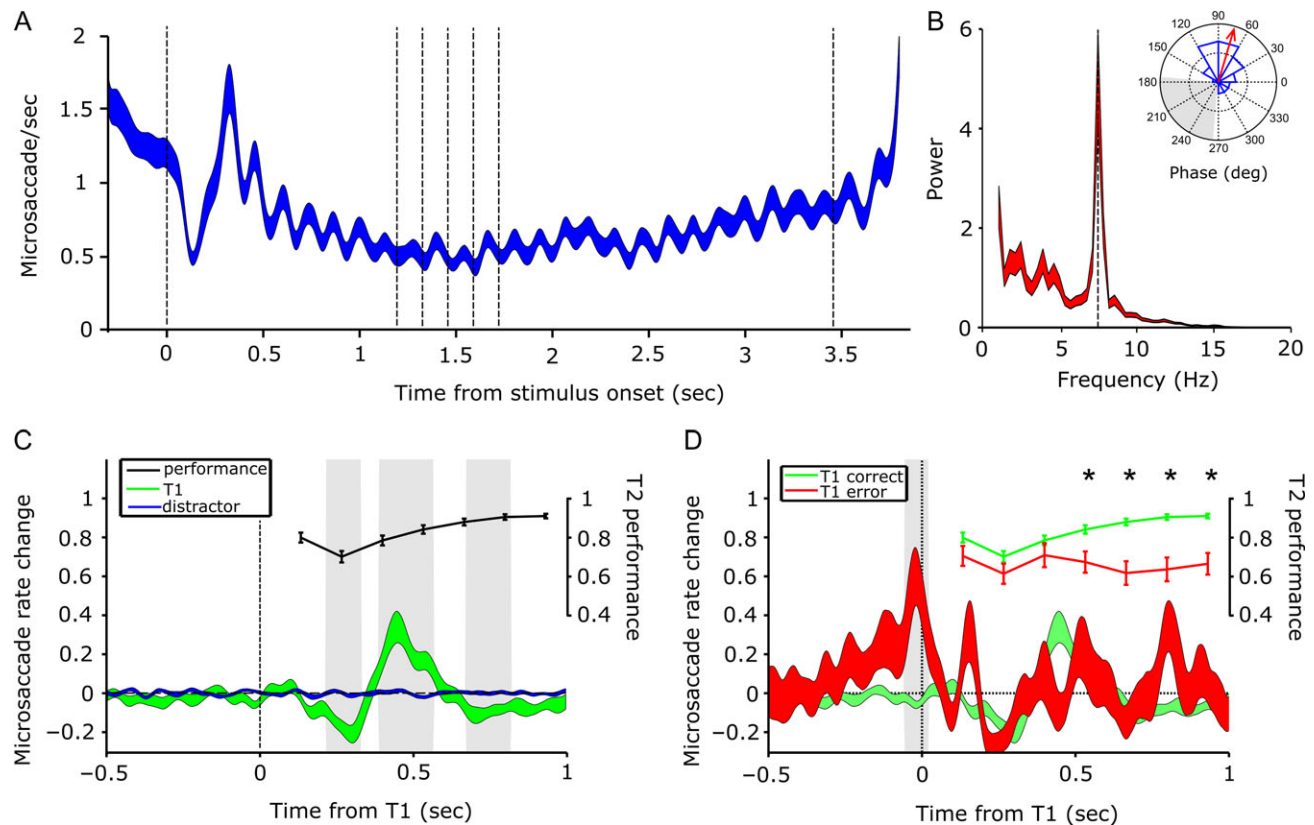
Figure 2. Microsaccade statistics as a function of duration in the trial. (A) Mean MSR (uncorrected, see data and analyses) with trials aligned to first stimulus onset. Line thickness shows standard error. T1 presentation time was randomized to 5 places in the RSVP stream ranging from place 10 to 14, indicated by five central vertical lines, additional vertical lines mark stimulus onset at time 0 and stimulus offset at time 3.4 seconds. T2 was presented at time points ranging from133 to 933 ms after T1 onset. (B) Power spectrum of mean MSR between 0.6 and 3.5 s after stimulus onset. Vertical dashed line corresponds to rate of letter onset, 7.5 Hz. Inset: histogram of phase of 7.5 Hz component per participant. Inner circle corresponds to 5 participants per bin, outer circle to 10. Gray shading indicates 33 ms period of stimulus presentation. Red arrow points to average phase over participants. (C) Variation in corrected MSR after correctly identified T1 (green) or after distracter (blue) onset (aligned to time zero). Distracters were stimuli other than T1 and T2, to which no behavioral response was required. Line width shows standard error. Gray background shows time periods of significant difference. Black line and error bars show T2 performance (mean, error bars standard error), using the rightward y-axis scale. (D) Comparison of corrected MSR around correctly (green, same data as in B) and incorrectly (red) identified T1 presentation. Gray background shading shows time periods of significant differences. Line and error bars show performance for each T2 lag position. Asterisks indicate significant differences between T1-correct and incorrect trials (paired *t*-test $P < 0.05$, uncorrected for multiple comparisons).

after T1 onset) as expected from the literature, but we also found large variability between individual participants (as indicated in the gray-scale coded T2 performance levels per participant). Studying individual differences in the attentional blink effect has provided valuable insight into the nature of the AB (see Willems and Martens 2016, for review). In the following analysis, we set out to relate individual differences in T2 detection performance to variations in microsaccade rate (MSR).

Microsaccade Rate Modulations Over the Trial

We first examined MSR over the course of the trial (Fig. 2A). This was calculated by convolving MS (onset) occurrences with a Gaussian kernel of 30 ms full width at half maximum. We found a pronounced reduction of MSR in the middle time points compared with the start of the trial and a recovery towards the end. We found in the total population of participants ($N = 31$) that MSR (per second) was 1.35 (SD = 0.6) during the 300 ms pre-stimulus period, 1.0 (SD = 0.5) during the first 500 ms showing the microsaccade signature, 0.55 (SD = 0.29) during the middle of the trial (1500–00 ms), and 0.89 (SD = 0.46) during the last 500 ms.

We observed a sharp suppression and rebound of MSR close to the start of the trial, which is a signature modulation of MSR that has been previously reported (Betta and Turatto 2006; Rolfs et al. 2005, 2008; Rolfs 2009; Hafed and Ignashchenkova 2013; Scholes et al. 2015; Xue et al. 2017). MSR suppression was centered at 137 ms after stimulus onset and MS rate rebound was centered at 325 ms after stimulus onset. We also observed a pronounced high-frequency variation in MSR throughout the trial in line with previous reports of rhythmic fluctuations of MSR during presentation of repetitive stimuli (Laubrock et al. 2008; Pastukhov and Braun 2010). Analysis of the power spectrum (without including the initial suppression and rebound phase, thus from 600 to 3500 ms after stimulus onset) showed a pronounced peak at 7.5 Hz (Fig. 2B), corresponding to the frequency of the letter presentation (vertical dashed line). The phase of the oscillation (Fig. 2B inset) was consistent across participants and corresponded to troughs of the 7.5 Hz rhythm coinciding with the presentation time of letters in the stream. Across participants the mean phase (red arrow) corresponded to MSR peaking 72 ms after letter onset (or 61 ms before the subsequent letter onset). Thus, participants appear to have adjusted their MSs, intentionally or otherwise, to avoid coincidence with letter presentation.

T1 onset times were away from the fast dynamics associated with the start of the trial, however MSR was also not stationary during this period. To test for MSR changes associated with the T1 onset, we subtracted the task-onset-locked mean MSR observed from 500 ms before to 1 s after the time of T1 onset from the observed rate on a trial by trial basis. This procedure removed both the gross differences in MSR observed between the five possible T1 onset times and the high-frequency variation in MSR associated with the onset of each letter.

Microsaccade Rate Modulations Relative to T1

After aligning MSR to correctly identified T1 onsets and correcting the observed rate by the task-start aligned mean, we observed that the average MSR rate triggered by T1 followed the MSR signature (Fig. 2C, green line). Maximum MSR suppression was at 296 ms after T1 while maximum MSR rebound was at 440 ms after T1. Thus, the MSR signature following T1 had a slower time profile to that elicited by the onset of the first stimulus in which suppression was centered 137 ms after task onset and rebound was centered at 325 ms. The MSR signature was also different from the high-frequency variation in raw MSR shown in Figure 2A in which each letter onset coincided with a dip in MSR. We had initially hypothesized that the timing of the rebound phase would align with the maximum reduction in T2 performance (observed at Lag 2, 266 ms after T1, solid

black line and error bars Fig. 2C). However, to the contrary, the period with *suppressed* MSR coincided with the reduction in T2 performance. To quantify the observed changes in MSR, we compared MSR aligned to the T1 onset with MS rate aligned to the onset of a distracter (Fig. 2C, blue line). Distracter onsets to which the data were aligned were chosen at random on each trial from stimulus Positions 10–14 (i.e., from the same range within which T1 was presented) and MSR around distractor onsets was correcting the observed rate by the task-start aligned mean in the same way as for T1 aligned data. We compared the T1 aligned data with the distracter-aligned data using a sliding window T-test with a permutation test for correction of multiple comparisons. Regions with significant ($P < 0.05$) deviation from the distracter-aligned data are highlighted in gray in Fig. 2C. We found three regions, the first corresponding to a reduction in MSR from 200 to 328 ms after T1 onset, the second corresponding to an increase (rebound) from 380 to 568, and a third corresponding to another decrease from 668 to 824 ms after T1 onset.

Microsaccade Rate Around T1 Errors

We next compared MSR around correctly (Fig. 2D, green) versus incorrectly (Fig. 2D, red) perceived T1 stimuli. The MSR curve in error trials was noisier, likely reflecting fewer trials. Nevertheless, when considering the time period following T1 onset, the error–trial curve (red) largely followed the correct–
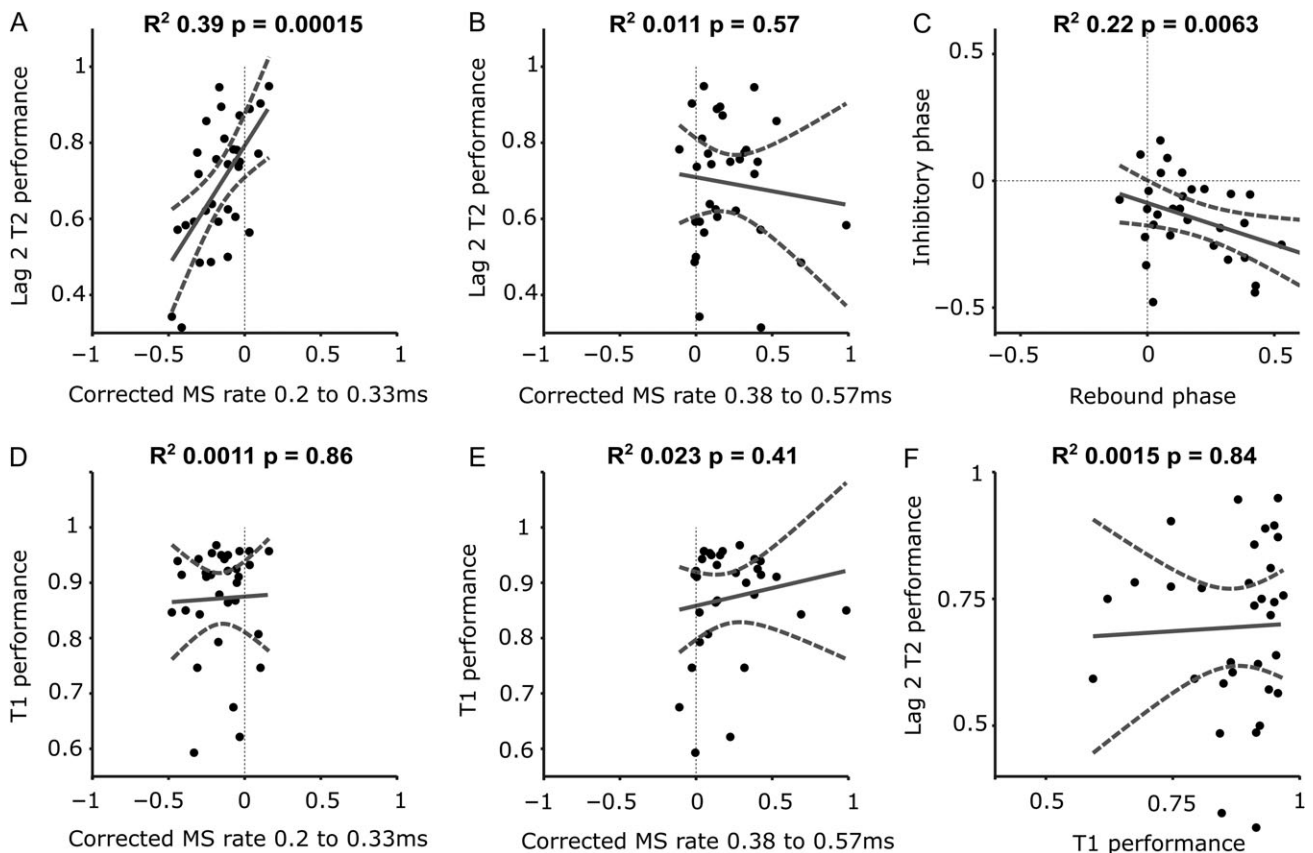

Figure 3. Correlations between task performance and the MSR signature. Top row. Lag 2 T2 performance correlates with MSR suppression (A) and rebound (B) of MSR signature. (A) Each point represents the MSR (x-axis) of one participant averaged from 0.2 to 0.33 s after T1 presentation (inhibition, first shaded area in Fig. 2C) and Lag 2 performance (y-axis). Values give R^2 and P value of linear regression, dashed lines show regression 95% confidence intervals. (B) The same but for MSR 0.38–0.57 s after T1 presentation (rebound, second shaded area in Fig. 2C). (C) Each point represents MSR inhibition (y-axis) and MSR rebound (x-axis). Bottom row. (D) T1 performance does not correlate with MS rate during the suppression or (E) rebound. (F) T1 performance does not correlate with T2 performance at Lag 2.

trial curve (green). Interestingly, however, a significant divergence between ($P < 0.05$, t-test, corrected using cluster-based permutation testing) the two curves occurred directly before and during T1 presentation, where MSR was significantly higher in error trials than in correct trials (gray shading). This is in line with the idea that perception was suppressed during the MS movement (recall that MS times were taken as the MS onset time). Performance in the T2 task was generally lower in T1 error trials (two factor ANOVA, Factor 1 correct versus error $F = 58.36$, $P < 0.001$, Factor 2 T2 lag $F = 2.21$, $P = 0.041$, interaction $F = 2.29$, $P = 0.035$), but was only significantly lower in Lags 4–7 (post hoc test). For the remaining analyses presented in this paper, we only used data from trials in which T1 was correctly identified.

Microsaccade Rate Signature Correlates with Attentional Blink

To quantitatively examine the relationship between MS rate modulation and T2 performance, pointed to in the last section, we plotted (Fig. 3A) individual participant's Lag 2 T2 performance as a function of MSR (always corrected by the mean MSR in the task-start aligned data) during period of MSR suppression (see first gray region in Fig. 2C, 200 to 320 ms after T1, see Fig. 2C). The two factors were significantly positively linearly correlated ($R^2 = 0.39$, $P < 0.001$) implying that participants who showed the largest MS rate suppression after T1 presentation also showed the largest T2 performance reduction. We also examined the correlation between the MSR in the rebound phase (see middle gray region in Fig. 2C, 380 to 568 ms after T1 onset) and T2 performance at Lag 2 (Fig. 3B). Here, there was no correlation between the two factors ($R^2 = 0.011$, $P = 0.57$). Interestingly, there was a significant correlation between the size of the suppressive phase and the rebound phase ($R^2 = 0.22$, $P = 0.006$, Fig. 3C).

We next considered the possibility that the correlation between T2 performance and the associated MSR suppression was related to the visibility of T1. That is, participants who were not sensitive to T1 may have shown neither an attentional blink nor a modulation in MS rate following T1 onset. Arguing against this possibility, we found no relationship between T1 performance and the strength of MS rate suppression (Fig. 3D) or rebound (Fig. 3E). T1 performance was moreover not related to T2 performance (Fig. 3F), in line with earlier reports (e.g., Shapiro et al. 1994). Visser (2007) found that T1 task difficulty did not modulate AB magnitude when T1 was followed by a backward mask (numbers presented in the Lag 1 position) but did in the absence of a mask. In our design, T1 was indeed masked by the letter at Lag 1, thus our data seems consistent Visser's conclusion.

Microsaccade Rate Signature Correlates with Perceptual Performance

To gain further insight into the relationship between MSR changes around the time of T1 presentation and the participants' performance in the task, we extended the correlation analysis shown in Figure 3 using a sliding time window (Fig. 4). At each position of the window (133 ms width, 4 ms steps), we calculated the correlation between each participant's MSR and their T2 performance. In addition to correlating MSR with performance at Lag 2, we also correlated MSR to performance at all other T2 lags and with T1 performance. The relationship of the
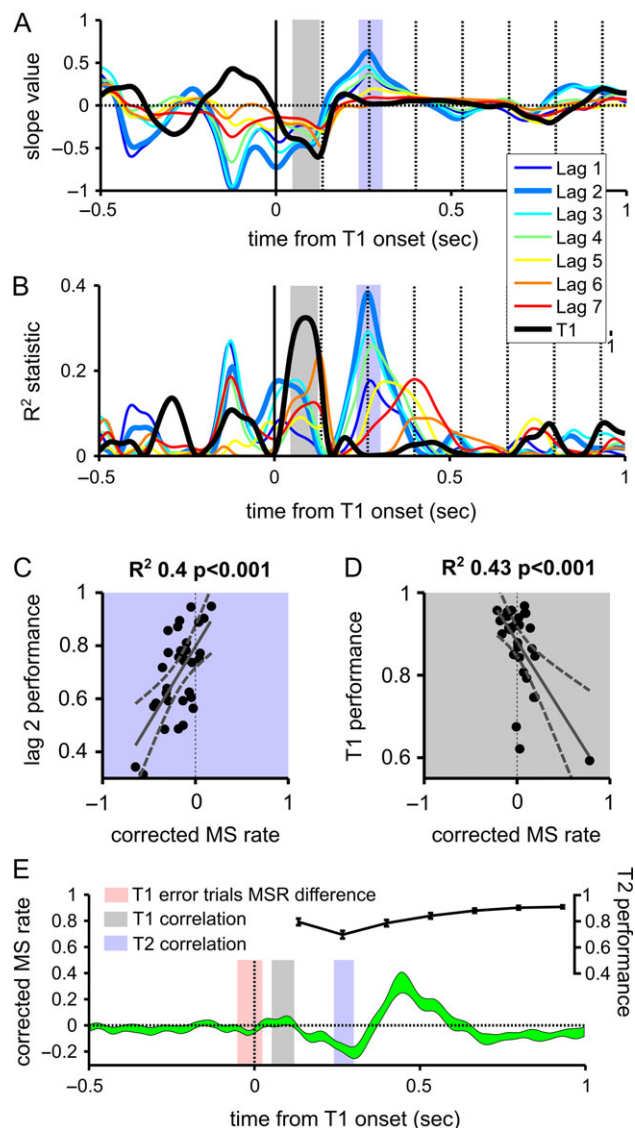

Figure 4. Sliding window correlation of MSR against task performance for T2 in each lag (line colors) and for T1 (black line) as a function of time from T1 onset. Conditions for which the correlation was significant at some point are shown in thicker lines (thick black line for T1 and thick blue line for T2 at Lag 2), and periods in which the correlation was significant are shown in colored backgrounds ($P < 0.05$, after correction for multiple comparisons). Vertical dashed lines indicate T2 onset times. (A) Regression slope of corrected MSR to performance. Positive slopes imply poor performance among subjects with low MS rate. (B) Correlation coefficient R^2 of corrected MSR to performance. (C) Correlation between corrected MSR and T1 performance during the gray period highlighted in A and B. (D) Correlation coefficient R^2 between corrected MSR and T2 performance during the blue period highlighted in A and B. (E) Summary figure, showing corrected MSR (in green) and behavioral T2 performance (in black, rightward y-axis scale) relative to T1 onset. Gray and blue highlighted periods are as in A and B, and indicate significant correlations of MSR with T1 performance (Fig. 4C) and Lag 2 T2 performance (Fig. 4D), respectively. Red highlight indicates region where MSR was significantly higher in T1 error trials than in T1 correct, as in Figure 2D. Hence, in Figure 4E, the red and gray highlighted regions are periods in which MSR suppression helps T1 performance, and the blue region is a period in which MSR suppression hurts T2 performance.

MSR in the sliding time window to eight different performance measures is shown in eight curves, color coded for T1 and Lags 1–7 (Fig. 4A, B). This relationship is shown as the slope value of
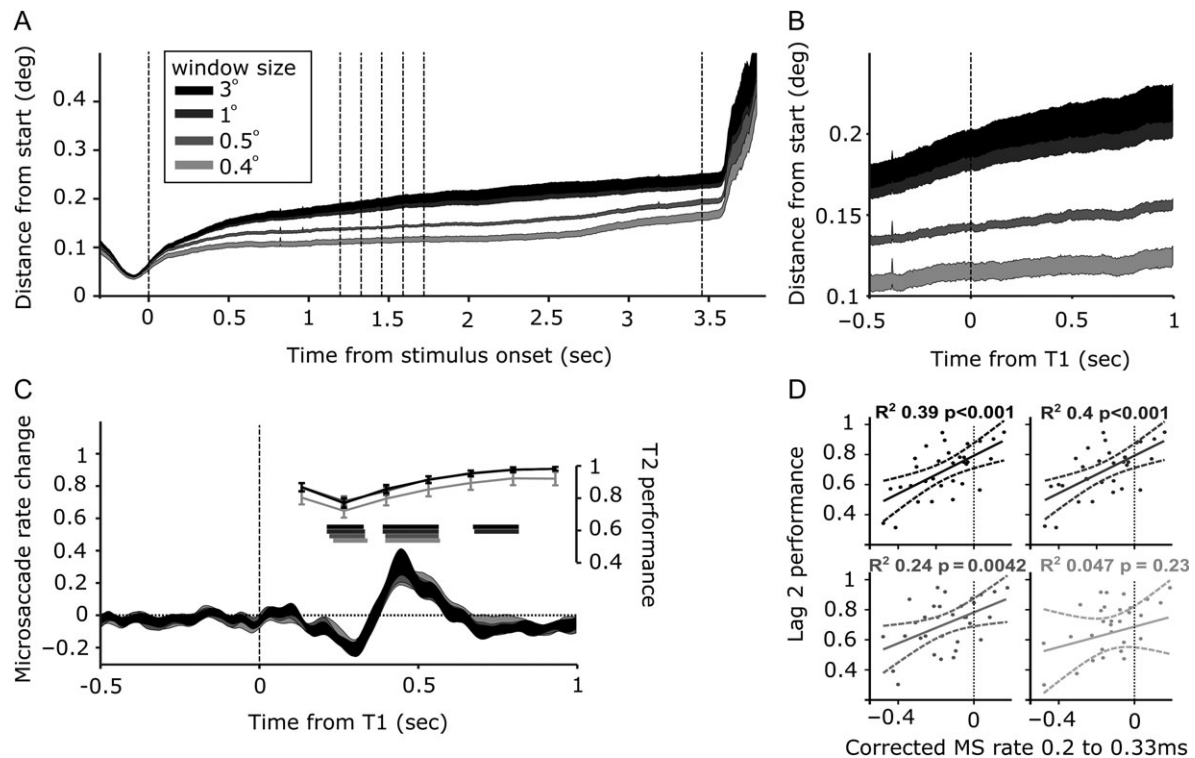
Figure 5. Eye position analysis. (A) Eye position relative to start position (mean position between −0.1 and 0 s before stimulus onset) as a function of time from stimulus onset. Line center indicates mean eye position, line width indicates standard error. Line colors indicate threshold for post hoc rejection of trials: black all trials, dark gray threshold=1°, mid gray 0.5°, and light gray 0.4°. Vertical dashed lines show stimulus onset (Time 0), T1 onsets (central five lines) and stimulus offset (3.4 s). (B) As in A but aligned to T1 onset (vertical dashed line). (C) Corrected MSR aligned to T1 onset (as in Fig. 2C) for each threshold. Horizontal lines indicate regions of significant difference from distractor onsets (distractor onset MSR curves not shown). Lines and error bars shown T2 performance at each lag. (D) Correlation between MSR inhibition and Lag 2 T2 performance for each eye position threshold. Respective R-squared and P values of correlation are given to the side.

a linear regression (Fig. 4A) or in terms of the squared correlation R^2 (Fig. 4B).

In Figure 4A, the relationship between Lag 2 T2 performance and suppression of MSR showed prominent positive slope values around the time of Lag 2 presentation (blue highlight in Fig. 4A), as well as a large peak in R^2 (blue highlight in Fig. 4B). This confirms the strong relationship between Lag 2 performance and MSR (see scatter plot in Fig. 4D). The present analysis uncovered a second prominent correlation; T1 performance was significantly related to MSR immediately after T1 onset (black line and gray highlight in Fig. 4A, B, scatter plot in Fig. 4D). The negative slope values in Figure 4A indicate that participants with the highest T1 performance had the lowest MSR during this period. All other performance measures (i.e., other than at T1 or at Lag 2) showed no correlation with MSR at any time window.

Summary

Figure 4E provides a summary of our findings. Our data indicate that optimal performance in the T1 task was achieved by suppressing MSR around T1 onset. In Figure 2C, we showed that following T1 MSR was initially suppressed and then rebounded. Conversely, in Figure 2D, we showed that T1 errors were associated with higher MSR during T1 presentation. Finally, in Figure 4A–C, we showed that participants with the best T1 performance showed lower MSR immediately following T1 presentation. Hence, we found that withholding MSs around T1 presentation was beneficial for T1 performance (faint red and gray regions in Fig. 4E). These findings are consistent with

reduced sensitivity during the moment of an eye movement. Related to this, it is striking that participants apparently adopted a strategy of making MSs between letter presentations (Fig. 2B). This is consistent with the idea that briefly withholding MSs is beneficial for stimulus processing. Notably, high T1 performance was associated with suppressed MSR just prior to *first* T2 presentation (Fig. 4A,B), a period associated with spared T2 performance; the so-called lag one sparing. It may be that performance in the first ~100 MS following T1 presentation benefits from MSR suppression during T1.

While suppression of MSR benefitted T1 performance the opposite was true for T2 performance, where the profound MSR suppression following T1 was correlated with poor performance at Lag 2 (blue blocks in Fig. 4). One may speculate that the MSR observed during and shortly after T1 presentation was the "optimal" level of suppression for letter identification. Accordingly, the MSR signature in response to T1 presentation (Fig. 2D) may have brought the level of MSR suppression beyond the optimal level, leading to reduced T2 performance. Our data indicate that optimal T1 performance was achieved by avoiding MS from ~50 ms prior to T1 presentation until ~130 ms after (faint red and gray regions in Fig. 4E). Following the ~180 ms period of optimal suppression around T1, continued suppression was associated with poor performance in the attentional blink interval (blue blocks, Fig. 4). Performance in the T2 task recovered together with MSR from the period of Lag 3 onwards.

Taken together, our data show that a brief period of relative MS reduction prior to and following T1 facilitated T1 performance. In addition, the MSR signature following correctly

discriminated T1 stimuli caused a further reduction of MSR coinciding with a period centered on Lag 2, which was associated with impaired T2 performance. This suggests that both the presence and the absence of MSs can impair performance, the "built in paradox" of the visual system (Martinez-Conde et al. 2006).

Post Hoc Restriction of Eye Position

We used a relatively wide fixation window, in which trials were aborted only if eye position moved beyond a 3° by 3° window. We used a large window to compensate, not for eye movements but for head movements which participants sometimes made between trials, since they were imperfectly stabilized only with a head and chin rest. Smaller windows were frustrating to our participants when they tried to start fixation, although once started they could generally complete the trial while maintaining good fixation. Nevertheless, the possibility exists that participants moved their gaze away from the letter stream over the course of the trial, which could contribute to poor performance. The perception of T2 could be particularly susceptible to shifts in eye position following T1 given Xue's (2017) data that MS amplitude was particularly large during a period of relative MSR inhibition following the onset of an attentional cue. While most MSs in our data were small (Fig. 1D), drifts and consecutive MSs in a consistent direction could shift the letters outside the fovea. To test for possible contributions of eye position to our findings, we calculated the time-resolved absolute distance between participants' eye position relative to their starting position at each trial (Fig. 5A). Average eye position gradually shifted away from the starting position over the course of the trial reaching 0.24° (standard deviation 0.06) by the end of the RSVP time (black lines show data from all trials, line width shows standard error). After aligning the eye position data to T1 onset (Fig. 5B), we saw no evidence that T1 presentation influenced the rate of this gradual drift. To further test for an influence of shifting eye position, we post hoc restricted eye position by rejecting trials in which eye position shifted by more than 1° (dark gray), 0.5° (mid gray), and 0.4° (light gray) from the starting position during the RSVP time. We thereby rejected respectively an average of 2%, 23%, and 39% of trials (standard deviation 2.8, 15.6, and 18.7). We repeated key analyses with these selected data. In all selections, the observed T2 performance reduction (performance difference between lags, ANOVA, $P < 0.001$ for all trials, 1° restriction and 0.5° thresholds, $P = 0.006$ for 0.4 threshold). The MSR signature following T1 onset remained statistically significant at the initial suppression and rebound phases (thick horizontal lines mark regions of significant difference from distractor onset, following convention of gray background filling in Fig. 2C). The second period of suppression was not significant for the 0.5° and 0.4° thresholds. The correlation between MSR inhibition and attentional blink magnitude was significant for the 1° and 0.5° threshold but not for the 0.4° threshold (see values in figure). These data are in line with the idea that participants faithfully maintained fixation on the letter stream and that any small shifts in eye position did not contribute to our findings.

Discussion

We have used a RSVP in which participants showed reduced correct detections of a target T2 presented at 266 ms following the correct discrimination of a target T1, a phenomenon referred to as the attentional blink (AB). Here, we investigated the contribution to these behavioral findings of variations in microsaccade rate (MSR). We found a pronounced MSR signature following T1 presentation characterized by MSR suppression during the period of time associated with reduced T2 detection, followed by MSR rebound. The strength of this suppression was moreover strongly correlated with the extent of the T2 performance deficit across participants, indicating a role for MSR suppression in the AB phenomenon. In addition, we found that error trials in the T1 task were associated with higher MS rate at the moment of T1 presentation. Thus, we found that poor performance in the T1 task was associated with MS occurrence, whereas poor performance in the T2 task was associated with MS absence.

Comparison with Electrophysiological Correlates of MS

Our behavioral data suggest that the presence of a MS, or the prolonged absence of MSs can lead to similar reductions in stimulus processing. Stimulus processing capacity is related to the excitability of visual cortex which may be indexed by multi-unit firing rates and by gamma band power and frequency (Hadjipapas et al. 2015; Lowet et al. 2015). Neuronal excitability is modulated by eye movements, where we and others find rapid suppression during and shortly before the movement (Leopold and Logothetis 1998; Hass and Horwitz 2011; Meirovithz et al. 2012; McFarland et al. 2015; Lowet et al. 2016), followed by a short-lived enhancement of activity (Leopold and Logothetis 1998; Kagan et al. 2008; Bosman et al. 2009; Hass and Horwitz 2011; Meirovithz et al. 2012; McFarland et al. 2015; Troncoso et al. 2015), although there is diversity in the extent of suppression and enhancement (see Martinez-Conde et al. 2013 for review). These effects are reported as early as the LGN, and we have previously shown in a model how the MS effects observed in early visual cortex can be approximated by modulating input using a double-exponential kernel fit to match observations in the LGN (Fig. 6A, see Lowet, Gomes et al. 2018 for full details). Under normal viewing conditions MS happen quasi-periodically (Fig. 6B dashed line) which refresh the image but lead to regular periods of poor perception at the moment of each MS (red portion corresponds to excitability below an arbitrary threshold). If a MS is left out (Fig. 6B solid line where MS 2 is missing), the refresh does not arrive and cortical excitability decays according to the time course of the kernel, leading eventually to poor perception (red solid line) which does not recover until the next MS. According to our interpretation, T1 errors arise due to the regular MS induced inhibition while T2 errors arise due to the decay when MSs are missing.

To illustrate this idea further, we inspected electrophysiological data from macaque area V1, recorded during prior experiments (Lowet et al. 2016; Gips et al. 2017). We combined spiking and time–frequency analysis representations across all MSs recorded in our macaque data (Fig. 6C). During and shortly before the movement (gray line), spike rates (black line) and gamma power (color scale) were suppressed. Saccadic suppression is a well-studied phenomenon that has been extended to MSs in a number of reports (e.g., Hass and Horwitz 2011; McFarland et al. 2015; Leopold and Logothetis 1998, see Martinez-Conde et al. 2013 for review). Such suppression could well be expected to interfere with the processing of incoming stimuli, in line with previous studies (Zuber and Stark 1966; Hass and Horwitz 2011; Xue et al. 2017; Scholes et al. 2018). Thus, we argue that the higher MSR we observed around T1 onset in T1 error trials (Fig. 6D) induced reduced cortical
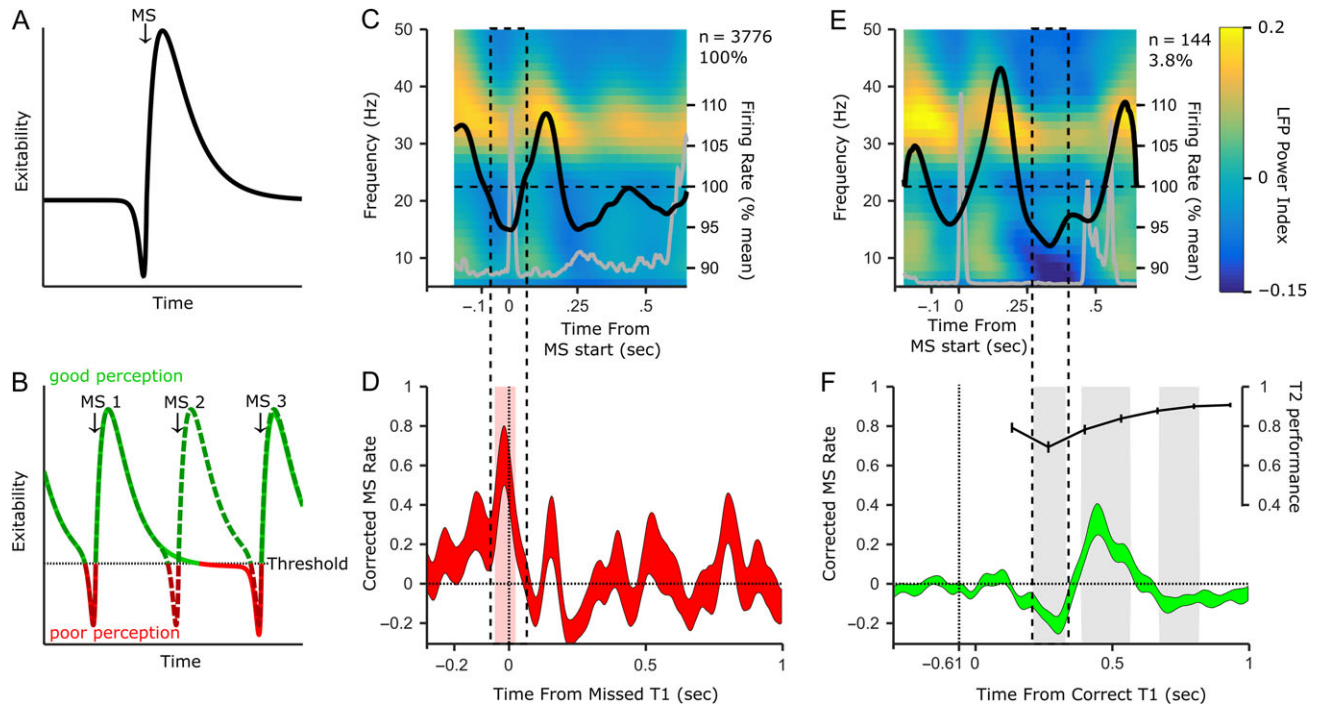
Figure 6. Comparison between experimental findings and electrophysiological model and data from separate experiments. (A) Model input kernel fit to match observations in LGN. B) Regular MSs are modeled by combining the input kernel placed at each MS time. Lower cortical excitability is assumed to lead to poorer perception, here an arbitrary threshold in placed slightly above 0 input. Regular MS lead to regular peaks and dips in cortical excitability (dashed line). If MS 2 is missed (solid line) cortical excitability remains low during the period of MS 2. (C and E) Electrophysiology from one macaque V1 around MSs. Color surface shows time–frequency representation of power in V1 LFP. Color-scale shows power change relative to pre-stimulus (power difference/power sum). Black line shows mean firing rate as a percentage of mean firing rate during the sustained stimulation period (from 250 ms after stimulus onset), values are given on the rightward y-axis, horizontal dashed line corresponds to mean firing rate. Gray line shows mean normalized eye velocity (normalized to peak velocity during MS, units not shown). We used normalized eye velocity around each detected MS to accept or reject the data. (C) Data associated with typical MSs; all detected MS ($N = 3776$) are included. (E) Data associated with MSs that were followed by prolonged MS suppression. Exclusion criteria were the following: maximum eye velocity 120–450 ms after the movement below 20% of the maximum, and eye velocity greater than 20% in the period 450–550 ms, matching MS rate suppression and rebound observed after correct T1 trials in the main experiment. Only 3.8% of the population satisfied the criterion (144 MSs). Lower plots: Human eye-movement data from attentional blink experiment. (C) MSR rate in T1 error trials, with highlighted region showing where MS rate differed significantly from that in T1 correct trials (as in Fig. 2D). Dashed box facilitates comparison of period of poor perception (centered on T1 onset, width= 133 ms) with electrophysiology. Notice that higher MSR at T1 onset might be expected to correspond to suppression of spiking and gamma power in V1, potentially contributing to poor perception. (D) MSR and T2 performance around T1 correct trials. Shaded regions show where MSR differed from MSR after distracter onset. Notice that the time axis of (B) is shifted to represent MSs occurring at the time between letter onsets indicated by vertical dotted line (see Fig. 2A,B), T1 onset thereby coincided with rising spiking and gamma power. Dashed box facilitates comparison of period of poor perception at Lag 2 presentation with corresponding neuronal activity. Prolonged absence of a second MS lead to a reduction of spiking and gamma power during the period corresponding to Lag 2 presentation.

excitability at the moment of T1 presentation (Fig. 6C), thereby interfering with T1 processing (see dashed box in Fig. 6C,D). Accordingly, avoiding MSs during T1 presentation will facilitate correct T1 performance.

In trials in which T1 was correctly reported we found MSR suppression followed by a rebound (Fig. 2C). To qualitatively compare this with neurophysiological data associated with MSs in the monkey, we selected MSs that were followed by stable eye position for 450 ms and a second MS in the period of 450–550 ms following the first (Fig. 6E). Because monkeys had a higher MS rate than the participants in our study, and humans in general (Pastukhov and Braun 2010; Otero-Millan et al. 2008), these made up only 3.8% of all MS we had observed, yet given the size of our database this corresponded to 144 observations. To align the time course of neurophysiological data (Fig. 6E) with the time course of MSR (Fig. 6F), one needs to take into account the finding that in T1 correct trials MSR peaked between letter presentation, at 61 ms before each letter onset (Fig. 2A,B). Therefore, we aligned Time 0 of Figure 6E (the MS onset in electrophysiology data) with –0.61 in Figure 6F (the attentional blink data). Plotting the selected electrophysiological data

against the human eye-movement and behavioral data, we constructed an idealized illustration of a correct T1 trial, in which a MS occurred just before T1 onset and was followed by suppressed MSR until a rebound period at around 500 ms after T1 onset.

Aligned in this way, we see in Figure 6E that "T1 onset time" coincided with a peak in gamma band activity and with rising spiking activity. The peak was however short lived; the spike rate returned to a mean level after 200 ms and continued to decline over the following ~150 ms. Remarkably, spike rate and gamma power reached a minimum during the period corresponding to the period Lag 2 in our behavioral data (Fig. 6F, see dashed box in Fig. 6E,F). Note that the short-lived boost of neuronal activity following a MS shown in Figure 6B has been reported also in other monkey neurophysiology studies (Leopold and Logothetis 1998; Bosman et al. 2009; Hass and Horwitz 2011; McFarland et al. 2015). One study (Troncoso et al. 2015) also observed significant neuronal response suppression after a period of post-microsaccade facilitation. However, these studies did not require a prolonged period of stable eye position after the trigger MS. It is, therefore, unclear whether they would also have seen the continued decline in activity we observe in

Figure 6E and which we related here to the reduced T2 performance in humans.

We emphasize that this analysis is aimed to provide only an idealized illustration of how the MSR changes we observed in the attention blink paradigm could interact with known MS-dependent neuronal excitability. The comparison between two very different data sources does not give direct support for a suppressed MSR-induced reduction of cortical excitability as a contributor to the attentional blink paradigm and is provided here simply to illustrate our interpretation. Limits in the comparability of the behavioral and neurophysiological data sets include the differences in stimuli (respectively centrally presented letter RSVP vs peripherally presented static gratings), including their spatial frequency content (Scholes et al. 2018), differences in task, and other factors. Nevertheless, we suggest that it is an interesting hypothesis that the modulation in MSR we observed following T1, and especially the period of MSR suppression, might lead to reduced V1 excitability during the Lag 2 presentation time, which in turn could contribute to the poor perceptual performance at that moment. Such a hypothesis could be tested in a combined behavioral, electrophysiological, and eye-movement study and could limit the role of higher-level cognitive factors, such as temporal attention, in explaining the attentional blink phenomenon.

MSR Signature and the Attentional Blink

We have suggested that the MSR suppression that coincides with the Lag 2 T2 errors is part of the MS signature induced by T1. In our experiment, as in the original AB experiments (Raymond et al. 1992), the T1 was colored differently from the distracters and would, therefore, be described as an oddball. The pattern of MS suppression after T1 onset reported here was similar to the pattern reported in response to a visual oddball in other RSVP tasks (Valsecchi et al. 2007; Valsecchi and Turatto 2009). Valsecchi et al. (2007) and Valsecchi and Turatto (2009) compared MSR between oddballs and standard stimuli and found the MSR suppression for both stimulus categories but rebound only for standard stimuli. This differs from our results in that we found both suppression and rebound following the T1. Interesting as well, is that suppression and rebound were more rapid in Valsecchi's experiments compared with what we observed for T1. In Valsecchi et al. (2007) and Valsecchi and Turatto (2009), the suppression peaked at around 100 ms post stimulus and the rebound at around 300 ms, which better matched the MSR signature we observed at the onset of the RSVP stimulus (Fig. 2A). This difference may reflect the different timing sequence between the two paradigms, whereby stimulus onsets were separated by 133 ms in our experiment and by 1000 ms or more in Valsecchi's experiments.

If the oddball status determines the MSR signature of a stimulus, then manipulations that would reduce the oddball status should affect the MSR signature. In line with this, Scholes et al. (2015) and Rofs et al. (2008) reported that changes in stimulus contrast affect the suppressive part of the MSR signature (as well as the rebound). Larger contrasts, which would strengthen the oddball status, elicited more pronounced MSR suppression. Correspondingly, higher contrast T1 has been found to induce a larger attentional blink effect (Chua 2005). Overall, these data further support the idea that the MSR suppression around T2 presentation is part of the T1-triggered MSR signature that is related to T1's oddball status. Given the strong positive correlation of the T2 performance deficit with the depth of MSR suppression, this in turn suggests a large contribution of an oddball-driven MSR signature on the suppression of T2 perception.

The classical interpretation of the reduced T2 performance in the AB paradigm is that the more attention is deployed to T1, either because it is more salient, more task relevant or requires more encoding into working memory, the greater the attentional blink will be. In line with this, drawing attention away from T1, for example by explicitly instructing participants to ignore it (Raymond et al. 1992) tends to reduce the T2 deficit. Given proposed links between eye movements and attention (Corbetta et al. 1998; Moore 2001; Thompson 2005), including links between MSs and attention (Hafed and Clark 2002; Engbert and Kliegl 2003; Galfano et al. 2004; Rolfs et al. 2005; Rolfs 2009; Laubrock et al. 2010; Pastukhov and Braun 2010; Pastukhov et al. 2013; Yuval-Greenberg et al. 2014; Meyberg et al. 2015; Tian et al. 2016; Lowet, Gomes et al. 2018) the question can be asked whether attention to T1 could contribute to the T1 MSR signature, which in turn appears to contribute to the T2 deficit. Specifically, would the MSR suppression we observed around T2 be reduced if T1 had been less attended? Interestingly, attention has been reported to modulate the MSR signature. For example, Dalmaso et al. (2017) found an enhanced rebound under conditions of low working memory load, but no difference in MSR suppression, whereas Valsecchi et al. (2007) found enhanced MSR suppression for oddballs that were behaviorally relevant. In our data, the microsaccade signature, including the MSR suppression, was unaffected by either the correctness of T1 trials or variations in the level of T1 performance across participants (which correlated with MSR before suppression became significant). When accepting that variations in the level of T1 performance or the correct/incorrect status of T1 discriminations indexes attentional variations, then our data do not show evidence that attention modulates the MSR signature. Moreover, the idea that reduced attention to T1 would reduce the T2 performance deficit is not a generalized observation, and notable exceptions have been reported (e.g., Stein et al. 2010; Van Der Burg et al. 2013). This can be seen as in line with the lack of correlation between T1 and T2 performance in our data, as also reported by others (Shapiro et al. 1994; Chua 2005; Visser 2007). Thus, neither T2 performance nor the amplitude of the MSR signature seemed to be driven by T1 performance. While this doesn't exclude an attentional contribution to the T2 performance deficit, it raises the question by which mechanisms this would occur, and whether the attentional contribution would take place in a more direct manner without the intermediary of MSs.

Reports of the AB in cross-modal paradigms are also relevant to the question about the extent to, and manner in which, a lack of attention resources drives the reduction in T2 performance. While there are many conflicting reports, there exist sufficient reports to show that under certain conditions an auditory T1 can induce a visual AB (Jolicoeur 1999). This could be seen as an argument for an attentional explanation of the T2 performance deficit that is not mediated by modulations in MSR. However, it is interesting to note in this regard that auditory stimuli can also to trigger a MSR signature (Rolfs et al. 2005; Valsecchi and Turatto 2009; Widmann et al. 2014). This suggests that even in cross-modal designs, the MSR signature could be an important contributor to the AB effect. Conversely, our current results suggest that the blink effect will be absent in tasks that do not yield a suppression of MSR following the T1.

Our data show that the T2 performance deficit was positively correlated with MSR suppression, which according to

neurophysiological data would be expected to reduce visual cortex excitability during the AB period. MSR suppression and T2 performance were neither dependent on variations in T1 performance across participants, nor on the correctness of T1 discriminations, which both may be seen as variations in attention paid to T1. Instead our data, in combination with reviewed literature, support that the physical attributes and oddball status of the T1 stimulus trigger MSR suppression around Lag 2 in a bottom-up manner. Hence, an interaction between the physical attributes of T1, MSR, and visual cortex excitability, may underlie the T2 performance deficit around Lag 2. This raises the question of the extent to which the attentional blink is attentional. Although the data presented here do not exclude a contribution of attention, the data invite a further investigation of the mechanisms by which attention would influence the T2 performance deficit, potentially in experiments that pit T1 visibility and (top-down) attentional manipulations against each other. Irrespective of attentional contributions, the present report shows for the first time that MSR suppression is an important factor contributing to the T2 performance deficit.

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