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Backward masking interrupts spatial attention, slows downstream processing, and limits conscious perception

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ABSTRACT

The attentional blink (AB) is a difficulty in correctly processing a target when it follows one or more other targets after a short delay. When no backward mask is presented after the last critical target, there is no or little behavioral AB deficit. The mask plays an important role in limiting conscious access to target information. In this electrophysiological study, we tested the impact of masking on the deployment and engagement of attention by measuring the N2pc and P3 components in an RSVP paradigm. We found that the presence of a mask in an AB paradigm reduced the amplitude of the N2pc, P3a, and P3b components. In addition to reducing encoding in memory, masking also reduced the effectiveness of the deployment and engagement of attention on the last target. We discuss the role of these findings in the context of current masking, consciousness, and AB models.

1. Introduction

Visual information processing has been studied extensively over several decades by pushing the systems implicated in this processing to their limits. The rapid serial visual presentation (RSVP) paradigm has proven useful to determine the temporal limits of visual processing (Potter & Levy, 1969). RSVP involves a sequence of stimuli usually presented at the same spatial location one after the other, each one for a brief period of time (e.g., 100 ms). It was discovered in 1987 (Broadbent & Broadbent, 1987; Weichselgartner & Sperling, 1987) that when subjects are asked to report two targets (T1 and T2) among distractors (D), separated by a 200–500 ms lag (e.g., D, D, D, T1, D, D, T2, D), the accuracy of report for the second target is lower compared to when it is presented later relative to the first target (e.g., D, D, D, T1, D, D, D, D, D, D, T2, D). This phenomenon gathered a lot of interest through the years from scientist seeking to understand capacity limitations in the information processing system and the correlates of consciousness. The cause of this deficit, termed attentional blink (AB; Raymond, Shapiro, & Arnell, 1992), has yet to be fully understood, despite a wide range of empirical papers on the subject and a growing number of proposed models (see Dux & Marois, 2009; Martens & Wyble, 2010, for reviews).

A number of factors have been found to influence the AB, one of which is the backward masking caused by the distractor following T2, which we will call T2 + 1. Masking is defined as the reduction in visibility of a stimulus (target) by a spatially or temporally close second stimulus (mask) (Bachmann, 1984; Breitmeyer & Öğmen, 2006). Giesbrecht and Di Lollo (1998) found that

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when the RSVP in an AB paradigm ended with the last target instead of an additional distractor (a mask), no behavioral AB occurred; accuracy for the last target was at ceiling. Later work showed that an AB can be found even when the last target is not masked as well as when distractors are replaced by blank intervals, but it is invariably smaller in amplitude compared with the AB found with a trailing mask (c.f., Arnell & Jolicœur, 1999; Nieuwenstein, Potter, & Theeuwes, 2009; Ptito, Arnell, Jolicœur, & Macleod, 2008). In the present work, we will use a backward noise mask designed to make more difficult the processing of both the shape of the target to be reported, as well as its color. The latter was important because color was the attribute used to determine which stimulus (among two) was relevant for the task. Although manipulations of masking have been used a number of times in the AB paradigm to study how processing unfolds over time, less is known about how masking affects the deployment of visual spatial attention in this paradigm. Masking is often assumed to erase the visual information from the target or to interrupt its processing, although theories which are seeking to explain masking are more complex and more nuanced (Breitmeyer & Öğmen, 2000). In general, a better understanding of the role of masking on the deployment of attention in AB would be desirable.

Here we recorded the electroencephalogram (EEG) and used the event-related potential method (ERP) to deepen our knowledge of how masking the last target in an RSVP stream affects the temporal dynamics of attention and other mechanisms involved in the processing of said target from stimulus input to representations that are amenable to conscious access. Our goal was to isolate ERP components associated with a precise cognitive process from the complex stream of processing stages mediating performance in the AB paradigm. We measured ERPs elicited by the last target in an AB paradigm and compared them between lag 3 (short delay between the last and penultimate targets) and lag 8 (long delay between the targets) trials and between trials in which the last target was either masked or not masked.

Vogel and Luck (2002) were among the first to study the impact of masking on ERP components in the AB. The P3b component yielded interesting results. This component is typically observed at mid-parietal electrode sites (i.e., Pz) and is thought to reflect working memory encoding (Fabiani & Donchin, 1995; Polich, 2007). As in previous studies, they found an almost completely suppressed P3b during the AB for masked trials at short lags (e.g., Vogel, Luck, & Shapiro, 1998). When T2 was not-masked however, the P3b component was not suppressed in terms of amplitude, but the onset of the P3b was delayed at lag 3 compared to lag 7 despite accuracy levels suggesting no AB (Vogel et al., 1998; see also Sessa, Luria, Verleger, & Dell'Acqua, 2007). These findings were particularly important because they suggested that the absence of an AB effect on accuracy of report of T2 could not be interpreted as an absence of AB interference on the processing of T2. The delay of the P3b provided strong evidence for either an interruption or a slowing of encoding of T2 resulting from concurrent processing of T1 (see Jolicoeur & Dell'Acqua, 1998). This important finding was replicated in other studies (e.g., Dell'Acqua et al., 2015; Ptito et al., 2008). Vogel et al. (1998) argued that the perceptual representation of T2 could be sustained for a relatively long period of time if it was not followed by another item (see also; Jolicœur, 1999a). This representation was therefore still available when the encoding of T1 was completed, allowing subsequent but delayed processing of T2. If a mask (a distractor) followed T2, however, its perceptual representation was lost and/or overwritten by the subsequent stimulus before encoding processes devoted to T1 were available for T2. This made the last target unavailable for conscious report. Interestingly, Jolicoeur and Dell'Acqua (1998) reported several experiments in which visual stimuli that had to be encoded for later report (at the end of each trial) were followed by a second stimulus that required an immediate speeded response. Response times increased as the delay between these two stimuli was reduced. This finding suggested that encoding visual stimuli for later report was sufficient to delay or slow the processing of trailing stimuli (see also Jolicœur, Dell'Acqua, & Crebolder, 2001). The delay of P3b onset at short lag in the AB is consistent with the increases in response time reported by Jolicoeur and Dell'Acqua (1998) or Jolicœur et al. (2001; see also Dell'Acqua, Jolicœur, Vespignani, & Toffanin, 2005; Jolicœur, 1999b).

If we assume that the P3b component reflects the process of encoding information into a general working memory system, then the results described briefly in the foregoing passages suggest that encoding T1 in the AB paradigm slows or postpones one or more operations prior to, or at, the passage of information into working memory. According to locus-of-slack logic (e.g., Pashler & Johnston, 1989), results reported in Jolicœur et al. (2001) suggested that effects of the contrast of T2 on response times were underadditive with decreasing SOA between T1 and T2, suggesting in turn that very early sensory processing of T2 takes place before—and is therefore not affected by—capacity limitations causing the AB (see Jolicœur et al., 2001, for a discussion of the locus-of-slack logic on the context of the AB). Furthermore, Vogel et al. (1998) found no effects of AB on the visual P1 component, also suggesting a locus of interference somewhere after early sensory encoding and at or before encoding into working memory. In the present study we will examine effects of masking to determine if components after the P1 but before the P3b might be affected during the AB. Finding such effects would suggest a locus of interference prior to the one reflected by the P3b (in addition to an effect on the P3b), while finding no other effects on earlier ERP components, would provide empirical support that the principal locus of AB interference would be at encoding in working memory.

One component worthy of examination in this context is the P3a. This more anterior component, preceding the P3b, is hypothesized to reflect stimulus-driven frontal attentional engagement on targets (Polich, 2007). There are several models of AB interference but they do not all agree on the role attentional engagement plays in the deficit. Chun and Potter's (1995) two-stage model, for example, proposes that only one target at a time can be consolidated in memory. Any subsequent target therefore has to wait until the first target is fully encoded before having access to the encoding stage. Meanwhile, if a second target is too close in time to the previous target, the perceptual trace of the second one fades (or is overwritten) before it can be encoded. It is not clear, in this model, what role attention might play. Dell'Acqua et al. (2015) however, did find a lag effect on P3a amplitude to T2 when T2 was not masked, suggesting a link between attentional engagement and the AB deficit. Consistent with this finding, many models put attentional mechanisms at the forefront of their model. For example, the episodic simultaneous type, serial token model (Wyble, Bowman, & Nieuwenstein, 2009) holds that activation of a target is enhanced by attentional mechanisms. Accordingly, the attentional blink deficit takes place because attention inhibits trailing distractors during the encoding of the first target. If the second target is displayed during this temporary inhibition, its activation cannot be enhanced and eventually encoded in working memory. Finally, some models describe the AB phenomenon as a mix of attention related and non-related disruptions (e.g., the corollary discharge of attention movement, CODAM model; by Fragopanagos, Kockelkoren, & Taylor, 2005). Models including attention-related explanations do not all agree on the cause of the attentional disruption, however, a subset include distractor or masking related impacts on attention. As for models of masking on the other hand, none appear to address possible links between the impacts of a mask on attention. One exception to this general statement pertains to the role of attention in the 4-dot masking phenomenon were this type of mask is more effective if attention is distributed among several targets (Dell'Acqua, Pascali, Jolicœur, & Sessa, 2003; Enns & Di Lollo, 1997). Note however that the link in this case may be more related to spatial attention influencing masking than masking influencing attention. It is therefore difficult to make clear, model based, predictions concerning the impact of masking the last target on attentional engagement. Our experiment will therefore clarify if such a link could explain part of the AB deficit and if it should be incorporated in models of the AB, models of masking, and models of cognition in which consciousness is considered.

Additional information might be uncovered by studying lateralized activity such as the N2pc component in the context of the AB and masking. The N2pc is a negative-going deflection contralateral to the visual field of the attended stimulus at posterior electrode sites (relative to the ipsilateral side), often from about 180 to 280 ms post-stimulus, under conditions of efficient visual search (Eimer, 1996; Kiss, Van Velzen, & Eimer, 2008; Luck & Hillyard, 1994). The N2pc can be used as a way to monitor the deployment of visual spatial attention (Woodman & Luck, 2003). This component has been shown to have a smaller amplitude at short relative to long lags during the AB (Akyürek, Leszczynski, & Schubö, 2010; Dell'Acqua, Sessa, Jolicœur, & Robitaille, 2006; Jolicœur, Sessa, Dell'Acqua, & Robitaille, 2006). Previous experiments examining the N2pc in AB paradigms, however, all contained a post-T2 mask. In tasks other than the AB, masked targets seemed to elicit an N2pc. This was the case for four-dot masking (Prime, Pluchino, Eimer, Dell'acqua, & Jolicœur, 2011; Woodman & Luck, 2003), metacontrast masking under certain conditions (Ansorge & Heumann, 2006; Ansorge, Horstmann, & Worschech, 2010; Jaskowski, van der Lubbe, Schlotterbeck, & Verleger, 2002), and for pattern masking (Robitaille & Jolicœur, 2006). Again, however, these experiments did not directly compare masking to conditions where no mask was present. Only Robitaille and Jolicœur (2006) compared trials where a pattern mask (another alphanumeric character) was presented to when no mask was presented and found no masking effect on the N2pc. Despite that, a sizable number of masking theories exist but little place is given to attention in these proposed mechanisms. The re-entrant perceptual hypothesis (Enns & Di Lollo, 2000) suggests that less attention will accentuate masking but does not broach the impact of the mask itself could have on attention.

To gather additional information on masking effects, we also tested the possibility that masking has a larger effect with increasing difficulty. To do this, we modulated the magnitude of the AB by manipulating the number of targets preceding the last one. Two consecutive targets in AB paradigms are usually both well reported (lag-one sparing; Potter, Chun, Banks, & Muckenhoupt, 1998) while making it harder to report a third target separated from the others by a few distractors (Dux, Wyble, Jolicœur, & Dell'Acqua, 2014). Fig. 1 illustrates an example of the stimuli and sequence of events on each trial. It is important to note that in our experiment, T2 did not always refer to the last target as it typically does in AB paradigms. The trials had three critical stimulus positions, S1, S2, which were contiguous, and S3 at lag 3 or 8 from S2. S2 was always a target (T2). S1 could be a distractor (D1) or a target (T1). And, S3 could be a distractor (D3) or a target (T3). For each level of T2-S3 lag, task difficulty was manipulated by including a target to be reported in the S1 position (T1), which created a higher processing load, or a distractor (D1), which created a lower processing load. We will refer to this manipulation as T1-absent versus T1-present trials.

In summary, we studied the impact of masking on working memory encoding, engagement of attention, and spatial attentional deployment on a target during the AB. To do so, we manipulated the lag (lag 3 or lag 8) between the S2 and S3 positions. We also manipulated the RSVP stream structure by changing the number of targets that were presented prior to S3 (i.e., T1-absent or T1-present) in order to control the difficulty of the task. Importantly, we presented the stimuli in a sequence with a mask after the last target for some participants and without one for others (between-subject design). We predicted that encoding in working memory would be least effective (i.e., smaller P3b amplitude) for trials in which the lag was short, S1 was a target (T1-present), and a mask followed S3 (mask-present). We also expected a similar impact on the onset of memory encoding, that is a delayed P3b onset for short lags, more previous targets, and in the presence of a mask. We also expected short lag trials and trials with more targets (three targets vs. two targets) to reduce the effectiveness of engagement of attention (i.e., smaller P3a amplitude) while not knowing what impact masking would have on P3a. Finally, there should be a lag effect for the deployment of attention, reducing its efficiency for short lags (smaller N2pc amplitude); and the experiment allowed us to examine potential effects of trial type (T1-absent vs. T1-present) and masking, which have not been studied previously.

2. Methods

2.1. Participants

The participants were eighty-five undergraduate students at Université de Montréal. Fifty-one were originally assigned to the no-mask group but eleven were excluded from analysis for various technical reasons (see the Electrophysiological Recording and Data Analysis section for details). Forty participants (30 females) between the ages of 19 and 33 (m = 21.71) were therefore kept for further analysis. Thirty-nine participants were assigned to the mask-present group. Thirty-four participants were kept for analyses (see the Electrophysiological Recording and Data Analysis section for details) (25 females) and they were between the ages of 18 and 26 (m = 21.45). All reported normal or corrected to normal vision, no history of neurological disorders, and all showed normal performance on the Ishihara color test. They received 20 $Can for their voluntary participation in the study after providing written

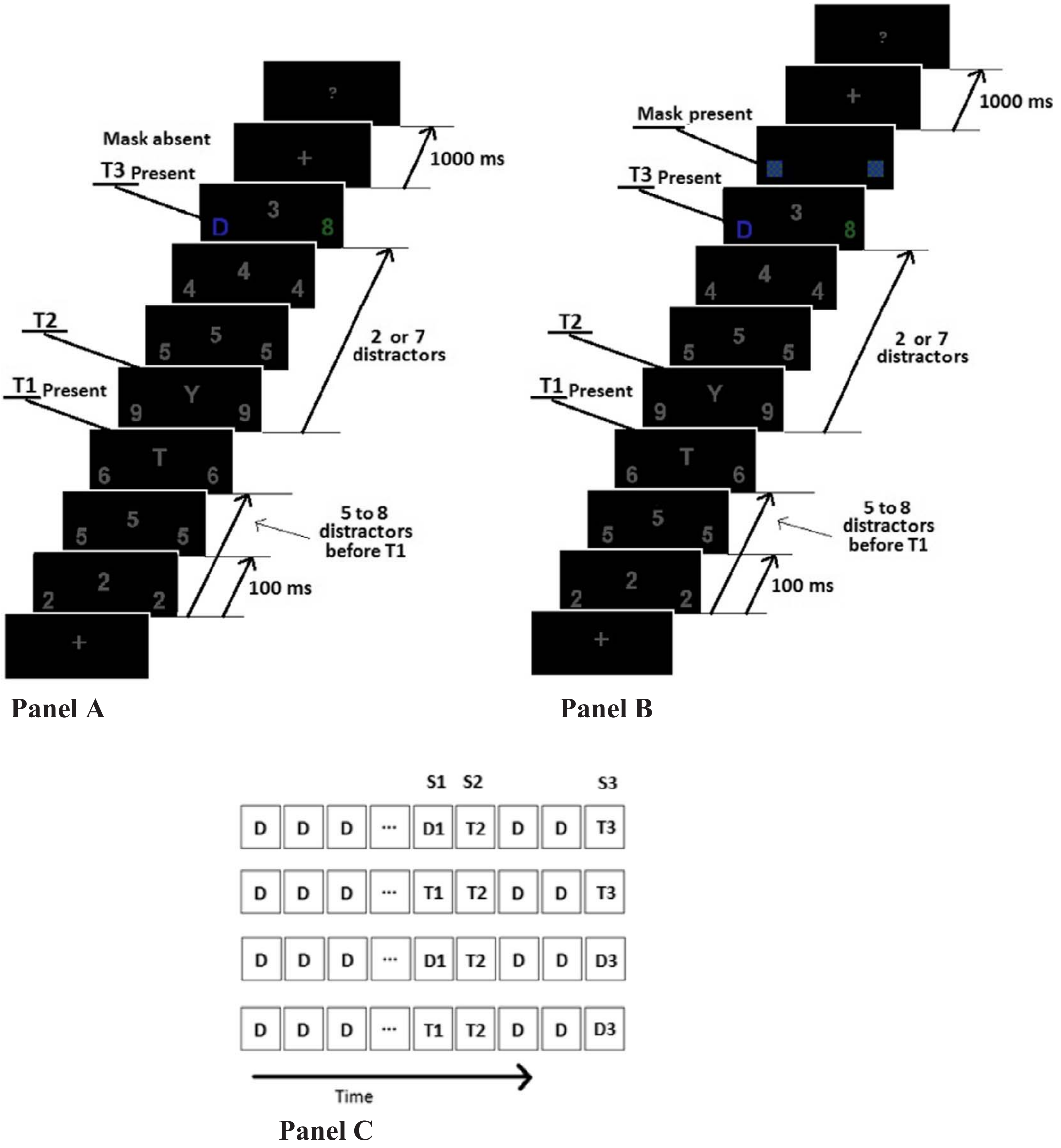

Fig. 1. RSVP structure. Stimulus sequence in each trial. Panel A: mask-absent. Panel B: mask-present. Panel C: RSVP structure possibilities with reference to the targets (shown here only for lag 3 and mask-absent conditions). In this notation, the number indicates the stimulus position in the overall sequence, not the position relative to presented targets. T2 for example is the target presented at S2 position but can be the second target or the first, depending on whether T1 was shown or not shown. In this experiment, T2 was always shown, whereas T1 and/or T3 were sometimes replaced by a distractor.

informed consent.

2.2. Stimuli

An example of the stimuli and sequence of events on each trial is illustrated in Fig. 1. Filler frames in the RSVP sequence consisted of 3 identical digit distractors (between 2 and 9) in a triple RSVP stream. Trials could contain one, two or three targets that consisted of uppercase letters from the English alphabet (excluding B, I, O, Z, and Q, to avoid confusion with digits). The characters were light gray in Courier New font on a black background. Stimuli were 1° of visual angle in height, at a distance of 57 cm from the screen of a cathode-ray tube computer monitor (maintained using a chin rest). In each frame, one stimulus was at fixation whereas the other two were displaced to the left or right by 3° and down by 1° of visual angle. Frames were presented for 100 ms with no inter-stimulus interval. There were three frames in which the digit distractor *could* be replaced by a letter target; in the S1, S2, or S3 position. The second position, S2, always contained a target (T2) while both the S1 and S3 positions could randomly contain a target (T) or a distractor (D) (i.e., DTD, DTT, TTD or TTT). The target at the S1 position (T1, if present) as well as the target at the S2 position (T2) were always displayed at fixation while the target at the S3 position (T3, if present) was to the left or right of fixation.

S1 was randomly in the 6th, 7th, 8th, or 9th frame. S2 position was in the frame immediately following the S1 position (frame S1 + 1). Finally, in order to elicit a deployment of attention that could be tracked by monitoring N2pc, the S3 position was in the right or left visual field of the final frame; 3 or 8 frames following S2 position (S2 + 3 or S2 + 8). For participants in the masking group, the S3 frame was followed by a five by five blue and green checkerboard of 1° of visual angle. Digits and letters were semi-randomly assigned during the sequence with no two same digits repeated from one frame to the next. Targets for a particular trial were always different letters from each other. For the last frame, participants were instructed to pay attention to a target color (blue or green; counterbalanced). S3, whether a distractor or target, was in the target color (e.g., blue[1]) while the middle digit was light gray and the digit on the other side was in the distractor color (e.g., green). Thus, the last frame containing characters contained S3, in the target color, and could be a target (T3) if it was a letter, or a distractor (D3) if it was a digit. The stimulus on the other side of fixation was always a distractor (digit). This manipulation allowed participants to know when and where to deploy attention when the last frame consisted only of distractors. The luminance of the three colours used (blue, green, and light gray) was adjusted to be approximately equiluminant using a Minolta CS100 chromameter (3.30 cd/m^2).

2.3. *Procedure*

Participants were seated in a dimly-lit electrically-shielded room and initiated each trial by pressing the space bar. A jittered delay of approximately 600 ms preceded the onset of the RSVP sequence. The task was to maintain fixation at the center of the screen and to encode letters presented at that location, and to encode a blue (for half of the subjects, green for the other half) letter in the final frame, for participants in the no-mask group, and in the frame just before the mask for those in the mask group. After the end of the sequence, they were to maintain fixation on a fixation cross for another 1 s, after which they entered all the letter(s) they saw on a standard keyboard after the fixation cross disappeared and a question mark appeared. The delay between the end of the RSVP sequence and the response period ensured that muscle activity and ocular artifacts from eye movements towards the keyboard did not overlap with ERPs of interest.

The experiment included 672 experimental trials divided into 21 blocks of 32 trials each, preceded by a practice block of 16 trials. There were four within-subject experimental variables (factors) each with two levels, lag between T2 and T3 (3 or 8), presence or absence of T1, presence or absence of T3, and side-of-presentation of T3 (left or right visual field), yielding 16 combinations. And, there was one between-subjects factor: presence or absence of a mask after the S3 frame position (Fig. 1). An equal number of trials (namely 2) in each of the 16 cells of the design were randomly mixed within each block.

2.4. *Electrophysiological Recording and data analysis*

EEG was recorded using a BioSemi Active Two system and an elastic cap with 64 Ag/Ag–Cl electrodes positioned according to the International 10/10 system. The sampling rate was 512 Hz and the signal was referenced to the average of left and right mastoids after the recording. A high-pass filter of 0.1 Hz and a low-pass filter of 30 Hz were applied offline. The horizontal electro-oculogram (HEOG) was obtained using the subtraction of activity from a pair of electrodes situated on the left and right eye outer canthi, which we used to monitor eye movement. Vertical eye movements and blinks (VEOG) were measured by subtracting data of an electrode situated below the left eye from the data above the left eye (Fp1). VEOG and HEOG channels were filtered with a 10 Hz low-pass filter and a 0.1 Hz high-pass filter to facilitate trial-by-trial ocular artifact rejection. An Independent Component Analysis (ICA) was performed to remove blink artifacts. Components related to ocular artifacts were selected by comparing the components identified by the ICA to the EOG signal waveforms and by examining the topography and time course of the components using the method described by Drisdelle, Aubin, and Jolicœur (2017). Any remaining fluctuation of > 50 μV over a 150 ms period of the VEOG signal was labeled as a blink that was missed by the ICA procedure (although it could simply be noise from another artifact). Trials containing such fluctuations were removed from the data set. Similarly, segments with an HEOG difference of > 35 μV over a 300 ms period were considered eye movements and were removed. Data from any channel exceeding ± 100 μV during a trial segment was interpolated, up to a maximum of 7 channels in any given trial. Trials with > 7 channels exceeding this range were rejected. Participants that had > 40% of trials rejected based on these criteria were excluded from further analysis. This resulted in the exclusion of data from 11 participants in the mask-absent group and 2 participants in the mask-present group. Additionally, three participants in the mask-present group were rejected because they correctly reported T3 in < 2% of trials were it was present no matter the lag. The other participants correctly reported T3 67% of the time. For these participants, an average of 7.65% of trials were rejected. EEG was segmented based on the onset of S3, with a 100 ms pre-stimulus baseline and a 1000 ms post-stimulus-onset period.

In order to isolate T3-locked P3a and P3b from the overlapping activity caused by the previous stimuli in the RSVP stream, we used trials where S3 was a distractor (D3). By subtracting target absent (D3) from target present conditions (T3), it was possible to isolate the P3a and P3b activity related to the processing of T3. This technique (or close variant) was used in previous related work (e.g., Ptito et al., 2008; Vogel & Luck, 2002). The P3b was measured at electrode POz and the P3a was measured at Fz. The S3-locked N2pc was measured at PO7/PO8 (where N2pc activity usually reaches its peak), and computed by subtracting electrical activity measured at an electrode ipsilateral to the attended stimulus from activity measured at a corresponding contralateral electrode. Only the last target was in a lateral location on the screen, ensuring that the stimulus of interest (last target) was the only one eliciting the N2pc component.

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[1] For interpretation of color in Fig. 1, the reader is referred to the web version of this article.

Table 1
Accuracy. Mean probability of correct report of each target in T1-absent and T1-present trials as a function of lag between S2 and S3 (3 vs. 8), and mask presence (trials were a mask was present vs. trials were the mask was absent). Accuracy was conditional on correct report of the preceding target(s).

	Stimulus position		
	S1	S2	S3
For T3-absent			
Mask absent, lag 3			
T1-absent	–	$M = 0.96$, $SD = 0.06$	–
T1-present	$M = 0.84$, $SD = 0.11$	$M = 0.91$, $SD = 0.09$	–
Mask absent, lag 8			
T1-absent	–	$M = 0.96$, $SD = 0.05$	–
T1-present	$M = 0.84$, $SD = 0.09$	$M = 0.91$, $SD = 0.08$	–
Mask present, lag 3			
T1-absent	–	$M = 0.97$, $SD = 0.03$	–
T1-present	$M = 0.85$, $SD = 0.08$	$M = 0.88$, $SD = 0.12$	–
Mask present, lag 8			
T1-absent	–	$M = 0.96$, $SD = 0.03$	–
T1-present	$M = 0.84$, $SD = 0.09$	$M = 0.90$, $SD = 0.10$	–
For T3-present			
Mask absent, lag 3			
T1-absent	–	$M = 0.96$, $SD = 0.05$	$M = 0.97$, $SD = 0.03$
T1-present	$M = 0.82$, $SD = 0.12$	$M = 0.88$, $SD = 0.11$	$M = 0.93$, $SD = 0.07$
Mask absent, lag 8			
T1-absent	–	$M = 0.96$, $SD = 0.06$	$M = 0.97$, $SD = 0.03$
T1-present	$M = 0.83$, $SD = 0.10$	$M = 0.90$, $SD = 0.09$	$M = 0.96$, $SD = 0.03$
Mask present, lag 3			
T1-absent	–	$M = 0.97$, $SD = 0.03$	$M = 0.66$, $SD = 0.26$
T1-present	$M = 0.84$, $SD = 0.09$	$M = 0.88$, $SD = 0.11$	$M = 0.48$, $SD = 0.23$
Mask present, lag 8			
T1-absent	–	$M = 0.96$, $SD = 0.03$	$M = 0.78$, $SD = 0.19$
T1-present	$M = 0.84$, $SD = 0.08$	$M = 0.87$, $SD = 0.12$	$M = 0.75$, $SD = 0.20$

3. Results

3.1. Behavioral

Because we were interested in the AB, only T3-present trials (last target) were considered for analyses and the mean proportion of correct report for each target was contingent on the correct report of preceding targets (see Table 1). ANOVAs were performed on the mean proportion of correct report for each target as a function of the trial type (T1-absent vs. T1-present) and lag (3 vs. 8) as within-subject factors and presence of the mask (mask-present trials vs. mask-absent trials) as a between-subject factor.

On average, subjects were more accurate in reporting T2 in T1-absent trials (T2-T3) than in reporting T1 in T1-present trials (T1-T2-T3), $F(1,72) = 252.64$, $MSE = 0.005$, $p < 0.0001$. That is, report of the first target in the stream was better when there were only two targets shown overall (T2-T3) than when there were three (T1-T2-T3). There was no significant difference between lags, however, $F(1,72) = 0.14$, $MSE = 0.0001$, $p = 0.709$, or between mask-present and mask-absent trials $F(1,72) = 0.40$, $MSE = 0.017$, $p = 0.531$, on accuracy of report of the first target in the stream (T1, in T1-present trials; T2 in T1-absent trials).

An ANOVA was carried out to compare the mean proportion of correct report for T3, that is, when S3 was a target, as a function of lag, mask presence, and trial type (T1-presence/absence) (conditional on correct report of the preceding target(s)). T3 accuracy was lower at lag 3 than at lag 8, $F(1,72) = 113.46$, $MSE = 0.007$, $p < 0.0001$. T3 accuracy was lower for T1-present trials than for T1-absent trials, $F(1,72) = 108.18$, $MSE = 0.002$, $p < 0.0001$. T3 accuracy was lower when T3 was followed by a mask compared with the no-mask condition, $F(1,72) = 74.99$, $MSE = 0.083$, $p < 0.0001$. There was also a significant three-way interaction between these factors, $F(1,72) = 30.21$, $MSE = 0.002$, $p < 0.0001$. T-tests with a Bonferroni-corrected alphas of 0.01 per test (0.05/5) indicated that there was no lag effect for T1-absent trials when no mask was present, $t(39) = 0.73$, $MSE = 0.004$, $p = 0.471$, while reliable lag effects were found for T1-absent trials when a mask was present, $t(33) = 5.89$, $MSE = 0.021$, $p < 0.0001$. Lag effects, though of different magnitude, were detected both when the mask was present, $t(33) = 10.99$, $MSE = 0.024$, $p < 0.0001$, and when the mask was absent, $t(33) = 3.01$, $MSE = 0.010$, $p = 0.005$. Results indicated that, in the absence of a mask, an AB effect was only found for T1-present trials, converging with prior studies (Dell'Acqua et al., 2015; Giesbrecht & Di Lollo, 1998; Jannati, Spalek, & Di Lollo, 2010, 2012; Sessa et al., 2007). As expected, in mask-present trials the AB deficit (lower accuracy for lag 3 trials vs. lag 8) was more pronounced when more targets were presented (T1-present vs. T1-absent) $t(33) = 7.37$, $MSE = 0.0193$, $p < 0.001$.

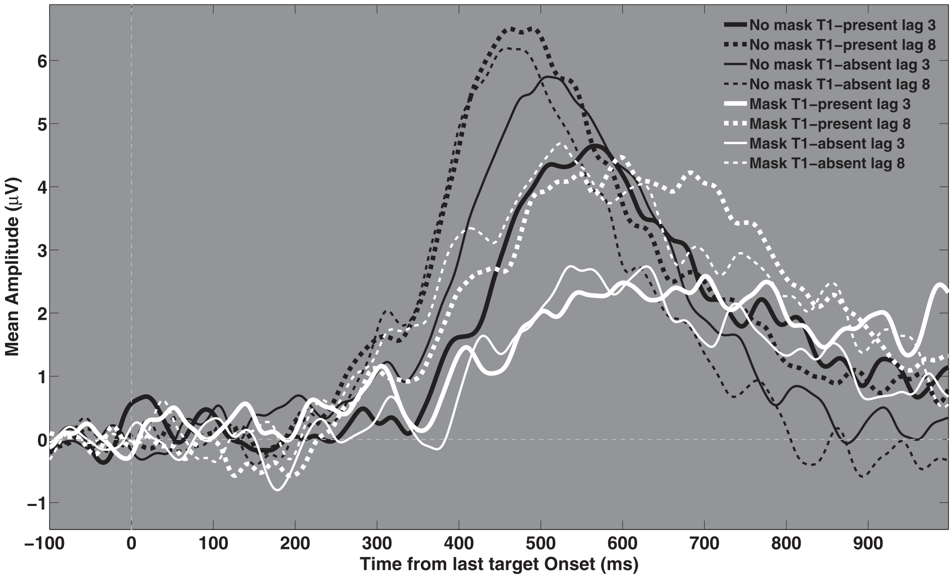

Fig. 2. P3b waveforms. Grand average ERP difference waves (T3-present minus T3-absent trials) showing the P3b component at POz electrode site for lags 3 and 8, T1-absent and T1-present trials, for the mask-absent group and the mask-present group.

3.2. *Electrophysiological results*

3.2.1. *P3b*

Fig. 2 shows the grand average P3b difference waves (T3-present minus T3-absent) for the various trial types at POz, while Table 2 lists the mean amplitudes of these waveforms. Amplitudes were obtained by measuring the mean amplitude in a 150 ms window (± 75 ms) centered on the peak of the waveform in mean grand averages for each condition. Peak amplitudes were reached at the following time points for mask-absent trials: conditions: T1-absent lag 3 = 507 ms, T-absent lag 8 = 457 ms, T1-present lag 3 = 566 ms and T1-present lag 8 = 490 ms. For mask-present trials, mean peak amplitudes were reached at the following time points: T1-absent lag 3 = 537 ms, T-absent lag 8 = 523 ms, T1-present lag 3 = 701 ms and T1-present lag 8 = 597 ms. The amplitudes for each subject were submitted to an ANOVA that considered lag (3 vs. 8) and trial type (T1-absent trials vs. T1-present trials) as within-subject factors, and mask presence (mask-present vs. mask-absent trials) as a between-subject factor. The mean amplitude of the P3b component was significantly smaller for Lag 3 than Lag 8, $F(1,72) = 33.98$, $MSE = 4.361$, $p < 0.0001$. The P3b had a smaller amplitude for mask-present then mask-absent trials, $F(1,72) = 18.67$, $MSE = 16.163$, $p < 0.0001$. There was however no significant amplitude difference between T1-absent and T1-present trials, $F(1,72) = 0.42$, $MSE = 3.148$, $p = 0.52$. There were no significant interaction in the analysis, all *ps* > 0.09.

Mean P3b latencies, estimated using a jackknife approach (Kiesel, Miller, Jolicœur, & Brisson, 2008; Ulrich & Miller, 2001) with individually derived values using the solution proposed by Brisson and Jolicoeur (2008) and Smulders (2010), were also compared (see Table 3). Latency values were calculated as the time-point when individual jackknife waveforms reached 50% of the area under the curve (for values above 0 µV) in a 190–990 ms window post S3. The same ANOVA model as described above was used. P3b latency was significantly delayed for lag 3 trials compared to lag 8 trials, $F(1,72) = 47.928$, $MSE = 4089.51$, $p < 0.0001$. P3b latency was also longer for T1-present trials compared to T1-absent trials, $F(1,72) = 28.35$, $MSE = 5226.7$, $p < 0.0001$. Furthermore, P3b latency was longer for mask-present trials compared to mask-absent trials, $F(1,72) = 29.39$, $MSE = 22280.306$, $p < 0.0001$. There were no significant interactions between these factors, all *ps* > 0.2. Collectively, the P3b results converge nicely with prior studies (Dell'Acqua et al., 2015) and indicate that encoding T3 in working memory is less efficient and delayed in the presence of a mask.

Table 2
P3b amplitude. Mean P3b amplitudes (µV) (T3-present minus T3-absent trials) for lags 3 and 8, T1-absent and T1-present trials, and for mask-absent and mask-present.

Mask presence	Trial type	Lag 3	Lag 8
Absent	T1-absent	*M* = 5.20, *SD* = 2,62	*M* = 5.54, *SD* = 2.88
Absent	T1-present	*M* = 4.21, *SD* = 2.36	*M* = 5.96, *SD* = 2.66
Present	T1-absent	*M* = 2.31, *SD* = 2.38	*M* = 4.08, *SD* = 2.62
Present	T1-present	*M* = 2.31, *SD* = 2.19	*M* = 4.12, *SD* = 2.99

Table 3

P3b latency. Mean latency (in ms) of the P3b difference waves (T3-present minus T3-absent trials) estimated from jackknife averages using the fractional-area latency method (latency of 50% of the area under the curve; see text for further details), for lags 3 and 8, T1-absent and T1-present trials, for mask-absent and mask-present trials.

Mask presence	Trial type	Lag 3	Lag 8
Absent	T1-absent	$M = 531$, $SD = 86$	$M = 485$, $SD = 61$
Absent	T1-present	$M = 595$, $SD = 114$	$M = 521$, $SD = 93$
Present	T1-absent	$M = 629$, $SD = 105$	$M = 586$, $SD = 97$
Present	T1-present	$\mathrm{M} = 669$, $SD = 111$	$M = 626$, $SD = 81$

3.2.2. The P3a

Fig. 3 shows the grand average P3a difference waves (T3-present minus T3-absent) at Fz. Mean amplitudes estimated using a window between 270 and 320 ms are listed in Table 4. The amplitude was larger for lag 8 trials compared to lag 3 trials, $F(1,72) = 7.65$, $MSE = 3.212$, $p = 0.007$, the difference between T1-absent and T1-present trials did not quite reach significance, $F(1,72) = 2.66$, $MSE = 2.368$, $p = 0.108$. There was a marginally significant crossover interaction between lag and trial type, $F(1,72) = 3.61$, $MSE = 1.916$, $p = 0.061$. For lag 3, more targets (T1-present) meant a smaller amplitude whereas the opposite was found for lag 8. Finally, P3a amplitude was larger for mask-absent trials than for mask-present trials, $F(1,72) = 7.11$, $MSE = 4.016$, $p = 0.009$. There were no other significant effects in the analysis, all $ps > 0.102$. Results indicate that masking makes engagement of attention less efficient during the AB.

3.2.3. N2pc

Fig. 4 displays the grand average contralateral minus ipsilateral waveforms for T3-present trials for T1-absent and T1-present trials, for each lag, and each group (mask-absent/mask-present). N2pc onset was about the same for the two masking conditions, but the peak amplitude and duration of N2pc was clearly different across masking conditions. For this reason, the window used to estimate the mean amplitude of N2pc was different for the two groups (200–300 ms for the mask-absent group, and 170–270 ms for the mask-present group; which was a 100 ms window around the approximate peak in the grand average difference waves). The mean amplitudes for each participant for each condition were submitted to an ANOVA with the same model as for the P3 analyses. The overall means are shown in Table 5. However, prior to the main analyses, we verified that the target color manipulation (recall that green was the target color for half of the participants and blue for the other half). We first analysed the data with an additional between-subjects factor of target color. There was a global effect of target color on N2pc amplitude but it did not interact with any of the other conditions of interest (lag, presence/absence of T1 or mask). We therefore did not look further into this effect.

Although we found an N2pc for T3-absent trials, given the presence of the selection in the S3 frame, preliminary analyses showed that N2pc was much smaller for T3-absent trials than for T3-present trials, presumably because attention could be quickly disengaged when S3 contained a distractor (T3-absent) rather than a target (T3-present). The much-reduced N2pc for T3-absent trials made it difficult to examine differences in N2pc as a function of other task variables. For that reason, we focused the following analyses on T3-present trials. N2pc amplitude did not vary significantly between lags, $F(1,72) = 1.78$, $MSE = 3.192$, $p = 0.186$, or between T1-

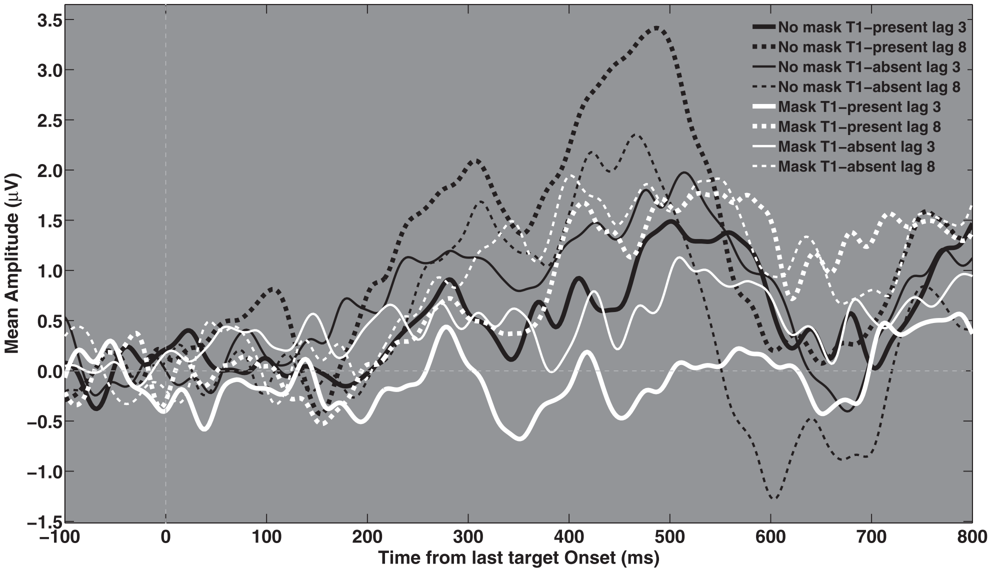

Fig. 3. P3a waveforms Grand average ERP difference waves (T3-present minus T3-absent trials) showing the P3a component at Fz electrode site for lags 3 and 8, T1-absent and T1-present trials, for the mask-absent group and the mask-present group.

Table 4

P3a amplitude. Mean P3a amplitudes (μV) (T3-present minus T3-absent trials) for lags 3 and 8, T1-absent and T1-present trials, and for mask-absent and mask-present.

Mask presence	Trial Type	Lag 3	Lag 8
Absent	T1-absent	$M = 1.02$, $SD = 1.83$	$M = 1.11$, $SD = 1.48$
Absent	T1-present	$M = 0.49$, $SD = 1.43$	$M = 1.65$, $SD = 2.10$
Present	T1-absent	$M = 0.51$, $SD = 1.71$	$M = 0.97$, $SD = 1.57$
Present	T1-present	$M = -0.16$, $SD = 1.47$	$M = 0.46$, $SD = 1.84$

absent versus T1-present trials, $F(1,72) = 1.72$, $MSE = 2.031$, $p = 0.193$. However, as is apparent upon inspection of Fig. 4, N2pc amplitude was larger for mask-absent relative to mask-present trials, $F(1,72) = 15.28$, $MSE = 5.671$, $p < 0.0001$. The analysis of N2pc amplitudes did not detect other significant effects, all $ps > 0.18$.

Visual inspection of Fig. 4 revealed marked differences in the offset of N2pc for mask-present and mask-absent trials. An ANOVA on jackknife latency estimates (see Table 6) confirmed this, $F(1,72) = 14.81$, $MSE = 18697.622$, $p < 0.0001$. However, N2pc offset latency did not vary between lags, $F(1,72) = 0.001$, $MSE = 6426.808$, $p = 0.975$, nor across T1-absent vs. T1-present trials, $F(1,72) = 0.14$, $MSE = 7288.793$, $p = 0.709$. There were no significant interactions between these factors, all $ps > 0.20$.

4. Discussion

We investigated the role of masking in the spatial deployment of attention, attentional engagement, and encoding of representations in working memory during the AB. To do so, we presented targets (letters) or distractors (digits) in the context of three concurrent RSVP streams consisting mainly of distractors (digits). There were three critical temporal positions in the sequences, S1, S2, and S3. S1 and S2 were always consecutive in the sequence, whereas the lag between S2 and S3 was either 3 or 8. S2 was always a target (T2), but S1 could be a target (T1) or a distractor (D1), and S3 could be a target (T3) or a distractor (D3). In one group of participants, S3 was the last stimulus (not masked), whereas in another group, S3 was followed by a mask. Together, this preparation produced a rich set of experimental conditions enabling an examination of the impact of masking and processing load on the AB, both on behavior and on event-related potentials isolated from concurrent measurements of the EEG during the task.

The processing load prior to the presentation of S3 was varied by presenting one or two previous targets (T2, or T1 + T2). Behaviorally, when the last target was masked, an AB effect (i.e., a reduction in the accuracy of report of T3 at lag 3 vs. lag 8) observed both in T1-absent and T1-present trials and this effect was larger for T1-present trials. When T3 was not masked, T1-absent trials no longer showed a behavioral AB effect while traces of an AB effect were still detected in 3T trials. The absence of an observable behavioral AB effect for mask-absent trials might be a consequence of the high accuracies, which might have led to a ceiling effect. It is therefore difficult to interpret the absence of AB deficit under such conditions. An experiment by Nieuwenstein et al. (2009) demonstrated that an AB deficit can be generated even when distractors are replaced by blank intervals. This suggests that masking might not be necessary in order to observe AB deficits. Difficulty of encoding was successfully manipulated as accuracy was overall lower when there were two targets before T3, as opposed to one target before T3. These results replicated what

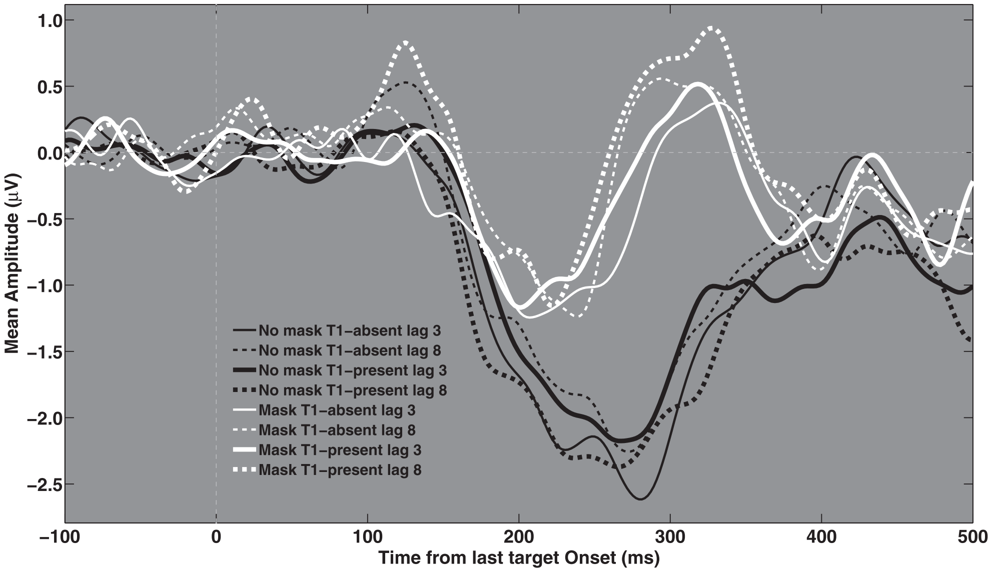

Fig. 4. N2pc waveforms. Grand average ERP difference waves (contralateral minus ipsilateral) showing the N2pc component at PO7/PO8 electrode sites for lags 3 and 8, T1-absent and T1-present trials, for the mask-absent group and the mask-present group (for T3-present trials only).

Table 5

N2pc amplitude. Mean N2pc lateralized amplitude (μV) (contralateral minus ipsilateral) for lags 3 and 8, T1-absent and T1-present trials, as well as for trials with a mask compared to trials without a mask.

Mask presence	Trial type	Lag 3	Lag 8
Absent	T1-absent	$M = -2.23$, $SD = 1.80$	$M = -1.88$, $SD = 1.82$
Absent	T1-present	$M = -1.94$, $SD = 1.67$	$M = -1.58$, $SD = 2.64$
Present	T1-absent	$M = -1.01$, $SD = 1.47$	$M = -0.77$, $SD = 1.67$
Present	T1-present	$M = -0.83$, $SD = 1.31$	$M = -0.66$, $SD = 2.22$

Table 6

N2pc latency. Mean N2pc latency (ms) (contralateral minus ipsilateral) for lags 3 and 8, T1-absent and T1-present trials, as well as for trials with a mask or trials without a mask.

Mask presence	Trial type	Lag 3	Lag 8
Absent	T1-absent	$M = 307$, $SD = 45.63$	$M = 304$, $SD = 79.35$
Absent	T1-present	$M = 305$, $SD = 0.63.39$	$M = 325$, $SD = 96.05$
Present	T1-absent	$M = 263$, $SD = 195.09$	$M = 252$, $SD = 35.29$
Present	T1-present	$M = 243$, $SD = 0.139.33$	$M = 238$, $SD = 59.93$

Dell'Acqua et al. (2015) found in a similar experiment. Importantly, accuracy was significantly impacted by the presence of a backward mask, making it harder for participants to report targets when a mask followed the last target, and masking magnified the AB effect, as expected from previous research.

The electrophysiological measures showed a smaller P3b amplitude for short lag trials as was previously reported in other studies (Dell'Acqua et al., 2015; Vogel & Luck, 2002; Vogel et al., 1998). This component was however not completely suppressed when the mask was present as was reported by Vogel and Luck (2002). This allowed us to observe a lag effect on latency of P3b in both the mask-absent and mask-present conditions (Dell'Acqua et al., 2015; Vogel & Luck, 2002).

The onset of the P3b was later at lag 3 compared with lag 8. As Dell'Acqua et al. (2015) reported, increasing the number of initial targets (T1-present compared with T1-absent) also delayed the P3b. These finding complement previous research suggesting that the P3b reflects processing in a capacity-limited mechanism, which is either delayed, and/or slowed, under some conditions. Importantly for this paper, using a backward mask, we observed similar patterns as in Vogel and Luck's (2002) experiment, namely a significant attenuation of the P3b in the short lag condition. It is possible that our attenuation was not as pronounced, as the one found by Vogel and Luck (2002) because we used a mask consisting of a small checkerboard, which perhaps approached masking by noise rather than by pattern. This result, however, suggests that the type of backward mask (at least between pattern and noise masks, at the target-mask SOA that was used) may not affect encoding in working memory differently, both of then making memory encoding a less efficient process. The backward mask delayed the P3b by 70–100 ms (depending on experimental conditions). It is very likely that this effect could be observed for other types of masks, as long as the strength of masking did not completely suppress the P3b, as occurred in the Vogel and Luck (2002) study. The present findings help to clarify the role of masking in the AB paradigm and refine our understanding of capacity limitations underlying the AB. Masking causes a delay of the P3b, suggesting that processing masked targets is less efficient than processing targets that are not masked. Processing a masked stimulus, under AB load, is slowed, or perhaps even postponed, which can be observed in delayed latency of relevant ERP components, as well as increased response times when the paradigm involves speeded responses (e.g., Jolicoeur & Dell'Acqua, 1998; Jolicœur, 1998, 1999a).

We also studied the impact of masking on the engagement of selective attention in the AB. We successfully replicated Dell'Acqua et al.s' (2015) lag effect on the P3a as it had larger amplitudes for longer lag trials, indicating that attentional engagement was implicated in the AB deficit. Importantly, both for mask-present and mask-absent trials, lag effects on the P3a amplitude to T3 were more pronounced when two leading targets had to be encoded (T1-present, T2-present) compared to one (T1-absent, T2-present). When two leading targets (T1-present, T2-present) were presented in masked trials, there was a particularly large lag effect leading to a complete suppression of the P3a at lag 3. Total encoding load, therefore, does affect selective engagement of attention on a subsequent target (here, T3) by exacerbating the lag effects during the AB. Of particular interest was the significant effect of the presence of the mask in suppressing the amplitude of the P3a response. The functional meaning behind the P3a component is still a matter of debate but the suppression by masking suggests that the functions reflected by P3a are strongly affected by masking. If, as some suggest, the P3a represents attentional engagement, or selection for engagement, our results would be consistent with models suggesting that AB reflects greater selection difficulty under some conditions. In the episodic simultaneous type, serial token model (Wyble et al., 2009) for example, during encoding of the first target, attention is inhibited so as to avoid distractor interference. This inhibition would impede the selection of the blinked target (see also; Wyble, Potter, Bowman, & Nieuwenstein, 2011).

Finally, we evaluated the effect masking had on the N2pc during the AB. In contrast with several earlier studies, we did not find a lag effect on N2pc amplitude. There was also no effect of the number of leading targets prior to T3 (i.e., no difference in N2pc to T3 across T1-absent and T1-present trials), despite clear effects of this manipulation on accuracy, and on P3b latency as well as a trend, that did not quite reach significance, towards a trial type effect for P3a amplitude, reproducing patterns seen in Dell'Acqua et al.' work (2016). These results were unexpected, and we have no clear-cut explanation for the apparent discrepancies with previous work (e.g., Akyürek et al., 2010; Dell'Acqua et al., 2006; Jolicœur et al., 2006; Pomerleau et al., 2014). Although many aspects of this experiment were similar to previous ones, one difference that was possibly responsible for this is the fact that the present experiment used three RSVP streams in order to mask the last lateralized target. The literature on the AB shows that dividing attention by adding an irrelevant task could reduce the AB (Olivers & Nieuwenhuis, 2005). Perhaps the three RSVP design created a divided attention situation since the side RSVP streams required attention but where irrelevant until the very end. This possibly reduced the AB impact on attentional deployment. Given the speed of presentation of the stimuli in the RSVP streams, it is possible that participants adopted a strategy of initially attempting to encode the stimuli on both side, before zeroing in on the target side (based on the color cue). At the time of publication of the article, we had no fully-convincing explanation for the absence of lag or T1-presence effect on the amplitude of N2pc.

Perhaps the most striking results of the experiment where the impressively large effects of masking on N2pc amplitude and duration. The trailing mask appeared to limit the duration of useful processing of the target, reflected in a shorter N2pc duration and lower overall amplitude, compared to the no-mask condition. The attenuation and early termination of the N2pc is consistent with the hypothesis that masking curtailed the attentional deployment on S3, slowing and attenuating downstream processing (reflected in poor performance and attenuated and delayed P3 responses). Considering this, processing of a masked last target would produce a representation that may be more vulnerable to interference and it is therefore not surprising that T3 accuracy is lower during the AB.

Importantly, although the early attentional component of processing was apparently shortened by the mask, later downstream processing was probably lengthened, assuming the representation of the masked target was not completely obliterated. It is thus interesting to consider our results in relation to the role the distractor following the first target plays in the AB. Raymond et al. (1992) found a larger AB when T1 (they only had two-target trials) was followed by an immediate distractor (T1 + 1), suggesting a lengthening of capacity-demanding processing of T1 (e.g., Jolicœur, 1999b). Brisson et al. (2010) found that masking T1 reduced the amplitude of the P3b, as it did for masking T2 (their last target). According to these authors these findings show that masking T1 could reduce processing efficiency, leading to greater cost for the last target. Perhaps the N2pc and P3a results we obtained by masking our last target would also be similar to that for a masked first target. As seen in the present work, masking the last target had a big impact on its processing but perhaps masking the first target also does and this effect might carry-over to the last target and accentuate downstream masking effects. While some authors give a central role to masking in the AB, some suggest that masking might not be necessary in order to observe AB deficits. This would explain how an AB can be measured when distractors are replaced by blank intervals (Nieuwenstein et al., 2009). It is however undeniable that masking played an important role in behavioral deficits in this experiment. It has been suggested in several studies (e.g., Ouimet & Jolicoeur, 2007; Vogel & Luck, 2002) that the AB is a consequence of limited-capacity of the underlying mechanisms leading to the AB. The impact masking had on various processing mechanisms in the present study suggests that, even if we suppose that it was not a central cause, it nevertheless played an important role in the AB by reducing efficiency of limited-capacity processing mechanism. To summarize, masking affected encoding in working memory (P3b), attentional engagement (P3a), and spatial deployment of attention (N2pc). Masking cut short spatial attention, whereas it lengthened later processing because of degraded representations, particularly under high processing load. These results demonstrate that masking affects numerous aspects of attention and encoding, and they help to clarify and refine our understanding of the impact of masking in the AB paradigm. Importantly, the apparent interruption of visual-spatial attention reflected by the N2pc appears to map directly onto a clear reduction in the probability of the creation of a representation that is accessible to consciousness. The interactions highlighted by the present research show how limited-capacity processing mechanisms at earlier and later stages of processing interact to either enable, or impede, the conscious perception of representations encoded from fleeting visual inputs.

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