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Enhanced frontal activation underlies sparing from the attentional blink: Evidence from human electrophysiology

ROBERTO DELL'ACQUA,[a,b] MATTIA DORO,[b] PAUL E. DUX,[c] TALIA LOSIER,[d] AND PIERRE JOLICŒUR[d]

[a]Cognitive Neuroscience Center, University of Padova, Padova, Italy
[b]Department of Developmental Psychology, University of Padova, Padova, Italy
[c]School of Psychology, The University of Queensland, St. Lucia, Australia
[d]Department of Psychology, Université de Montréal, Montréal, Québec, Canada

Abstract

Using the ERP method, we examined the processing operations elicited by stimuli that appear within the same temporal attention window. Forty subjects searched for letter targets among digit distractors displayed in rapid serial visual presentation (RSVP). ERPs were examined under conditions where a single target was embedded among distractors and compared to those recorded when two consecutive targets were embedded among distractors. Standard and independent component analyses revealed two temporally and topographically distinct ERP responses, a midfrontal P3a component peaking at about 300 ms followed by a midparietal P3b component peaking at about 450 ms. With minimal latency variations, the frontal P3a was amplified when elicited by two consecutive targets relative to a single target. The parietal P3b response was also amplified when elicited by two consecutive targets compared to a single target but, in contrast to P3a, it was also associated with a substantially longer time course. These results provide evidence for the involvement of frontal brain regions in the close-to-concurrent selection of two consecutive targets displayed in RSVP, and of posterior brain regions in the serial encoding of targets in visual working memory. The present findings are discussed in relation to current models of temporal gating of attention and the attentional blink effect.

Descriptors: Attentional blink, Event-related potentials, Frontoparietal neural circuit, P3 component, Independent component analysis (ICA)

Humans do not seem to have any particular difficulty in detecting one specific object embedded in a flow of rapidly changing information (Sperling, Budiansky, Spivak, & Johnson, 1971). This apparent efficiency belies, however, the complexity of the chain of processes required to parse the visual continuum into discrete objects, which is key to generating a stable and coherent mental representation of our dynamic visual environment. This topic has been a matter of intense investigation over the last 25 years using the rapid serial visual presentation (RSVP) paradigm, where two target objects (e.g., Target 1 [T1] and Target 2 [T2]) must be detected among a stream of distracting elements. An RSVP phenomenon that has undergone considerable investigation is the attentional blink (AB; Raymond, Shapiro, & Arnell, 1992): When T1 and T2 are displayed at less than about half a second of each other, T1 is usually detected successfully, whereas T2 is often missed.

Current cognitive-neuroscientific models of the AB share the core assumption that attention is initially deployed to T1 through top-down amplification conveyed through a reentrant frontoparietal neural circuit (e.g., Di Lollo, Enns, & Rensink, 2000; Kranczioch, Debener, Schwarzbach, Goebel, & Engel, 2005; Lamme & Roelfsema, 2000; Marois & Ivanoff, 2005; Marois, Yi, & Chun, 2004; Scalf, Dux, & Marois, 2011). Specifically, attention engagement to T1 is held to be necessary for passing this information on to higher-level encoding stages of processing such as those associated with visual working memory. Reeves and Sperling (1986; see also Nakayama & Mackeben, 1989) have shown that the time course of attention deployment to RSVP targets is well approximated by a gamma function, with a steeply rising rate of information accumulation peaking at about 100–150 ms after target onset, followed by a gradual return to baseline. Given that RSVP items are often presented at rates close to 10 Hz, this implies that attention deployment to RSVP items is likely to be at its peak when the T1 + 1 item is displayed. Indeed, a wealth of evidence shows that if the T1 + 1 item is T2, then it is often spared from the AB, a phenomenon known as lag-1 sparing (Potter, Chun, Banks, & Muckenhoupt, 1998). Two classes of behavioral findings have been taken as further support for this view. When T1 and T2 are consecutive items, (1) there is typically better report accuracy for T2 relative to T1 (e.g., Bowman & Wyble, 2007; Olivers, Hilkenmeier, & Scharlau, 2011), and (2) there are typically a disproportionate number of reversals in the order of report of the targets (e.g., Akyürek et al., 2012; Hommel & Akyürek, 2005).

Here, we offer a fine-grained characterization of attention deployment to targets in RSVP based on a recent set of results from

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Address correspondence to: Roberto Dell'Acqua, Centre for Cognitive Neuroscience, Via Venezia 8, 35131 Padova, Italy. E-mail: dar@unipd.it

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our group suggesting that attentional deployment and working memory encoding generate temporally and topographically distinguishable patterns of ERPs (Dell'Acqua et al., 2015). In this previous work, we employed a multitarget RSVP design and examined the electrophysiological P3 component (e.g., Dien, Spencer, & Donchin, 2004; Polich, 2003; Verleger, 1988) elicited by an unmasked target terminating an RSVP sequence. This last target-locked P3 activity was detected both at midfrontal and midparietal recording sites, and decomposed through independent component analysis (ICA). Specifically, the ICA analysis revealed two distinct P3 components, a frontal P3a peaking at about 300 ms posttarget, followed by a parietal P3b peaking at 400–450 ms posttarget. The parietal P3b varied with intertarget lag, with diminished amplitude and postponed latency at short relative to long lags—a typical ERP signature of the AB (Akyürek, Leszczyński, & Schubö, 2010; Brisson & Bourassa, 2014; Kranczioch, Debener, & Engel, 2003; Ptito, Arnell, Jolicœur, & MacLeod, 2008; Sessa, Luria, Verleger, & Dell'Acqua, 2007; Vogel & Luck, 2002; Vogel, Luck, & Shapiro, 1998). In addition, the frontal P3a was reduced in amplitude under analogous conditions, but, importantly, no onset latency variations were observed. These ERP modulations were more pronounced when the ERPs were time-locked to T3 in three-target RSVP trials (i.e., when T3 was preceded at varying lag by consecutive T1 and T2) relative to when ERPs were time-locked to T2 in standard two-target RSVP trials (i.e., when T2 was preceded at varying lag by T1). In both two-target and three-target trials, the P3a amplitude was inversely correlated with P3b latency, and this finding led us to propose that the efficiency of attention deployment to the last target, reflected in P3a amplitude, determined the amount of processing deferment of its encoding in visual working memory, reflected in P3b latency.

The goal of the Dell'Acqua et al.'s (2015) report was to characterize the time course of target processing under conditions where a target was preceded at varying lags by one (T1) or two consecutive targets (T1 and T2). However, given the richness of the electroencephalographic dataset, portions of it were left unexplored. In particular, P3a and P3b responses time-locked to the initial target(s), preceding the last one, in two-target and three-target RSVP trials were not compared. Reconsidering this specific part of Dell'Acqua et al.'s (2015) dataset is vital to further our understanding of the neurophysiological correlates of attentional selection and memory encoding in RSVP tasks. That is, whereas the original ERP analyses focused primarily on neurophysiological variations arising from a lag-dependent interaction between distinct attention episodes—one enabling the encoding of the initial target(s) and one enabling the encoding of the last target in RSVP—here, the aim is to provide a neurophysiological characterization of the processing occurring within an attention episode enabling the encoding of either a single target or two successively presented targets.

Two computational accounts of the AB,[1] which have specifically addressed RSVP-sparing phenomena and made explicit claims concerning ERP findings, are useful in the present context. These theories make predictions regarding how T1-locked P3a, reflecting attention deployment to target(s) and associated with neural structures localized frontally, should vary as a function of whether the T1 + 1 item is a distractor (in two-target trials) or a subsequent target (i.e., T2 in three-target trials). Both models predict that, with minimal latency variations, P3a amplitude should be greater when T1 is immediately followed by T2 relative to when T1 is followed by a distractor. According to Olivers and Meeter (2008), a distractor trailing T1 curtails attention deployment by eliciting an inhibitory response. The attentional response would, in contrast, have time to unfold to a greater extent when the T1 + 1 item is T2, that is, when the inhibitory response—elicited by the T2 + 1 distractor—is postponed by a time corresponding to T2 exposure duration. This activation asymmetry between a weakly activated T1 and a strongly activated T2 has been raised as the cause of order reversals in consecutive target report and for the better report of T2 relative to T1 at lag 1 (Olivers et al., 2011). Similarly, Wyble, Potter, Bowman, and Nieuwenstein (2011) propose that T1 and T2 both elicit attentional responses, but are processed in the same attentional window when presented sequentially, with T1 enhancing attention deployment to T2. Attentional enhancement would be discontinued when the T1 + 1 item is a distractor. Thus, both Olivers and colleagues and Wyble et al. (2011) maintain that order reversals in target report and the increased report accuracy for T2 relative to T1 are determined by the resulting asymmetry in target activations, with T2 overtaking T1 on a sizable proportion of trials.

Despite some similarities, these two models differ substantially with reference to time course and localization of ERP responses following P3a. The root cause of the AB in Olivers and Meeter's (2008) model is a transient inhibition (so-called bounce response) elicited by the T1 + 1 distractor to contrast the initial attention boost to T1 and prevent access of trailing nontarget items to working memory. In support of this hypothesis, Olivers and Meeter (2008) cite ERP evidence described by Martens, Munneke, Smid, and Johnson (2006), who explored the processing differences between blinkers (i.e., subjects who show average AB effects with RSVP) and nonblinkers (i.e., subjects who appear to be immune to the AB and tend to miss T2 in less than 10% of RSVP trials). Martens et al. (2006) reported that T1 elicited an initial positive component recorded in a 180–350 ms time range post-T1 at frontal electrode sites (F7 and F8), dubbed frontal selection positivity (FSP; e.g., Smid, Jakob, & Heinze, 1999), followed by a negative component observed at these frontal electrodes. Although this negative component was not parametrically investigated by Martens et al. (2006), Olivers and Meeter (2008) noted that the time course of the post-FSP frontal negative component, held to be the correlate of the bounce response, had a temporal extension of 300–500 ms after the offset of the FSP component, displaying therefore an interesting overlap with the time course of the AB. Olivers and Meeter (2008) further elaborated on the possible repercussions of this inhibitory response on classic ERP findings concerning the P3b response elicited by T2 in prior studies, raising the possibility of a direct link between the propagation of the post-FSP negative activity originating at frontal sites and T2-locked P3b suppression typically observed later and more posteriorly when a masked T2 is missed during the AB. In fact, this finding has been repeatedly associated with T2-locked P3b onset postponement when an unmasked T2 is displayed during the AB time window (e.g., Sessa et al., 2007). Referred to the present context, one distinctive prediction that can be derived from Olivers and Meeter's (2008) model is

1. In prior occasions (e.g., Dell'Acqua et al., 2012), we made reference to three computational AB models in order to produce and compare qualitative fits of empirical data collected using the RSVP paradigm, that is, the two computational models considered in this article (see text) and the model put forward by Taatgen, Juvina, Schipper, Borst, and Martens (2009). We note that the theory of Taatgen and colleagues, though of paramount importance for predicting the modulatory role of a number of factors on the AB phenomenon (among which, in particular, the critical role of individual differences in the AB), lacks a sufficiently clear formal description of the exact dynamics underlying lag-1 sparing to allow us to generate specific predictions concerning this effect and expected ERP modulations.

that the T1-locked frontal positive response[2] should be immediately followed by a frontal negative component indexing attentional inhibition elicited by the first target-trailing distractor. The onset timing of this frontal negative component should therefore differ between two-target and three-target trials, because the first target-trailing distractor is displayed in the T1 + 1 position in two-target trials, and in the T1 + T2 + 1 position in three-target trials. Furthermore, a second prediction emerges when taking into account a recent set of results obtained using a close variant of the present three-target versus two-target design in which the magnitude of the AB effect was estimated by monitoring the percent correct report of a masked last target trailing at varying lags a single target (i.e., T1) in two-target trials or two consecutive targets (i.e., T1 and T2) in three-target trials (Dux, Wyble, Jolicœur, & Dell'Acqua, 2014). Across four experiments, a consistent finding was that, at stimulus onset asynchrony (SOA) ranging from 168 to 420 ms, the last target's report suffered an AB that was substantially intensified when preceded by two consecutive targets in three-target trials compared to when the last target was preceded by a single target in two-target trials. In three-target trials, the correct report of T3 in three-target trials hovered at around 38%, whereas the correct report of T2 in two-target trials hovered at around 69%. From 420 ms onward, the correct report of T2 and T3 in two-target and three-target trials, respectively, did not differ significantly, implying that the AB was not characterized by a longer-lasting time course. These results rather suggested that the AB was just more intense in three-target versus two-target trials at short relative to long lags. These observations lead us to predict that a post-FSP/P3a negative ERP component should not just show a clear sign of being time-locked to the first target(s)-trailing distractor, but should also be of greater amplitude—reflecting stronger distractor-induced inhibition causing an AB magnification—in three-target trials than in two-target trials.

According to the model of Wyble et al. (2011), the AB is symptomatic of the visual system's overarching goal of generating episodically distinguishable episodes. As surmised above, targets in RSVP undergo attentional enhancement, which we proposed as indexed by an increment of frontal positivity, in order to bring their early sensory (and conceptual) representations beyond a certain threshold such that targets can be subsequently "tokenized" as reportable episodes and stored in working memory. Upon detection of a discontinuity in target presentation (e.g., upon detection of a distractor), attention enhancement is discontinued and tokenization encompasses all target information subject to attentional enhancement. Once tokenization is under way, no further targets can be subject to attention enhancement, with increased probability for nonattended targets to be missed, bringing about an AB effect. Crucially, this model explicitly predicts that, upon detection of T1, tokenization is immediately activated, whether T1 is trailed by a distractor or by T2, and this prediction has been tested in an ERP study by Craston, Wyble, Chennu, and Bowman (2009) showing that two sequential targets in RSVP elicit a single P3b response. Referred to the present context, a first prediction is therefore that, following a T1-locked frontal positive response, the onset latency of T1-locked P3b should not vary whether T1 is trailed by a distractor or by T2 in two-target and three-target trials, respectively. The second prediction arises from how the tokenization stage is characterized in the model, that is, as a stage where spatiotemporal information about target occurrence is bound to information about target identity. This leads to the hypothesis that processing required to generate a single token in working memory is increased when two such tokens must be generated for working memory storage. To note, tokenization in this perspective is strongly akin to the function ascribed to the stage of memory consolidation proposed by Jolicœur and Dell'Acqua (1998), who characterized this stage as operating serially on sequential targets (see Craston et al., 2009; Kihara, Kawahara, & Takeda, 2008, for analogous proposals). On these premises, if tokenization (or consolidation) takes longer for two targets relative to one target, then P3b should offset later in three-target trials, when T1 is trailed by T2 and both targets must be consolidated in working memory, rather than in two-target trials, when T1 is the only to-be-consolidated target.

To summarize, both the models of Wyble et al. (2011) and Olivers and Meeter (2008) predict a frontal positive ERP following the onset of T1. This frontal response is hypothesized to reflect the detection/selection of this target and should be of larger amplitude on three- versus two-target trials. However, the models diverge substantially in regard to the predicted ERPs following this initial frontal positive response. According to Olivers and Meeter (2008), a frontal negative component, indexing attentional inhibition, should be observed after this frontal positive response, with a delayed onset in three-target trials compared to two-target trials. Based on evidence showing a magnified AB effect following two consecutive targets in three-target trials relative to the magnitude of the AB elicited by a single target in two-target trials (Dux et al., 2014), the amplitude of the frontal negative activity following the frontal positive response in three-target trials should therefore be magnified relative to equivalent activity detected in two-target trials. This model does not provide sufficient details with reference to P3b activity—other than the shared assumption that P3b reflects target(s) consolidation in visual working memory—to allow us to make distinctive predictions about this component. However, one linchpin of the model is that there are no functional impediments to consolidate targets in visual working memory prior to the onset of the first target-trailing distractor. On this premise, there are no P3b modulations resulting from the present test that could be diagnostic of the appropriateness of the model to account for the AB and its ERP correlates.

According to Wyble et al. (2011), ERP activity following the initial frontal positive response should manifest itself as a P3b component primarily modulated by the time taken to consolidate target(s) in visual working memory. Crucially, the onset of the P3b component should not differ between two-target and three-target trials, whereas a temporally prolonged P3b offset is predicted for the consolidation of two consecutive targets in three-target trials relative to the consolidation of a single target in two-target trials. Here, we test these predictions.

Method

The dataset considered in this article was from the experiment originally described by Dell'Acqua et al. (2015). For the sake of completeness, we describe the design and methods of the experiment in full, and indicate in the section EEG/ERP Recordings and Preprocessing the conditions of interest for the present investigation.

Participants

Forty students at the University of Padua (23 females) participated in the experiment after giving informed consent. Their mean age

2. The issue of the similarities/differences between P3a and FSP components and their relative functional connotations is dealt with in Discussion.

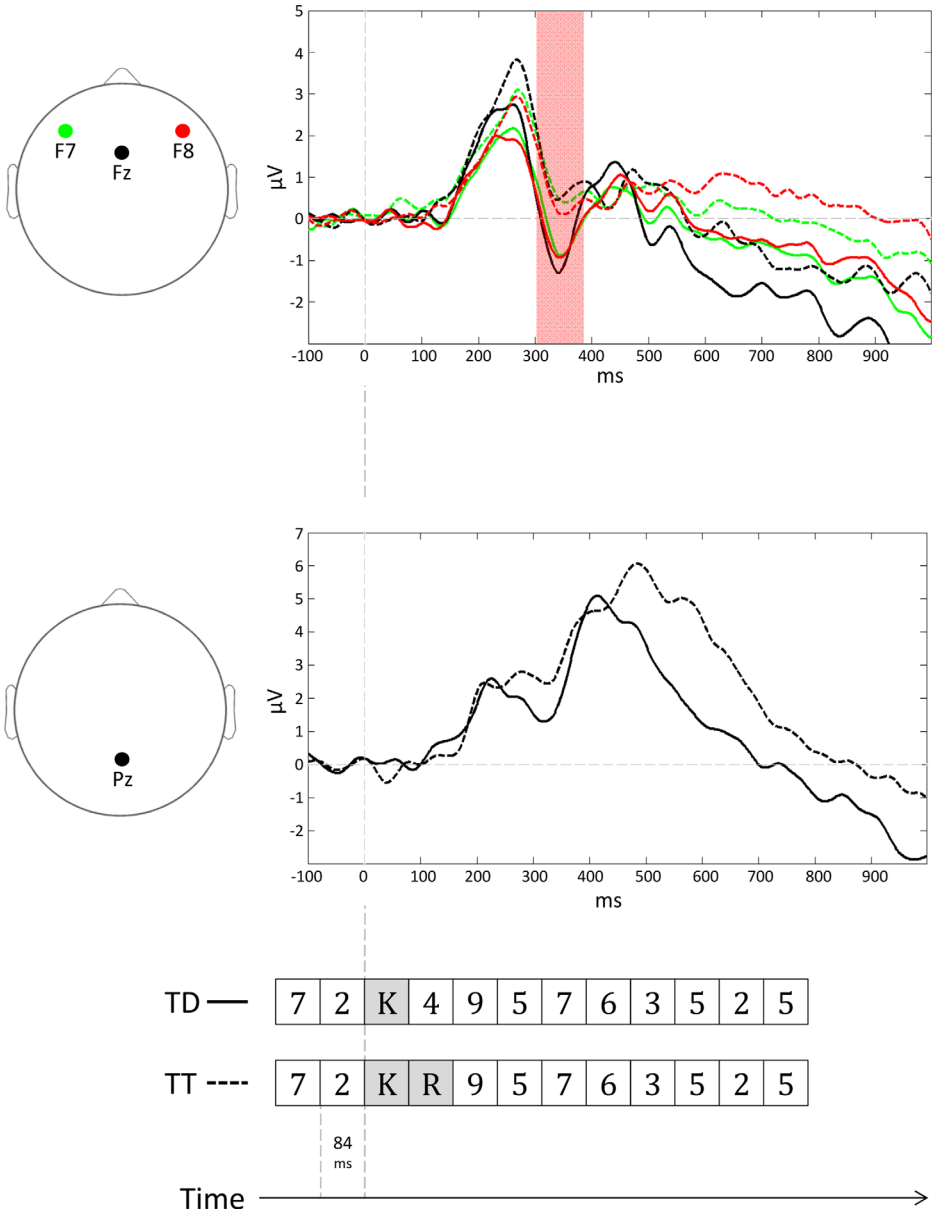

Figure 1. Lower: Gantt diagram illustrating the conditions of interest compared in the present article. In TT trials, T1 and T2 (highlighted as shaded letters for illustrative purposes) were always consecutive items, whereas T1 was trailed by a distractor in TD trials. Reported here are target-absent trials. Half of the trials were composed of target-present trials, namely, trials in which RSVP streams ended with a further target displayed after 8 distractors (SOA = 756 ms). Upper: T1-locked P3a and P3b components in TT (dashed lines) and TD trials (solid lines) flanked by a color-coded topographical indication of the originating electrode sites. The red shaded area provides information on the time window used for the post-P3a frontal negativity amplitude values calculation.

was 24.8 years ($SD = 4.6$). All had normal or corrected-to-normal visual acuity, and no history of neurological/psychiatric disorders.

Materials and Procedure

The stimuli were the digits 2 to 9 and the 22 letters of the English alphabet excluding *B*, *I*, *O*, and *Z*. The stimuli were displayed in light gray (34 cd/m^2) Romantri font against a black (6 cd/m^2) background. Luminance measurements were performed using a Minolta LS-100 Chroma Meter. Stimuli appeared on a 19″ CRT monitor running at 60 Hz, placed at a viewing distance of approximately 60 cm from the subject, controlled by an i686 IBM clone computer running MEL 2.0 software. RSVP streams were composed of distractor digits randomly selected from the available set, plus two or three different target letters (T1, T2, and T3) presented in various positions in the stream. Identical distractor digits in the RSVP stream were separated by a minimum of three different stimuli. Each stimulus was displayed for 84 ms, and was immediately replaced by the next stimulus (interstimulus interval, ISI = 0 ms). The lag between pairs of critical targets (i.e., T1–T2 lag in the two-target RSVP streams, or T2–T3 lag in three-target RSVP streams) was manipulated by varying the number of distractors between T1 and T2, or between T2 and T3. The number of distractors preceding T1 was varied randomly across trials from six to 11, and each RSVP stream ended with T2 in two-target RSVP streams, or T3 in three-target RSVP stream, which were replaced by a digit distractor

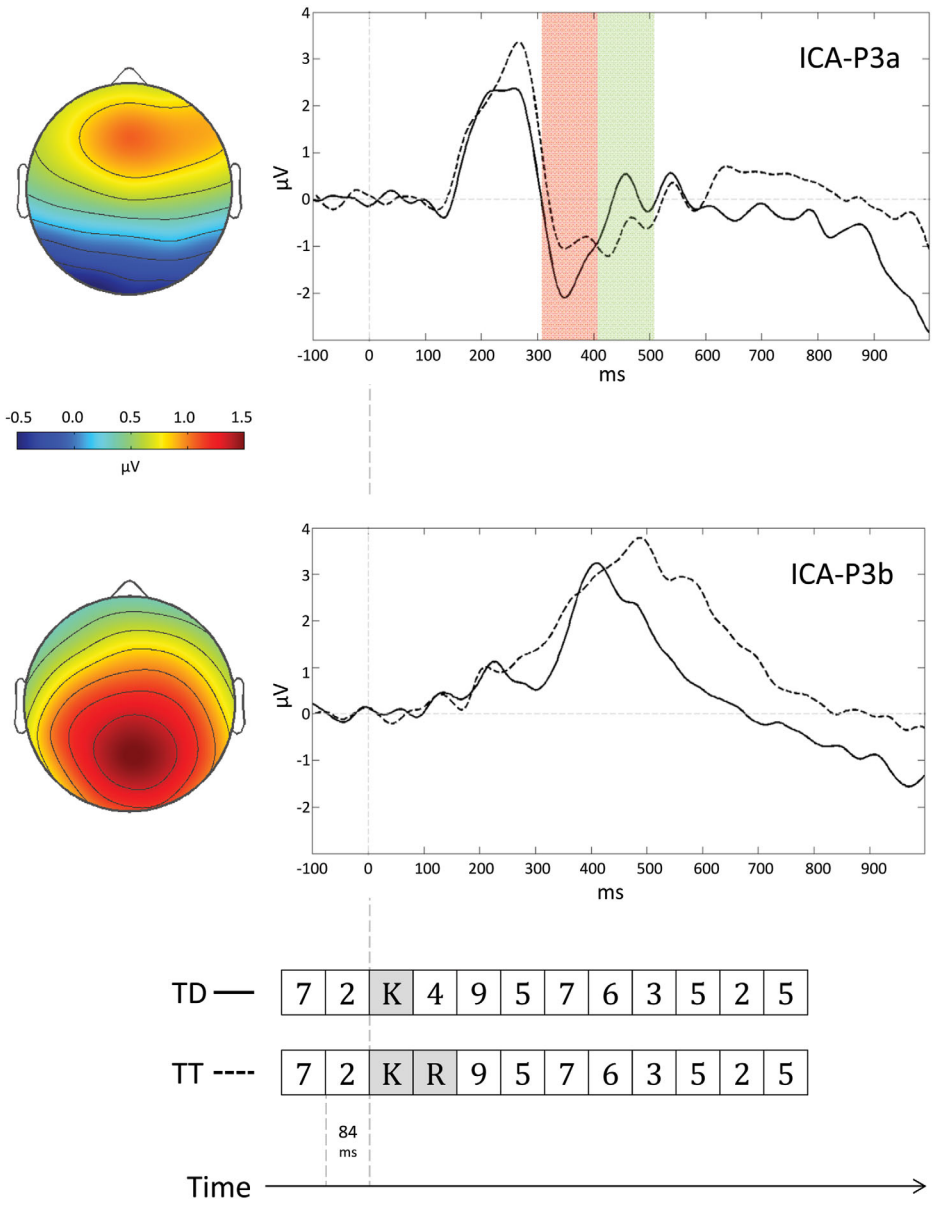

Figure 2. Lower: Gantt diagram illustrating the conditions of interest compared in the present article. Upper: Results of ICA decomposition of both T1-locked P3a and P3b components in TT (dashed lines) and TD trials (solid lines) flanked by the corresponding scalp plots of peak activity. The red and green shaded areas provide information on the time windows used for amplitude estimation of ICA-decomposed post-P3a frontal negativity in TD trials (red) and TT trials (green).

in the same position when the last target was not displayed. All stimuli were scaled to fit in a central, square portion of the monitor measuring 1.0° × 1.0° of visual angle.

In three-target RSVP streams, T1 and T2 were always consecutive items. The lag between T1 and T2 in two-target RSVP streams and between T2 and T3 in three-target RSVP streams was manipulated by presenting 2 (lag 3, SOA = 252 ms) or 8 (lag 9, SOA = 756 ms) distractors between these targets.

Each subject performed 648 trials, organized into 18 blocks of 36 trials each. Each lag condition appeared an equal number of times in each block, but their order was pseudorandomized, with the constraint that no more than three consecutive trials had the same lag. The last target in two-target (i.e., T2) or three-target (i.e., T3) RSVP streams was displayed on half of the (target-present) trials within each block, and replaced with a digit distractor in the same position on the other half of (target-absent) trials. Four randomly ordered RSVP streams in each block contained no targets (no-target trials). Half of the subjects started with nine consecutive blocks of two-target RSVP streams, followed by nine consecutive blocks of three-target RSVP streams. The opposite order applied for the other half of the subjects.

Each trial began with the presentation of a number of horizontally aligned plus signs in the center of the monitor denoting the number of targets that would appear in the forthcoming RSVP stream (i.e., two or three plus signs). Pressing the spacebar initiated a trial, causing the plus signs to disappear, and the RSVP to start 800 ms later. A question was displayed 800 ms after the end of the RSVP stream, requesting report of the targets by pressing the corresponding keys on the keyboard. Subjects were instructed to report all letters in the RSVP streams, with no emphasis on their order or

response speed. Feedback on an incorrectly reported target was provided at the end of each trial by replacing the plus sign in the position congruent with target order (from left to right, T1, T2, and T3, when present) with a minus sign. Experimental data were collected after no fewer than 20 RSVP streams for practice in each of two-target and three-target conditions.

EEG/ERP Recordings and Preprocessing

EEG activity was recorded continuously from 28 active electrodes (at positions Fp1, Fp2, Fz, F3, F4, F7, F8, FCz, C3, C4, Cz, CP1, CP2, CP5, CP6, P3, P4, Pz, O1, O2, Oz, T7, T8, TP9, PO9, PO10, P7, and P8 in the 10-10 system) placed on an elastic Acti-Cap (Brain Products), referenced to the left earlobe. Horizontal electrooculogram (HEOG) activity was recorded bipolarly from electrodes positioned on the outer canthi of both eyes. Vertical EOG (VEOG) activity was recorded bipolarly from two electrodes, above (Fp1) and below the left eye. Impedance at each electrode site was maintained below 5 KΩ. EEG, HEOG, and VEOG activities were amplified, filtered using a band-pass of 0.016–80 Hz, digitized at a sampling rate of 500 Hz, and referenced offline to the average of the left and right earlobes. ICA was used to identify blink and saccade components in the continuous EEG recordings and remove them from the data (Delorme & Makeig, 2004; Jung et al., 2000). The corrected EEG was high-pass filtered at 0.1 Hz and low-pass filtered at 20 Hz and then segmented into 1,100-ms epochs starting 100 ms prior to the onset of T1 in the RSVP stream and ending 1,000 ms after, and baseline-corrected using the mean activity in the interval [−100, 0] ms. In order to ensure no residual artifacts remained on the EOG channels, each segment was examined in the interval [−100, 1,000] ms relative to the onset of T1 for voltage deviations greater than 80 μV in any period of 150 ms for the VEOG difference waveform, or a deviation greater than 45 μV in any 300-ms period for the HEOG difference waveform. Segments with residual ocular artifacts were removed from the data set. EEG channels were flagged when the signal exceeded ± 100 μV anywhere in the analysis segment. If a segment had seven or fewer flagged data channels, these channels were interpolated using a spherical spline interpolation algorithm in EEGLAB (Delorme & Makeig, 2004) for that segment. Segments with more than seven flagged channels were discarded.

As illustrated in the lower part of Figure 1 and 2, the critical analyses were carried out on separate T1-locked ERP waveforms generated in trials at the longer lag only. This was done in order to minimize the overlap between ERP waveforms elicited by T1 in two-target trials and by consecutive T1 and T2 in three-target trials, and ERP waveforms elicited by the last target in the RSVP streams. Henceforth, for ease of exposition, we will refer to two-target trials as TD trials (to indicate that T1 was followed by a distractor) and to three-target trials as TT trials (to indicate that T1 was followed by another target, T2).

T1-locked ERP waveforms in TD and TT conditions were estimated by averaging EEG epochs recorded on both target-present and target-absent trials (i.e., with and without a final target ending the RSVP streams) associated with the correct report of T1 in TD trials, and T1 and T2 in TT trials. ERPs recorded in no-target trials were subtracted from these ERP waveforms to eliminate EEG oscillations in phase with the rate of presentation of RSVP items (about 12 Hz; alpha band; cf. Dell'Acqua et al., 2015).

The mean amplitude of the subtracted T1-locked P3a and P3b components was quantified as the mean value in a 150-ms window centered on the peak of each grand-averaged ERP. Given the explicit reference of Olivers and Meeter (2008) to the frontal modulations of ERP activity reported by Martens et al. (2006), frontal activity in the P3a time range was analyzed at F7, Fz, and F8 electrodes. The P3b component was analyzed at Pz (Polich, 2003). The mean latency of the subtracted P3a and P3b components at the same recording sites was estimated using the jackknife approach (Kiesel, Miller, Jolicœur, & Brisson, 2008; Ulrich & Miller, 2001), and individual values were derived with the solution proposed by Brisson and Jolicœur (2008; see also Smulders, 2010). Onset latency values were calculated as the time point when the ascending portion of individual jackknife time course reached 75% of the peak amplitude, consistent with the original work of Dell'Acqua et al. (2015). Offset latency values were calculated as the mean time point when the descending portion of individual jackknife ICA time course crossed the 75% amplitude value. The Greenhouse-Geisser correction for nonsphericity was applied when appropriate.

Results

Behavior

Separate analyses of variance (ANOVAs) were carried out to compare the mean proportion of correct target report in TD and TT trials. Subjects were more accurate in reporting T1 in TD trials (95.4%) than in TT trials (79.2%), $F(1,39) = 104.5$, $\eta_p^2 = .732$, $p < .0001$. In TT trials, subjects were more accurate in reporting T2 (93.3%) than T1, $F(1,39) = 100.4$, $\eta_p^2 = .724$, $p < .0001$. T1 report in TD trials was also superior to T2 report in TT trials, $F(1,39) = 5.7$, $\eta_p^2 = .130$, $p < .05$. Block order (i.e., whether subjects started the experiment with three-target or two-target trial blocks) did not exert any effect on behavioral performance, max $F < 1$. Furthermore, in 46.1% of TT trials, T1 and T2 were correctly reported albeit in reversed order. In short, as repeatedly observed in prior investigations, under TT conditions T2 was reported more accurately than T1 and, on a substantial proportion of trials, was reported as the first target.

ERPs

The artifact screening procedures described above resulted in the exclusion of 0.74% of the segments. For most subjects, less than 1% of the data were excluded. Two subjects had exclusion rates of about 7%. Visual inspection of their ERPs suggested their results were comparable to those of the other subjects, and thus their data were included in the final analyses. The final sample included all 40 participants tested in the experiment. In all the following ERP analyses, block order (i.e., whether subjects started the experiment with three-target or two-target trial blocks) was included in the various ANOVA designs. However, given that block order was never associated with significant main effects or interactions with the other considered factors, max $F < 1$, min $p > .43$, the influence of this factor is not discussed in the forthcoming sections. The most important T1-locked ERP waveforms observed in the present experiment are reported in Figure 1.

P3a. The mean P3a amplitude values observed in TT versus TD trials were 2.95 μV and 2.09 μV at Fz, 2.44 μV and 1.66 μV at F7, and 2.1 μV and 1.68 μV at F8. An ANOVA carried out on individual P3a amplitude values indicated that P3a was of greater amplitude in TT than TD trials, $F(1,38) = 5.8$, $\eta_p^2 = .132$, $p < .021$. Furthermore, P3a amplitude differed across electrode sites, $F(2,76) = 5.2$, $\eta_p^2 = .120$, $p < .008$. False discovery rate (FDR; Benjamini & Hochberg, 1995) corrected t tests indicated that P3a

amplitude was greater at Fz relative to both F7, $t(79) = 3.0$, $p < .01$, and F8, $t(79) = 3.44$, $p < .001$, which did not differ significantly, $t < 1$, $p > .34$. The mean P3a onset latency in TD trials (219 ms) and in TT trials (225 ms) did not differ significantly, $F(1,38) = 1.2$, $p > .4$. The mean P3a offset latency in TD trials (283 ms) and in TT trials (294 ms) also did not differ significantly, $F(1,38) = 2.1$, $p > .2$. No difference in P3a onset/offset latencies was found across F7, Fz, and F8 electrodes, $F < 1$, $p > .35$.

Post-P3a frontal negativity. In order to test Olivers and Meeter's (2008) prediction concerning distractor-induced attention inhibition being indexed by a peak of negativity following the initial frontal activation reflected by the T1-locked P3a, ERP activity trailing P3a was explored at each frontal electrode considered in the P3a analyses (i.e., F7, Fz, and F8) in a time window starting 303 ms post-T1 (i.e., a value corresponding to the mean time point at which the descending portion of P3a crossed the baseline in TT and TD trials) and ending at 390 ms post-T1 (highlighted in red in Figure 1). The mean amplitude of this component was −.39 μV on TD trials, and .74 on TT trials. An ANOVA revealed that these values differed significantly, $F(1,38) = 14.8$, $\eta_p^2 = .281$, $p < .001$, and were comparable across electrode sites, $F = 1.3$, $p > .20$. As shown in Figure 1, separate one-tailed t tests indicated that negative ERP activity (i.e., significantly less than 0) was detected in TD trials, $t(119) = -2.1$, $p < .05$, but not in TT trials, where the component was positive.

P3b. The mean amplitude of P3b was 4.03 μV in TD trials, and 5.31 μV in TT trials. The ANOVA showed that these values were significantly different, $F(1,38) = 14.6$, $\eta_p^2 = .269$, $p < .001$. The P3b onset latency was not different between TD trials (386 ms) and TT trials (393 ms), $F < 1$, $p > .7$. The P3b offset latency was, however, substantially postponed in TT trials (496 ms) relative to TD trials (596 ms), $F(1,38) = 70.3$, $\eta_p^2 = .641$, $p < .001$.

ICA of ERPs

The same EEGLAB routine as that described in Dell'Acqua et al. (2015) was used to decompose T1-locked ERPs through ICA (Delorme & Makeig, 2004). This was done to provide a more faithful depiction of the ERP results by decomposing the various components explored through standard analyses into maximally spatiotemporally independent signals available in the channel data, and minimize the influence of their potential overlap/summation on the interpretation of the above findings. One hypothesis in particular that had to be ruled out is that the post-P3a frontal negativity (absent) in TT trials may have been camouflaged by spatiotemporal superimposition with a surge of positive activity trailing P3a, which could unpredictably have been more intense and/or anticipated in TT versus TD trials.

Individual ERPs in TT and TD trials were first analyzed using singular value decomposition to determine the dimensionality of the signal subspace containing most of the relevant event-related activity. A scree plot of the singular values showed a clear break after the first three components, leading us to retain the first four dimensions, which accounted for 51.8% of the variance. The ICA analysis was thus restricted to this subspace of the signal space using an initial principal component analysis (PCA). The ICA decomposition isolated two components of the P3 family, namely, an earlier anterior component (ICA-P3a) and a later posterior component (ICA-P3b). The grand-averaged time courses and relative topographies for these two components in TD and TT trials are illustrated in Figure 2.

ICA: P3a. The mean amplitude of ICA-P3a was significantly greater in TT trials (2.45 μV) than in TD trials (1.89 μV), $F(1,38) = 5.46$, $\eta_p^2 = .123$, $p < .03$. The mean onset latency of ICA-P3a in TD trials (201 ms) and TT trials (223 ms) did not differ significantly, $F(1,38) = 1.9$, $p > .35$. The mean offset latency of ICA-P3a was 272 ms in TD trials and 289 ms in TT trials. These values were statistically different, $F(1,38) = 4.31$, $\eta_p^2 = .101$, $p < .05$.

ICA: Post-P3a frontal negativity. As Figure 2 suggests, our speculations that post-P3a negative activity may have been influenced by spatiotemporal overlap with contrasting positive activity that varied between TT and TD trials was correct. Contrary to the results observed for the standard ERP analyses, the ICA decomposition revealed that negative ERP activity trailed P3a in both TD and TT trials.

An ANOVA carried out on latency values indicated that the onset latency of ICA-decomposed post-P3a frontal negativity did not differ between TD trials (322 ms) and TT trials (327 ms), $F = 1.6$, $p > .2$. However, inspection of Figure 2 suggests that TT and TD trials elicited T1-locked ERP time courses seemingly compatible with the prediction that post-P3a frontal activity in TD trials should be anticipated relative to equivalent activity in TT trials (i.e., in which the distractor is postponed by 84 ms). In a 310–510 ms time window (i.e., from the mean time point at which the descending portion of the P3a component in TD trials crossed the baseline to the final convergence of TT and TD waveforms), the post-P3a negative deflection in TD trials is in fact more skewed toward an earlier peak in TD trials, and more symmetrical around a later peak in TT trials. The crucial test on the relative amplitude of the post-P3a negative component in TD and TT trials—guided by assuming the postponement of post-P3a activity in TT trials versus TD trials, predicted on the basis of Olivers and Meeter's (2008) model—was performed by splitting the 310–510 ms time window in half, and by comparing the amplitude of post-P3a negative activity recorded in a 310–410 ms time window for TD trials with the amplitude of post-P3a negative component recorded in a 410–510 ms time window for TT trials. Two preliminary one-tailed t tests confirmed that the recorded activity was indeed negative (i.e., significantly less than 0) in both TD trials, $t(39) = -4.9$, $p < .001$, and TT trials, $t(39) = -2.3$, $p < .02$. Contrary to the predicted magnification of the amplitude of the post-P3a negative component, a subsequent analysis revealed that the amplitude of post-P3a negative activity was greater in TD trials (−1.45 μV; 310–410 ms) than in TT trials (−.74 μV; 410–510 ms), $F(1,38) = 5.31$, $\eta_p^2 = .144$, $p < .03$. A final analysis was conducted to compare the overall amplitude of post-P3a negative activity in the entire 310–510 ms time window. The result of this analysis revealed that post-P3a negativity amplitude in TT trials (−.731 μV) was basically identical to post-P3a negativity amplitude in TD trials (−.733 μV), $F = .05$, $p > .99$.

ICA: P3b. The mean amplitude of the ICA-P3b was significantly greater in TT trials (3.29 μV) than in TD trials (2.48 μV), $F(1,39) = 13.34$, $\eta_p^2 = .255$, $p < .0001$. The mean onset latency of ICA-P3b was not different between TD trials (382 ms) and TT trials (397 ms), $F(1,38) = 2.0$, $p > .2$. However, the mean offset latency of the ICA-P3b was substantially longer in TT trials (492 ms) than in TD trials (600 ms), $F(1,38) = 68.4$, $\eta_p^2 = .642$,

$p < .001$. This 108-ms difference between ICA-P3b offset latencies was significantly longer than the 17-ms difference between ICA-P3a offset latencies in TD versus TT trials, $F(1,76) = 52.8$, $\eta_p^2 = .410$, $p < .001$.

Discussion

In the present study, we analyzed a portion of a large ERP dataset that was left unexplored in a prior investigation (Dell'Acqua et al., 2015). The selected ERP data were those bearing critically on cognitive events and neural underpinnings of the selection and encoding of a single target, in TD trials, or two consecutive targets, in TT trials, embedded in RSVP streams of distractors. Standard analyses and an ICA reconstruction of the spatiotemporal T1-locked ERP patterns were consistent in revealing that the difference between TT and TD trials was reflected primarily in modulations of two subcomponents of the P3 complex. Specifically, both analytical approaches produced results indicating a frontocentral T1-locked P3a waveform of larger amplitude in TT trials than in TD trials. This P3a amplitude increase elicited by consecutive targets was accompanied by a 17-ms postponement of the corresponding P3a offset latency. Hints of negative activity trailing P3a were found only in TD trials using a standard ERP approach, and generally more marked in TD versus TT trials using the ICA approach. A centroparietal P3b was also observed to be of greater amplitude in TT trials relative to TD trials, with a postponement of P3b offset latency in TT trials that was, however, one order of magnitude more substantial than that for P3a, amounting to 108 ms. There is reasonable agreement on the role of the dorso- and ventrolateral prefrontal cortices in the generation of P3a (e.g., Ranganath & Rainer, 2003). Indeed, these current results are in broad agreement with evidence indicating the involvement of the frontoparietal network in enabling attentional selection of task-relevant information, both when displayed simultaneously with arrays of spatially distributed distracting information (Corbetta, 1998; Todd & Marois, 2004; Xu & Chun, 2006; Yantis et al., 2002) and when embedded in a spatially overlapping, but temporally distributed, sequence of distracting events (Dell'Acqua, Sessa, Jolicœur, & Robitaille, 2006; Husain, Shapiro, Martin, & Kennard, 1997; Joseph, Chun, & Nakayama, 1997; Lagroix, Grubert, Spalek, Di Lollo, & Eimer, 2015; Marcantoni, Lepage, Beaudoin, Bourgouin, & Richer, 2003; Marois, Chun, & Gore, 2000). There is also good agreement that more posterior regions, including the temporoparietal junction and inferotemporal cortices, are likely involved in the generation of P3b (e.g., Polich, 2003, 2007).

Specific predictions about the possible ERP modulations in the present design were derived from two current neurocomputational models of temporal selective attention. Predictions from Olivers and Meeter (2008) and Wyble et al. (2011) concerning the time course of attention deployment to the first target(s) encountered in RSVP were confirmed by the amplitude increase of T1-locked P3a when T1 was trailed by another target relative to when T1 was displayed as a single target in RSVP. According to Olivers and Meeter (2008), attention deployment to RSVP targets is necessary to transfer these stimuli into visual working memory. As detailed in the introduction, this model predicts that attentional deployment to T1 in TD trials would be curtailed by the inhibitory response elicited by the distractor trailing T1, which would attenuate the T1-locked P3a response. This would not occur in TT trials given the presence of T2 trailing T1, which would provide more time for the P3a response to grow further, as was in fact observed. According to Wyble et al. (2011; see Figure 6, p. 493), attention is deployed to RSVP targets to enhance their sensory traces so as to enable them to activate corresponding "types," namely, nodes in conceptual short-term memory (Chun & Potter, 1995; Potter, 1976). Types in turn can be encoded as tokens, that is, reportable items, once they are bound to physical features promoting episodic distinctiveness. In this model, the summation of attentional responses to T1 and T2 would be the cause of the increased P3a amplitude in TT trials compared to TD trials. In line with our prior observations (Dell'Acqua et al., 2015), the offset latency difference of P3a between TT and TD trials was minimal. This suggests that processing of two consecutive targets at stages prior to memory encoding overlap considerably, dovetailing with earlier reports using faster RSVP presentation rates than typically employed. For example, Potter et al. (2005) displayed two synchronous RSVP sequences of nonwords, one above and one below a central fixation point at 20 Hz, each embedding one target word, T1 and T2. T1 and T2 were names of semantically related real-world concepts on half of the trials, and unrelated concepts on the other half of trials. Critically, at SOA ranging from 0 to 120 ms, a semantically related T2 primed T1, thus supporting the idea that, when presented in close temporal proximity, type nodes were simultaneously active in conceptual short-term memory.

One may wonder why T2 in TT trials, whose onset coincided temporally with the bulk of attention accumulation indexed by P3a, was reported less correctly than T1 in TD trials. The AB models used to generate the predictions tested in the present study provide different explanations for this often-observed effect. Both accounts postulate that encoding two consecutive targets incurs some form of intertarget interference. The models differ, however, relative to the locus of this interference. Olivers and Meeter (2008) propose a visual working memory locus, wherein encoded targets compete for maintenance and recall (see also Raymond et al., 1992, for an analogous proposal). Wyble et al. (2011) posit mutual inhibition of concurrently active types, and this is reflected in slightly lower report accuracy for consecutive targets relative to when targets are displayed in RSVP separated by intervals outlasting the AB window (cf. Dell'Acqua, Dux, Wyble, & Jolicœur, 2012, for supporting evidence).

One issue to be elucidated is whether the frontal positivity that we labelled P3a here and in the Dell'Acqua et al. (2015) study is really a "true" P3a or some other ERP component. Labeling ERP components and/or assigning them a specific functional connotation is often a matter of contention (e.g., Dien et al., 2004). On the one hand, in our earlier report (Dell'Acqua et al., 2015), we provided justifications that the present P3a is probably not a frontal P2 described in some other AB studies (e.g., Vogel et al., 1998). However, it must be noted that views regarding the functional significance of the P3a component have changed over the last decade, from a focus on novelty/deviance detection (usually explored using oddball designs) to the currently shared position of P3a as linked to top-down attention control (e.g., Akyürek & Meijerink, 2012; Barceló, Escera, Corral, & Periáñez, 2006; Barceló, Periáñez, & Knight, 2002; Polich, 2007; Prada, Barceló, Herrmann, & Escera, 2014). One hypothesis is that the P3a corresponds to the transient enhancement of frontal selection positivity (FSP) found by Martens et al. (2006; see also Potts, 2004; Smid et al., 1999) and measured in a 180–350 ms window post-T1 onset. Martens et al. (2006) concluded that FSP reflected attention control over target(s) selection, which is germane to the present idea of the function indexed by P3a (see also Dell'Acqua et al., 2015). Thus, a small difference aside in the topography of the FSP component, whose peak was found at F7/F8 by Martens et al. (2006), the overlap of temporal

parameters and proposed functional connotations of FSP and P3a suggests that the recruitment of the frontal brain regions for attention-guided selection in RSVP is reflected in rapid increments of frontal positive activity upon detection of T1.

Limited evidence was found for attention inhibition induced by the lag-1 distractor as predicted by Olivers and Meeter (2008). Based on the boost and bounce architecture, a negative component with a time course corresponding to that of the AB should have been observed following P3a at frontal electrode sites. This negative component, reported by Martens et al. (2006) in a post-FSP/P3a T1-locked time window, was parametrically investigated by Niedeggen, Hesselman, Sharaie, Milders, and Blakemore (2004), who proposed that this component could index the activation of an "attention-gating mechanism" temporarily halting processing of visual information trailing a leading, attention-demanding visual event. In this perspective, given the temporal shift in onset of the distractor trailing one single target or two consecutive targets in TD and TT trials, respectively, a negative component with a postponed latency was expected in TT trials relative to TD trials. The results produced using the standard and ICA approaches do not appear in line with this prediction. No latency variations compatible with the hypothesis that post-P3a negative activity was elicited by the first target(s)-trailing distractor were detected when the negative component emerged following the ICA decomposition of the multivariate spatiotemporal distribution of the T1-locked ERP signal. Furthermore, when the overall amplitude of post-P3a negative component was explored in a 310–510 ms time window, the results indicated an equivalence between TD and TT trials, which is incongruent with behavioral findings reflecting a much more pronounced AB in TT versus TD trials when tested with the behavioral variant of the present design (i.e., by masking the last target and monitoring its correct report; Dux et al., 2014). So, although a precise functional characterization of the post-P3a negative activity is beyond the scope of the present investigation and certainly worth further experimental inspection, the present ERP results appear to generally run counter to the idea of a distractor-induced nature of the AB.

The ICA-P3b results were clear-cut: The onset of the P3b did not differ in TD versus TT trials, and its duration was longer in TT trials than in TD trials. These results suggest that encoding two targets took longer than encoding one target (Dell'Acqua et al., 2012; Dell'Acqua, Jolicœur, Luria, & Pluchino, 2009; Dux, Asplund, & Marois, 2008; Jolicœur & Dell'Acqua, 1998), and converge with proposals about the target-locked essence of the AB effect (see Dux & Marois, 2009, and Martens & Wyble, 2010, for extensive surveys and comparisons of models of temporal attention hinging on this principle). Collectively, the P3b findings appear to be congruent with predictions based on Wyble et al. (2011).

Two aspects of the P3b response time course deserve particular consideration. The P3b responses recorded in both TT and TD trials showed comparable onsets but clearly different offsets, that is, the P3b offset latency was postponed in TT relative to TD trials. In other words, the P3b response was unimodal, much like the P3b response to consecutive targets reported by Craston et al. (2009). These authors interpreted the unimodal P3b response elicited by two consecutive targets as evidence for the targets' integration into a single attention episode (see also Kessler et al., 2005, for analogous evidence produced using magnetoencephalography, MEG). The present results complement and extend those of Craston et al. (2009) by establishing a direct link between the amount of information that is ultimately encoded in visual working memory and the temporal extension of an attention episode.

The second aspect emerges from the comparison between P3b duration (difference between offset and onset latencies) across TD and TT trials. The 108-ms difference in P3b duration for TT trials relative to TD trials suggests it takes, on average, 108 ms more to encode two targets compared with one target. This may seem surprising given the apparently relatively long time required for the P3b to reach its peak amplitude. However, the peak of the P3b presumably reflects encoding as well as all processes taking place prior to encoding. The difference between TT and TD conditions presumably subtracts out some of these differences, leaving a closer estimate of the mean encoding duration. Interestingly, Jolicœur and Dell'Acqua (1998), using dual-task methods and computer simulation, arrived at an estimate of about 169 ms of additional time to encode two letters (suggesting an encoding cost of about 84 ms per item, which is not far from the present estimate of 108 ms; see their Table 2 and Experiment 7). Encoding into working memory, or short-term consolidation, appears to be a slow process with high variance but for which the cost of additional items hovers around 108 ms, a value that converges nicely with the estimate reported by Craston et al. (2009) of 100 ms estimated from the T1-locked P3b offset in lag-1 trials compared to that in lag-8 trials.

Conclusion

Here, we assessed rapid visual information processing for items that appear within the same temporal attention window using EEG. Contrasting two prominent computational models of the AB, we observed that the P3 complex could be fractionated into distinct components, which were affected differently by whether or not an attentional window contained two targets or a target and a distractor. Specifically, whereas only frontal P3a amplitude was influenced by increased target load, both amplitude and latency of the parietal P3b were increased by target load. The results suggest that within temporal attention windows there are two stages of information processing subserved by distinct neural substrates. Selection appears to occur close-to-concurrently for multiple targets and draws on frontal regions of the brain. This then leads to encoding of this information in a serial manner that prominently taps the temporoparietal lobes.

References

Akyürek, E. G., Eshuis, S. A. H., Nieuwenstein, M. R., Saija, J. D., Başkent, D., & Hommel, B. (2012). Temporal target integration underlies performance at lag 1 in the attentional blink. *Journal of Experimental Psychology: Human Perception and Performance*, *38*, 1448–1464. doi: 10.1037/a0027610

Akyürek, E. G., Leszczyński, M., & Schubö, A. (2010). The temporal locus of the interaction between working memory consolidation and the attentional blink. *Psychophysiology*, *47*, 1134–1141. doi: 10.1111/j.1469-8986.2010.01033.x

Akyürek, E. G., & Meijerink, S. K. (2012). The deployment of visual attention during temporal integration: An electrophysiological investigation. *Psychophysiology*, *49*, 885–898. doi: 10.1111/j.1469-8986.2012.01380.x

Barceló, F., Escera, C., Corral, M. J., & Periáñez, J. A. (2006). Task switching and novelty processing activate a common neural network for cognitive control. *Journal of Cognitive Neuroscience*, *18*, 1734–1748. doi: 10.1162/jocn.2006.18.10.1734

Barceló, F., Periáñez, J. A., & Knight, R. T. (2002). Think differently: A brain orienting response to task novelty. *NeuroReport*, *13*, 1887–1892. doi: 10.1097/00001756-200210280-00011

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of*

the Royal Statistical Society: Series B, *57*, 289–300. doi: 10.2307/2346101

Bowman, H., & Wyble, B. (2007). The simultaneous type, serial token model of temporal attention and working memory. *Psychological Review*, *114*, 38–70. doi: 10.1037/0033-295X.114.1.38

Brisson, B., & Bourassa, M. È. (2014). Masking of a first target in the attentional blink attenuates the P3 to the first target and delays the P3 to the second target. *Psychophysiology*, *51*, 611–619. doi: 10.1111/psyp.12204

Brisson, B., & Jolicœur, P. (2008). Express attentional re-engagement but delayed entry into consciousness following invalid spatial cues in visual search. *PLoS ONE*, *3*, e3967. doi: 10.1371/journal.pone.0003967

Chun, M. M., & Potter, M. C. (1995). A two-stage model for multiple target detection in rapid serial visual presentation. *Journal of Experimental Psychology: Human Perception and Performance*, *21*, 109–127. doi: 10.1037/0096-1523.21.1.109

Corbetta, M. (1998). Frontoparietal cortical networks for directing attention and the eye to visual locations: Identical, independent, or overlapping neural systems? *Proceedings of the National Academy of Sciences (USA)*, *95*, 831–838. doi: 10.1073/pnas.95.3.831

Craston, P., Wyble, B., Chennu, S., & Bowman, H. (2009). The attentional blink reveals serial working memory encoding: Evidence from virtual and human event-related potentials. *Journal of Cognitive Neuroscience*, *21*, 550–566. doi: 10.1162/jocn.2009.21036

Dell'Acqua, R., Dux, P. E., Wyble, B., Doro, M., Sessa, P., Meconi, F., & Jolicœur, P. (2015). The attentional blink impairs detection and delays encoding of visual information: Evidence from human electrophysiology. *Journal of Cognitive Neuroscience*, *27*, 720–735. doi: 10.1162/jocn_a_00752

Dell'Acqua, R., Dux, P. E., Wyble, B., & Jolicœur, P. (2012). Sparing from the attentional blink is not spared from structural limitations. *Psychonomic Bulletin & Review*, *19*, 232–238. doi: 10.3758/s13423-011-0209-3

Dell'Acqua, R., Jolicœur, P., Luria, R., & Pluchino, P. (2009). Reevaluating encoding-capacity limitations as a cause of the attentional blink. *Journal of Experimental Psychology: Human Perception and Performance*, *35*, 338–351. doi: 10.1037/a0013555

Dell'Acqua, R., Sessa, P., Jolicœur, P., & Robitaille, N. (2006). Spatial attention freezes during the attentional blink. *Psychophysiology*, *43*, 394–400. doi: 10.1111/j.1469-8986.2006.00411.x

Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, *134*, 9–21. doi: 10.1016/j.jneumeth.2003.10.009

Dien, J., Spencer, K. M., & Donchin, E. (2004). Parsing the late positive complex: Mental chronometry and the ERP components that inhabit the neighborhood of the P300. *Psychophysiology*, *41*, 665–678. doi: 10.1111/j.1469-8986.2004.00193.x

Di Lollo, V., Enns, J. T., & Rensink, R. A. (2000). Competition for consciousness among visual events: The psychophysics of reentrant visual processes. *Journal of Experimental Psychology: General*, *129*, 481–507. doi: 10.1037/0096-3445.129.4.481

Dux, P. E., Asplund, C. L., & Marois, R. (2008). An attentional blink for sequentially presented targets: Evidence in favor of resource depletion accounts. *Psychonomic Bulletin & Review*, *15*, 809–813. doi: 10.3758/PBR.15.4.809

Dux, P. E., & Marois, R. (2009). The attentional blink: A review of data and theory. *Attention, Perception, & Psychophysics*, *71*, 1683–1700. doi: 10.3758/APP.71.8.1683

Dux, P. E., Wyble, B., Jolicœur, P., & Dell'Acqua, R. (2014). On the costs of lag-1 sparing. *Journal of Experimental Psychology: Human Perception and Performance*, *40*, 416–428. doi: 10.1037/a0033949

Hommel, B., & Akyürek, E. G. (2005). Lag-1 sparing in the attentional blink: Benefits and costs of integrating two events into a single episode. *Quarterly Journal of Experimental Psychology*, *58A*, 1415–1433. doi: 10.1080/02724980443000647

Husain, M., Shapiro, K., Martin, J., & Kennard, C. (1997). Abnormal temporal dynamics of visual attention in spatial neglect patients. *Nature*, *385*, 154–156. doi: 10.1038/385154a0

Jolicœur, P., & Dell'Acqua, R. (1998). The demonstration of short-term consolidation. *Cognitive Psychology*, *36*, 138–202. doi: 10.1006/cogp.1998.0684

Joseph, J. S., Chun, M. M., & Nakayama, K. (1997). Attentional requirements in a preattentive visual search task. *Nature*, *387*, 805–807. doi: 10.1038/42940

Jung, T. P., Makeig, S., Humphries, C., Lee, T. W., McKeown, M. J., Iragui, V., & Sejnowski, T. J. (2000). Removing electroencephalographic artifacts by blind source separation. *Psychophysiology*, *37*, 163–178. doi: 10.1111/1469-8986.3720163

Kessler, K., Schmitz, F., Gross, J., Hommel, B., Shapiro, K., & Schnitzler, A. (2005). Target consolidation under high temporal processing demands as revealed by MEG. *NeuroImage*, *26*, 1030–1041. doi: 10.1016/j.neuroimage.2005.02.020

Kiesel, A., Miller, J., Jolicœur, P., & Brisson, B. (2008). Measurement of ERP latency differences: A comparison of single-participant and jackknife-based scoring methods. *Psychophysiology*, *45*, 250–274. doi: 10.1111/j.1469-8986.2007.00618.x

Kihara, K., Kawahara, J.-I., & Takeda, Y. (2008). Electrophysiological evidence for independent consolidation of multiple targets. *NeuroReport*, *19*, 1493–1496. doi: 10.1097/WNR.0b013e32830fe4e8

Kranczioch, C., Debener, S., & Engel, A. K. (2003). Event-related brain potential correlates of the attentional blink phenomenon. *Cognitive Brain Research*, *17*, 177–187. doi: 10.1016/S0926-6410(03)00092-2

Kranczioch, C., Debener, S., Schwarzbach, J., Goebel, R., & Engel, A. K. (2005). Neural correlates of conscious perception in the attentional blink. *NeuroImage*, *24*, 704–714. doi: 10.1016/j.neuroimage.2004.09.024

Lagroix, H. E. P., Grubert, A., Spalek, T. M., Di Lollo, V., & Eimer, M. (2015). Visual search is postponed during the period of the AB: An event-related potential study. *Psychophysiology*, *52*, 1031–1038. doi: 10.1111/psyp.12435

Lamme, V. A. F., & Roelfsema, P. R. (2000). The distinct modes of vision offered by feedforward and recurrent processing. *Trends in Neuroscience*, *23*, 571–579. doi: 10.1016/S0166-2236(00)01657-X

Marcantoni, W. S., Lepage, M., Beaudoin, G., Bourgouin, P., & Richer, F. (2003). Neural correlates of dual task interference in rapid visual streams: An fMRI study. *Brain and Cognition*, *53*, 318–321. doi: 10.1016/S0278-2626(03)00134-9

Marois, R., Chun, M. M., & Gore, J. C. (2000). Neural correlates of the attentional blink. *Neuron*, *28*, 299–308. doi: 10.1016/S0896-6273(00)00104-5

Marois, R., & Ivanoff, J. (2005). Capacity limits of information processing in the brain. *Trends in Cognitive Sciences*, *9*, 296–305. doi: 10.1016/j.tics.2005.04.010

Marois, R., Yi, D. J., & Chun, M. M. (2004). The neural fate of consciously perceived and missed events in the attentional blink. *Neuron*, *41*, 465–472. doi: 10.1016/S0896-6273(04)00012-1

Martens, S., Munneke, J., Smid, H., & Johnson, A. (2006). Quick minds don't blink: Electrophysiological correlates of individual differences in attentional selection. *Journal of Cognitive Neuroscience*, *18*, 1423–1438. doi: 10.1162/jocn.2006.18.9.1423

Martens, S., & Wyble, B. (2010). The attentional blink: Past, present, and future of a blind spot in perceptual awareness. *Neuroscience and Biobehavioral Reviews*, *34*, 947–957. doi: 10.1016/j.neubiorev.2009.12.005

Nakayama, K., & Mackeben, M. (1989). Sustained and transient components of focal visual attention. *Vision Research*, *29*, 1631–1647. doi: 10.1016/0042-6989(89)90144-2

Niedeggen, M., Hesselman, G., Sharaie, A., Milders, M., & Blakemore, C. (2004). Probing the prerequisites for motion blindness. *Journal of Cognitive Neuroscience*, *16*, 584–597. doi: 10.1162/089892904323057317

Olivers, C. N. L., Hilkenmeier, F., & Scharlau, I. (2011). Prior entry explains order reversals in the attentional blink. *Attention, Perception, & Psychophysics*, *73*, 56–67. doi: 10.3758/s13414-010-0004-7

Olivers, C. N. L., & Meeter, M. (2008). A boost and bounce theory of temporal attention. *Psychological Review*, *115*, 836–863. doi: 10.1037/a0013395

Polich, J. (2003). Overview of P3a and P3b. In J. Polich (Ed.), *Detection of change: Event-related potential and fMRI findings* (pp. 83–98). Boston, MA: Kluwer.

Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. *Clinical Neurophysiology*, *118*, 2128–2148. doi: 10.1016/j.clinph.2007.04.019

Potter, M. C. (1976). Short-term conceptual memory for pictures. *Journal of Experimental Psychology: Human Learning and Memory*, *2*, 509–522. doi: 10.1037/0278-7393.2.5.509

Potter, M. C., Chun, M. M., Banks, B. S., & Muckenhoupt, M. (1998). Two attentional deficits in serial target search: The attentional blink and an amodal task-switch deficit. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *24*, 979–992. doi: 10.1037/0278-7393.24.4.979

Potter, M. C., Dell'Acqua, R., Pesciarelli, F., Job, R., Peressotti, F., & O'Connor, D. H. (2005). Bidirectional semantic priming in the attentional blink. *Psychonomic Bulletin & Review*, *12*, 460–465. doi: 10.3758/BF03193788

Potts, G. F. (2004). An ERP index of task relevance evaluation of visual stimuli. *Brain and Cognition*, *56*, 5–31. doi: 10.1016/j.bandc.2004.03.006

Prada, L., Barceló, F., Herrmann, C. S., & Escera, C. (2014). EEG delta oscillations index inhibitory control of contextual novelty to both irrelevant distracters and relevant task-switch cues. *Psychophysiology*, *51*, 658–672. doi: 10.1111/psyp.12210

Ptito, A., Arnell, K. M., Jolicœur, P., & MacLeod, J. (2008). Intramodal and crossmodal processing delays in the attentional blink paradigm revealed by event-related potentials. *Psychophysiology*, *45*, 794–803. doi: 10.1111/j.1469-8986.2008.00677.x

Ranganath, C., & Rainer, G. (2003). Neural mechanisms for detecting and remembering novel events. *Nature Reviews Neuroscience*, *4*, 193–202. doi: 10.1038/nrn1052

Raymond, J. E., Shapiro, K. L., & Arnell, K. M. (1992). Temporary suppression of visual processing in an RSVP task: An attentional blink? *Journal of Experimental Psychology: Human Perception and Performance*, *18*, 849–860. doi: 10.1037/0096-1523.18.3.849

Reeves, A., & Sperling, G. (1986). Attention gating in short-term visual memory. *Psychological Review*, *93*, 180–206. doi: 10.1037/0033-295X.93.2.180

Scalf, P. E., Dux, P. E., & Marois, R. (2011). Working memory encoding delays top–down attention to visual cortex. *Journal of Cognitive Neuroscience*, *23*, 2593–2604. doi: 10.1162/jocn.2011.21621

Sessa, P., Luria, R., Verleger, R., & Dell'Acqua, R. (2007). P3 latency shifts in the attentional blink: Further evidence for second target processing postponement. *Brain Research*, *1137*, 131–139. doi: 10.1016/j.brainres.2006.12.066

Smid, H. G. O. M., Jakob, A., & Heinze, H. J. (1999). An event-related brain potential study of visual selective attention to conjunctions of color and shape. *Psychophysiology*, *36*, 264–279. doi: 10.1017/S0048577299971135

Smulders, F. T. Y. (2010). Simplifying jackknifing of ERPs and getting more out of it: Retrieving estimates of participants' latencies. *Psychophysiology*, *47*, 387–392. doi: 10.1111/j.1469-8986.2009.00934.x

Sperling, G., Budiansky, J., Spivak, J. G., & Johnson, M. C. (1971). Extremely rapid visual search: The maximum rate of scanning letters for the presence of a numeral. *Science*, *174*, 307–311. doi: 10.1126/science.174.4006.307

Taatgen, N. A., Juvina, I., Schipper, M., Borst, J. P., & Martens, S. (2009). Too much control can hurt: A threaded cognition model of the attentional blink. *Cognitive Psychology*, *59*, 1–29. doi: 10.1016/j.cogpsych.2008.12.002

Todd, J. J., & Marois, R. (2004). Capacity limit of visual short-term memory in human posterior parietal cortex. *Nature*, *428*, 751–754. doi: 10.1038/nature02466

Ulrich, R., & Miller, J. (2001). Using the jackknife-based scoring method for measuring LRP onset effects in factorial designs. *Psychophysiology*, *38*, 816–827. doi: 10.1111/1469-8986.3850816

Verleger, R. (1988). Event-related potentials and cognition: A critique of the context updating hypothesis and an alternative interpretation of the P3. *Behavioral and Brain Sciences*, *11*, 343–427. doi: 10.1017/S0140525X00058015

Vogel, E. K., & Luck, S. J. (2002). Delayed working memory consolidation during the attentional blink. *Psychonomic Bulletin & Review*, *9*, 739–743. doi: 10.3758/BF03196329

Vogel, E. K., Luck, S. J., & Shapiro, K. L. (1998). Electrophysiological evidence for a postperceptual locus of suppression during the attentional blink. *Journal of Experimental Psychology: Human Perception and Performance*, *24*, 1656–1674. doi: 10.1037/0096-1523.24.6.1656

Wyble, B., Potter, M. C., Bowman, H., & Nieuwenstein, M. (2011). Attentional episodes in visual perception. *Journal of Experimental Psychology: General*, *140*, 488–505. doi: 10.1037/a0023612

Xu, Y., & Chun, M. M. (2006). Dissociable neural mechanisms supporting visual short-term memory for objects. *Nature*, *440*, 91–95. doi: 10.1038/nature04262

Yantis, S., Schwarzbach, J., Serences, J. T., Carlson, R. L., Steinmetz, M. A., Pekar, J. J., & Courtney, S. M. (2002). Transient neural activity in human parietal cortex during spatial attention shifts. *Nature Neuroscience*, *5*, 995–1002. doi: 10.1038/nn921

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