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The Attentional Blink Modulates Activity in the Early Visual Cortex

Grit Hein[1,2,3], Arjen Alink[2,4], Andreas Kleinschmidt[5,6], and Notger G. Müller[1,2]

Abstract

■ The attentional blink (AB) documents a particularly strong case of visual attentional competition, in which subjects' ability to identify a second target (T2) is significantly impaired when it is presented with a short SOA after a first target (T1). We used functional magnetic resonance imaging to investigate the impact of the AB on visual activity in individually defined retinotopic representations of the target stimuli. Our results show reduction of neural response in V3 and marginally in V2 and V1, paralleling the behavioral AB effect. Reduction of visual activity was accompanied by reduced neural response in the inferior parietal cortex. This indicates that attentional competition modulates activity in higher-order parietal regions and the early visual cortex, providing a plausible neural basis of the behavioral AB effect. ■

INTRODUCTION

There is compelling evidence that simultaneous visual events often compete to be processed (e.g., Kastner, De Weerd, Desimone, & Ungerleider, 1998; Duncan, 1984; Broadbent, 1958). A particularly strong case of visual attentional competition is documented in attentional blink (AB) experiments. Here, subjects' ability to identify a second target (T2) is significantly impaired when it is presented with a short target to target stimulus onset asynchrony (SOA) after a first target (T1) (Raymond, Shapiro, & Arnell, 1992). The behavioral AB is known to be stronger and more robust within the visual modality than, for example, across modalities (Hein, Parr, & Duncan, 2006; Arnell & Jenkins, 2004; Soto-Faraco & Spence, 2002; Duncan, Martens, & Ward, 1997). Within the visual modality, it is more pronounced for targets which share similar features than for targets with different visual properties (Awh et al., 2004; Chun & Potter, 1995; Raymond, Shapiro, & Arnell, 1995). These observations suggest that the AB might be associated with modulation of neural responses as early as in modality-specific sensory regions, and not only in higher-order brain areas as reported previously.

Previous functional magnetic resonance imaging (fMRI) studies investigated the neural correlates of the AB with letters (Feinstein, Stein, Castillo, & Paulus, 2004; Kranczioch, Debener, Schwarzbach, Goebel, & Engel, 2004; Marcantoni, Lepage, Beaudoin, Bourgouin, & Richer, 2003; Marois, Chun, & Gore, 2000) or scenes and faces as targets (Shapiro, Johnston, Vogels, Zaman, & Roberts, 2007; Marois, Yi, & Chun, 2004). According to these studies, T2 misses correlate with reduced activity in fronto-parietal areas. In line with these findings, electrophysiological (EEG) results showed modulation of the amodal P300 (Kranczioch, Debener, Maye, & Engel, 2007; Martens, Elmallah, London, & Johnson, 2006; Martens, Munneke, Smid, & Johnson, 2006; Sergent, Baillet, & Dehaene, 2005; Kranczioch, Debener, & Engel, 2003; Vogel & Luck, 2002; Vogel, Luck, & Shapiro, 1998). Marois and Ivanoff (2005) reviewed the results of fMRI studies with different attention paradigms, such as the AB, the Psychological Refractory Period (PRP), visual short-term memory (VSTM), and multiple object tracking (MOT) tasks. They conclude that effects in the inferior parietal cortex are more specific for attentional competition between visual targets, as for example in the visual AB, whereas similar effects in the dorsolateral prefrontal cortex (DLPFC) are rather unspecific and found in a variety of attention paradigms (Marois & Ivanoff, 2005, see also Hein, Alink, Kleinschmidt, & Müller, 2007). So far, the analysis of AB effects in the visual cortex has been focused on higher-order visual regions in the occipito-temporal cortex, involved in the processing of letters (Kranzioch et al., 2004) and scenes (Shapiro et al., 2007; Marois et al., 2004). There is evidence for reduced activity in the parahippocampal place area for T2 misses compared to T2 hits (Marois et al., 2004; but see Shapiro et al., 2007; Kranzioch et al., 2004).

Attentional modulation of activity in the fronto-parietal cortex and higher-order visual regions in the AB is in line

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with the findings from visual attention studies using other paradigms (reviewed in Kastner & Ungerleider, 2000). However, the results from various human imaging studies and single-cell experiments indicate that visual attentional competition is also associated with modulation of activity in retinotopically organized early visual cortices (e.g., Beck & Kastner, 2005; reviewed in Kastner & Ungerleider, 2000). Within the visual hierarchy, attention effects seem to be strongest at more advanced processing levels as V3 or V4 (Kastner & Ungerleider, 2000; Kastner et al., 1998), but have also been found in as early stages as V1 (e.g., Müller & Kleinschmidt, 2003, 2004, 2007; Jack, Shulman, Snyder, McAvoy, & Corbetta, 2006; Müller, Bartelt, Donner, Villringer, & Brandt, 2003; Ress & Heeger, 2003; Saenz, Buracas, & Boyton, 2002; Martinez et al., 2001; Ress, Backus, & Heeger, 2000; Brefczynski & DeYoe, 1999; Somers, Dale, Seiffert, & Tootell, 1999) and even in the lateral geniculate nucleus (O'Connor, Fukui, Pinsk, & Kastner, 2002). In the context of these findings, it is plausible to assume that, in addition to fronto-parietal effects, the strong attentional competition in the visual AB might also induce activity modulations in retinotopic regions. So far, it is unknown whether the visual AB is related to modulation of activity in the early visual cortex, because the design of previous AB studies did not permit to investigate AB effects in retinotopic regions.

In our study, we therefore examined the impact of the AB effect on activity in retinotopic areas V1, V2, and V3. We mapped retinotopic regions in individual subjects and developed a new AB paradigm with stimuli that are known to elicit robust activation in early visual regions (see Jack et al., 2006; Ress & Heeger, 2003; Ress et al., 2000). Subjects identified horizontal and vertical gratings, tilted clockwise or counterclockwise (Figure 1A). Based on previous behavioral experiments (Hein et al., 2006), we predicted that these simple stimuli should elicit a robust behavioral AB effect. On the neural level, we expected an impairment of T2 identification in the AB to be reflected in neural activity in the early visual cortex. Moreover, we expected that, as in studies on spatial attention, this effect would decrease from intermediate areas as V3 to an early processing level as V1 (Kastner & Ungerleider, 2000; Kastner et al., 1998). Finally, we expected to confirm previous findings, associating the AB with activity in the DLPFC and the inferior parietal cortex (Kranczioch et al., 2004; Marois et al., 2000, 2004).

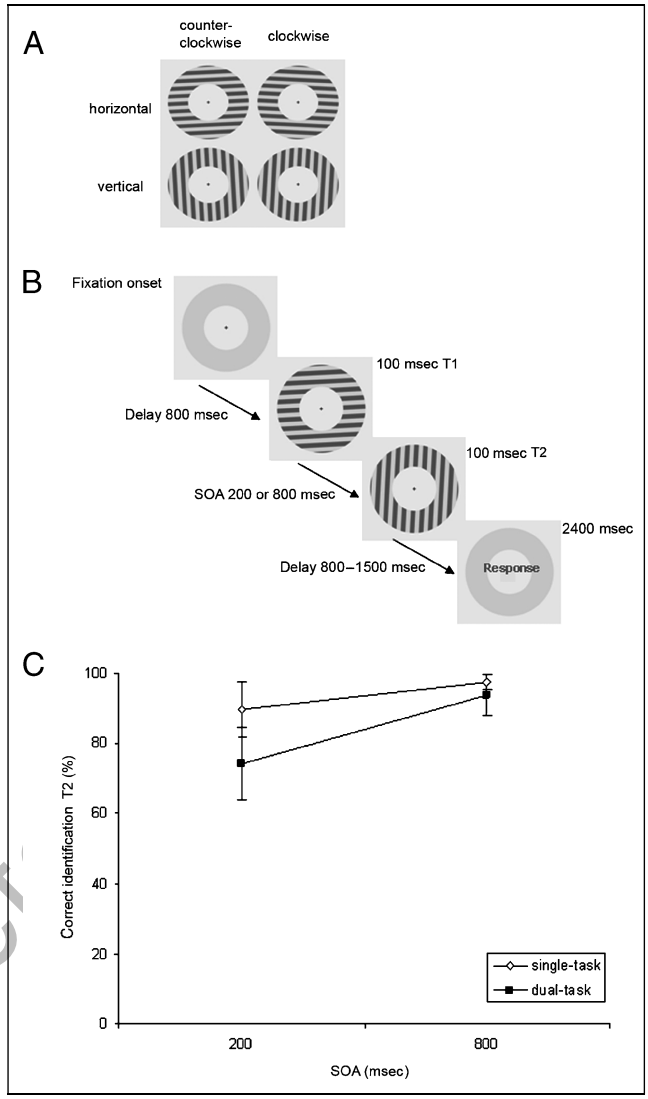

Figure 1. (A) Horizontal and vertical gratings tilted clockwise or counterclockwise off fixation presented as first target (T1) and second target (T2). (B) Example trial. SOA from T1 to T2 was 200 or 800 msec. Unspeeded responses were given in a 2.4-sec response period at the end of the trial. (C) Behavioral data for T2 performance from fMRI session, showing a behavioral attentional blink. Calculation of T2 performance scores in dual-task blocks was based on trials with a correct response to T1. Behavioral performance scores in single-task blocks are shown for trials in which the relevant target was presented as T2. Error bars indicate standard errors.

METHODS

Participants

Fifteen subjects participated in an fMRI session and a preceding behavioral training outside the scanner. The reported data are based on 11 subjects because we had to discard four datasets—two because of an error rate of >50% in the behavioral data recorded in the fMRI session, two because of massive drifts in the raw data fMRI signal. All participants gave informed consent in accordance with the ethic committee of the Johann Wolfgang Goethe University Hospital, Frankfurt, Germany.

Stimuli

The target patterns were dark gray horizontal or vertical gratings, tilted 0.4° clockwise (cw) or counterclockwise (ccw) off fixation. They were presented for 100 msec in a light gray annulus around the fixation cross (inner

circle, 4°; outer circle, 8°; Figure 1A). We chose an annulus instead of a circle because of the difficulty to separate visual subareas representing the foveal region. The stimuli were presented using an MR-compatible goggle system with two organic light-emitting diode displays (MR Vision 2000, Resonance Technology, Northridge, CA). The screen had a width of 30° and a height of 22.5°.

Procedure

Each trial lasted 5 sec (Figure 1B). Trials began with onset of the fixation screen, showing the annulus and the fixation cross without a target pattern at screen center. In ''null'' trials, this frame was maintained throughout the entire trial. In all other trials, a first target (T1) pattern was presented on the annulus 800 msec after trial onset. A second target pattern (T2) occurred randomly at 200 or 800 msec from onset of T1, yielding T1–T2 SOAs of 200 and 800 msec. Each orientation (vertical/horizontal) and rotation (cw/ccw) was assigned equally often to T1 and T2. They occurred in random combinations, except that there was always one horizontal and one vertical target in each trial. Subjects used a four-key button box to indicate how the vertical or horizontal gratings in T1 and T2 were tilted (cw or ccw), irrespective of the order of presentation. Responses to the horizontal grating were given with the left hand (left index = tilted cw; left middle = tilted ccw), responses to the vertical target pattern with the right hand (right index = tilted cw; right middle = tilted ccw). To ensure unspeeded responses, subjects were instructed to respond only during a 2.4-sec response window at the end of the trial, marked by the word ''Response'' (in German) (Figure 1B).

In our AB paradigm, we deliberately decided against backward masking of T2, which was used in previous AB studies (Kranczioch et al., 2004; Marois et al., 2000, 2004). The mere presence of a backward mask is known to modulate activity in the visual cortex, independently of any attention manipulation (Green et al., 2005; Noguchi & Kakigi, 2005). Because this would make it difficult to interpret potential AB effects in the visual cortex, we used unmasked targets inspired by previous behavioral work (Hein et al., 2006).

The experiment consisted of dual-task and single-task blocks and a passive control condition. In dual-task blocks, subjects responded to both targets. In single-task blocks, subjects focused only on vertical or only on horizontal target patterns, which occurred equally often as T1 or as T2. In the control condition, they passively viewed the stimuli, which enabled us to analyze the impact of stimulation independently of the attention manipulation. The fMRI session was preceded by behavioral training including two single-task blocks (one vertical, one horizontal) and one dual-task block (32 trials each) with feedback. Inside the scanner, subjects again performed 10 trials per condition (single task_vertical; single task_horizontal; dual task) with feedback to get used to the button box. The fMRI main experiment included two single-task blocks (one vertical, one horizontal), two dual-task blocks, and one block of the passive control condition. The order of the blocks was counterbalanced across subjects. Each block had 100 trials (20 null trials, 20 short SOA trials T1 vertical/T2 horizontal, 20 short SOA trials T1 horizontal/T2 vertical, 20 long SOA trials T1 horizontal/T2 vertical, 20 long SOA trials T1 vertical/T2 horizontal), presented in random order. After the experimental blocks, retinotopic visual areas were determined individually for each subject (see below).

MRI Data Acquisition and Processing

Subjects were scanned on a 3-T Siemens Magnetom Allegra scanner with a standard head coil. A gradient-recalled echo-planar imaging (EPI) sequence was used with the following parameters: 36 slices; TR = 2000 msec; TE = 30 msec; FOV = 192 mm; in-plane resolution = 3 × 3 mm^2; slice thickness = 3 mm; gap thickness = 0.3 mm; 303 scans were acquired per run, including two dummy scans to allow T1 equilibration at the start of each run. To maximize the quality of the EPI images, we ran an additional 30 sec sequence before each run. In this sequence, we used a point-spread function to estimate the disturbance of the magnetic field. The parameters determined by this point-spread function were then applied to correct the EPI images acquired in the following run (e.g., Zaitsev, Hennig, & Speck, 2004). After functional scanning, we acquired for each subject high-resolution anatomical images using a magnetization-prepared rapid-acquisition gradient-echo (MP-RAGE) sequence (TR = 2300 msec; TE = 3.49 msec; FA = 12°; matrix = 256 × 256; voxel size 1.0 × 1.0 × 1.0 mm^3).

All fMRI data were processed and analyzed using Brain Voyager QX software (Brain innovation, Maastricht, The Netherlands). Standard preprocessing was conducted comprising three-dimensional motion correction using the Levenberg–Marquardt algorithm, linear-trend removal, and temporal high-pass filtering at 0.0054 Hz and slice timing correction. The preprocessed data were then analyzed with a deconvolution approach (Glover, 1999). We estimated the effect size for each condition in 10 time bins of 2 sec each, whereby the first time bin represented the onset of the trial. Time courses of activity (Figures 2 and 3) show group-averaged beta values in 7 of the 10 time bins, covering a time window of 14 sec after trial onset.

Retinotopic Mapping and Regions of Interest

Time courses of BOLD responses for correct dual-task trials, for dual-task trials in which T2 was incorrect (but T1 was correct), and for single-task trials with a correct response to the task-relevant target were extracted

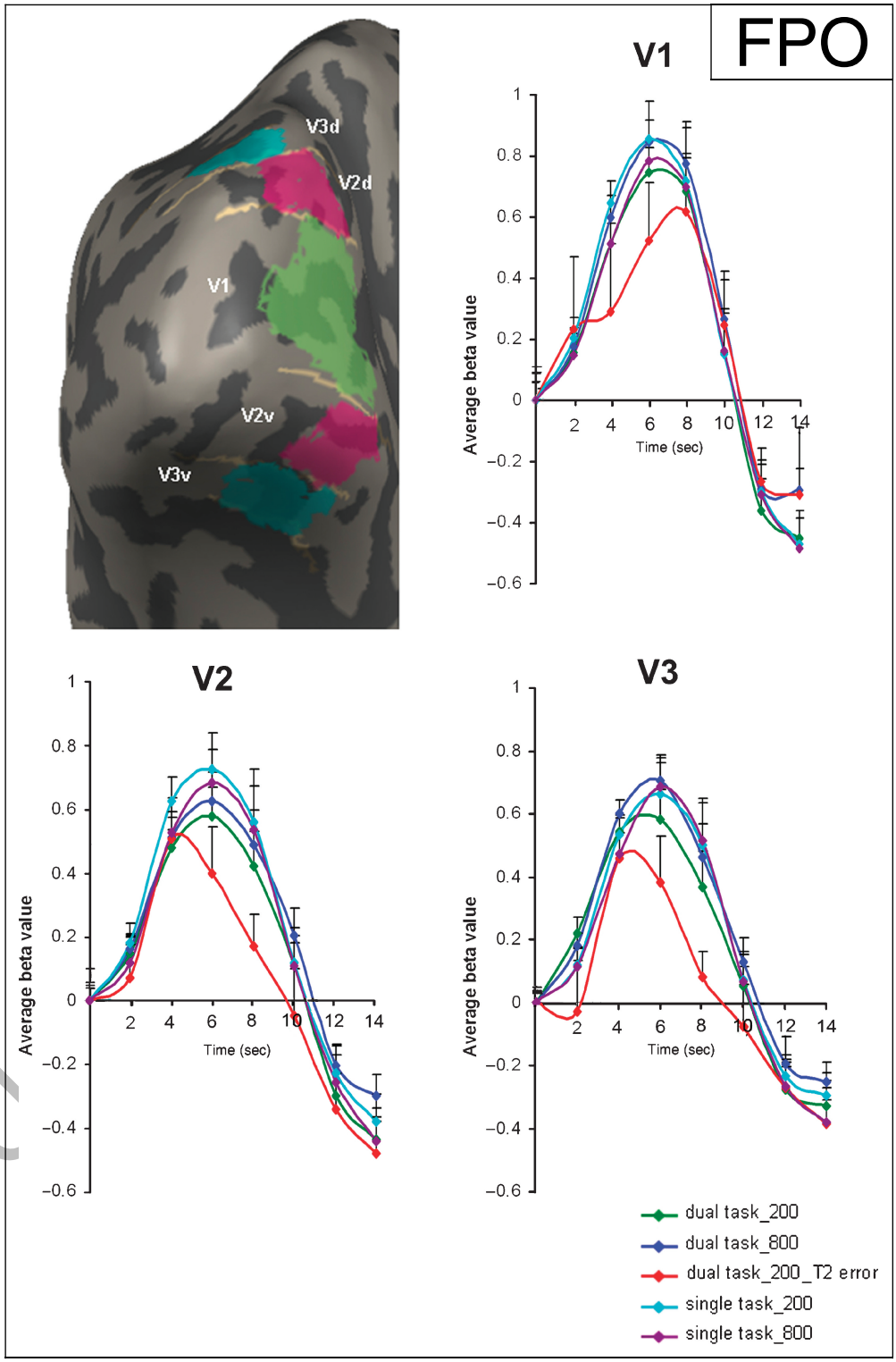

Figure 2. Results in the visual cortex. Retinotopic regions in the left hemisphere of a representative subject and time courses of averaged beta values for correct dual-task trials with short and long SOA (green and dark blue, respectively), dual-task trials with short SOA and T2 errors (red), correct single-task trials with short and long SOA (light blue and purple, respectively). v = ventral; d = dorsal; dual task_200 = correct dual-task trials with short SOA; dual task_800 = correct dual-task trials with long SOA; dual task_200_T2 error = dual-task trials with short SOA and correct T1, but incorrect T2; single task_200 = correct single-task trials with short SOA; single task_800 = correct single-task trials with long SOA. Error bars indicate standard errors.

from individual ROI in retinotopic visual areas, using all voxels of the cluster. Additionally, we extracted time courses of activity for trials with short and long SOA of the passive viewing control condition. Similar to previous studies (Müller et al., 2003; Saenz et al., 2002; Ress et al., 2000), ROIs were mapped separately by 8 Hz black-and-white checkerboard stimulation at the corresponding locations and subdivided according to retinotopic boundaries (bilateral ventral and dorsal visual areas V2 and V3; bilateral V1) that were again separately mapped by checkerboard stimulation along the horizontal and vertical meridians (Sereno et al., 1995). Additional eccentricity mapping was used to control that the cortical patches within each visual area corresponded to the

retinotopic representation of the annulus. The visual ROIs were marked on the reconstructed and inflated cortical surface of each subject. Figure 2 shows the retinotopic visual regions in the left hemisphere of one representative subject.

Further, we examined time courses of activity in frontal and parietal regions. Frontal and parietal ROIs were defined based on the group contrast of all correct dual-task and single-task trials (both SOAs) with the null trial baseline (Figure 3; Table 1). From these frontal and parietal ROIs, time courses were then extracted for individual subjects.

Statistical Data Analysis

Behavioral data collected during fMRI scanning were analyzed with SPSS software. Performance scores were assessed for T1 and T2 for each SOA in single-task and dual-task blocks, for the latter independently of the order of responses. Calculation of T2 performance scores in dual-task blocks was based on trials with a correct response to T1. Behavioral performance scores in single-task blocks were assessed separately for T1 and T2. Mean accuracy scores were then submitted to an analysis of variance (ANOVA) including condition (single task vs. dual task) and SOA.

For statistical analysis of imaging data, we first extracted beta values of time courses of activation for each experimental condition in each subject in each ROI. Secondly, we determined the peak of activation for each condition in each subject by averaging over time bins 2, 3, and 4, which correspond to a time interval of 4 to 8 sec after trial onset. These individual values of peak activation for each experimental condition were then

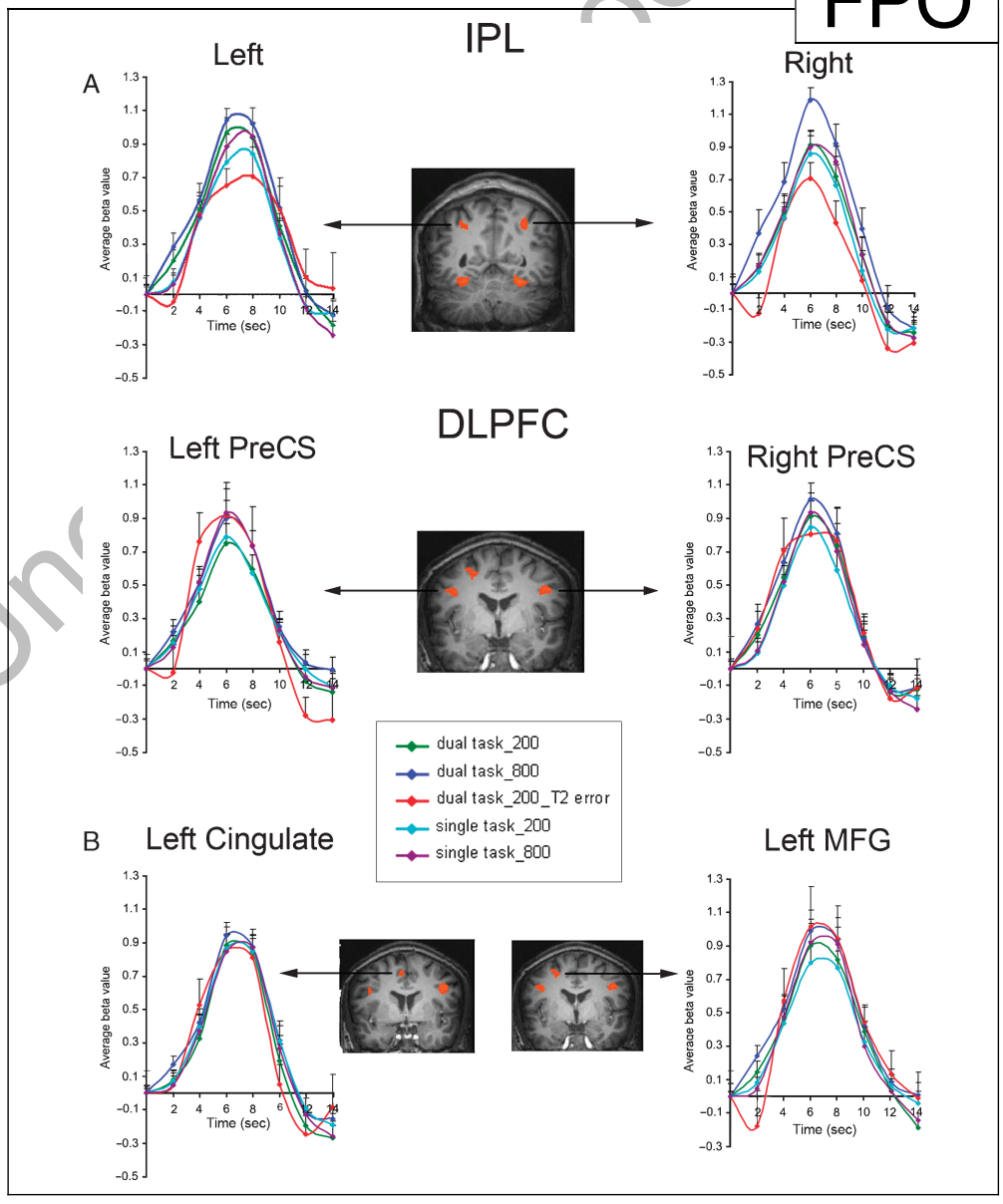

Figure 3. Results in the frontal and parietal cortex. Time courses of averaged beta values for correct dual-task trials with short and long SOA (green and dark blue, respectively), dual-task trials with short SOA and T2 errors (red), correct single-task trials with short and long SOA (light blue and purple, respectively) in ROIs in the frontal and parietal cortex (see Table 1 for details). IPL = inferior parietal lobe; DLPFC = dorsolateral prefrontal cortex; PreCS = precentral sulcus; MFG = middle frontal gyrus; dual task_200 = correct dual-task trials with short SOA; dual task_800 = correct dual-task trials with long SOA; dual task_200_T2 error = dual-task trials with short SOA and correct T1, but incorrect T2; single task_200 = correct single-task trials with short SOA; single task_800 = correct single-task trials with long SOA. Error bars indicate standard errors.

Table 1. Brain Regions Activated by Correct Dual- and Single-task Trials with Short and Long SOA

	Talairach Coordinates				
Brain Region	*x*	*y*	*Z*	*Mean t Value*	*Voxel*
Left inferior parietal lobe[a]	−28	−56	36	3.8	449
Right inferior parietal lobe[a]	28	−61	34	3.9	1119
Left frontal lobe/precentral sulcus[a]	−42	−1	29	3.8	389
Right frontal lobe/precentral sulcus[a]	44	0	31	4	934
Left frontal lobe/middle frontal gyrus[a]	−26	−6	49	3.8	336
Left frontal lobe/cingulate[a]	−6	4	46	3.9	367
Left occipital lobe/inferior temporal gyrus	−41	−68	−3	3.9	502
Left occipital lobe/precuneus	−27	−67	23	4.12	718
Left occipital lobe/lingual gyrus	−2	−76	−3	4.49	20078
Right claustrum	31	16	7	3.8	145
Left claustrum	−30	16	10	3.87	145

The Talairach coordinates indicate the center of mass significantly activated ($q < 0.05$, FDR corrected); cluster threshold > 100.
[a]Clusters used as ROIs.

submitted to ANOVAs. For visualization, we determined the average of beta values across participants for the experimental conditions, which are shown Figure 2 (see Jack et al., 2006). In V2 and V3, individual subjects' data for bilateral ventral and dorsal ROIs, in V1 the data of bilateral ROIs (see the Retinotopic mapping and ROIs section above) was collapsed, because we did not have a quadrant-related hypothesis for retinotopic areas.

The analysis of time course of activity in frontal and parietal ROIs was identical, but calculated separately for the left and right hemispheres, taking into account potential hemispheric differences in attention-related activity (e.g., Corbetta & Shulman, 2002). The average of frontal and parietal time courses of activity across all subjects is plotted in Figure 3.

RESULTS

Behavioral Results

T1 Performance

T1 performance in dual-task trials did not vary with SOA, but was lower than in single-task trials in which T1 was the relevant target [SOA: $F(1, 10) = 1.4, p > .2$; Condition × SOA: $F(1, 10) < 1$; Condition: $F(1, 10) = 7.6, p < .03$; dual-task trials, T1 mean % correct short SOA = 82.7, $SD = 13.4$; T1 mean % correct long SOA = 86.5, $SD = 13.7$; single-task trials, T1 mean % correct short SOA = 87.5, $SD = 9.4$; T1 mean % correct long SOA = 93.1, $SD = 7.5$]. There was no significant effect of SOA on T1 performance in single-task trials [$t(10) = -1.8, p > .08$]. In dual-task trials with short SOA, T1 performance was significantly higher than T2 performance [$t(10) = 2.6, p < .03$]. There was no significant difference in T1 and T2 performance in dual-task trials with long SOA [$t(10) = -1.9, p > .1$] and in single-task trials [T1 relevant vs. T2 relevant, short SOA, $t(10) < 1$, long SOA, $t(10) = -2.1, p > .07$].

T2 Performance

Figure 1C shows T2 performance in dual-task blocks and in single-task trials with T2 as the relevant target. Performance improved with increasing SOA and was significantly lower when subjects were required to identify T1 and T2 than when they only identified T2 [Condition × SOA: $F(1, 10) = 5.5, p < .05$; SOA: $F(1, 10) = 20.8, p < .01$; Condition: $F(1, 10) = 9.5, p < .02$]. These two results are characteristics of the AB and replicate the behavioral findings of previous studies (Hein et al., 2006; Kranczioch et al., 2004; Marois et al., 2004; Raymond et al., 1992). In a first step, we investigated whether the AB is reflected in modulation of neural activity related to retinotopic target representations in early visual areas. In a second step, we tested for neural correlates of the AB in fronto-parietal regions.

Imaging Results

Visual Cortex

Figure 2 shows the time courses of BOLD activity in V1, V2, and V3 for dual-task trials with short SOA and long SOA, in which subjects successfully identified T1 and T2 (green and dark blue, respectively), and for dual-task trials with short SOA and correct T1, but incorrect T2

responses (red). Because of the very small number of T2 errors in dual-task trials with long SOA, we refrained from extracting time courses for this condition. Moreover, we extracted the time courses of activity for single-task trials with short and long SOA (light blue and purple, respectively), in which subjects correctly responded to the task relevant target. Because there was no behavioral difference between T1 and T2 performance in single-task trials (see above) and no difference in neural activity between single-task trials with correct responses to T1 and T2 ($p > .05$), we pooled the data from these conditions.

In V3, the amplitude of the BOLD response in dual-task trials with short SOA and T2 errors was significantly smaller than in short SOA trials with successful T2 identification [$F(1, 10) = 7.3$, $p < .03$] and long SOA trials with T2 hits [$F(1, 10) = 10.6$, $p < .01$]. Activity for single-task trials and correct dual-task trials was not affected by SOA [single task: $F(1, 10) < 1$; dual task: $F(1, 10) = 2.6$, $p > .13$].

In V2, activity in dual-task trials with T2 errors was reduced compared to correct dual-task trials with short SOA, but the effect was weaker than in V3 [$F(1, 10) = 4.2$, $p = .068$]. Similar to V3, there was no significant difference between single-task trials and correct dual-task trials with short and long SOA [single task: $F(1, 10) = 1.5$, $p > .2$; dual task: $F(1, 10) < 1$].

The results in V1 resembled the pattern of results in V2. Again, there was a trend for reduced activity in dual-task trials with T2 errors compared to correct dual-task trials with short SOA [$F(1, 10) = 4.5$, $p = .058$], and no significant effects in single-task trials and correct dual-task trials [single task: $F(1, 10) < 1$; dual task: $F(1, 10) = 1.6$, $p > .2$].

Additionally, we analyzed time courses of BOLD responses for trials with short and long SOA in the passive viewing control condition. The results showed no significant effect of SOA, thus rendering unlikely the possibility that the dual-task effects in V3, V2, and V1 were merely caused by the difference in temporal structure of physical input between the trial types [all $F(1, 10) < 1$].

Taken together, these results indicate that the behavioral AB effect is associated with reduced activity in retinotopic early visual regions, most strongly in V3.

Fronto-parietal Cortex

Previous AB studies showed modulation of activity in the frontal cortex (precentral regions, cingulate gyrus) and in the inferior parietal lobe (IPL) (Kranzcioch et al., 2004; Marois et al., 2004; Marcantoni et al., 2003). In a next step, we investigated AB effects in the fronto-parietal cortex. To define fronto-parietal ROIs, we contrasted correct dual-task and single-task trials with short and long SOA with ''null'' trial baseline activity. This contrast revealed robust clusters of activity in the DLPFC along the precentral sulcus (PreCS), in the middle frontal gyrus (MFG), in the cingulate cortex, and in the bilateral IPL (Figure 3; Table 1), which were then used as ROIs.

The main result was significant reduction of activity for dual-task trials with short SOA and T2 errors in the inferior parietal cortex [left IPL: $F(1, 10) = 6.9$, $p < .03$; right IPL: $F(1, 10) = 6.7$, $p < .03$; Figure 3, top]. The left IPL showed no significant effects in single-task trials and correct dual-task trials [single task: $F(1, 10) = 2.2$, $p > .16$; dual task: $F(1, 10) = 3.2$, $p > .1$]. In the right IPL, activity in dual-task trials with long SOA was significantly stronger than in correct dual-task trials with short SOA, possibly indicating stronger attentional competition at short SOA even if T2 was identified correctly [$F(1, 10) = 16.7$, $p < .003$].

There was no significant difference between T2 target hits and errors in the frontal cortex, right PreCS, left PreCS, left MFG, left cingulate [all $F(1, 10) < 1$; Figure 3, middle and bottom panels]. The left PreCS and the MFG showed a main effect of SOA, indicating reduced activity for short SOA trials under both dual-task and single-task conditions [left PreCS: $F(1, 10) = 18.7$, $p < .001$; MFG: $F(1, 10) = 8.8$, $p < .02$]. One possibility is that this result reflects the switch of attention from the horizontal to the vertical grating, which is harder at short SOA than at long SOA, and discussed as potential source of the AB effect (Potter, Chun, Banks, & Muckenhoupt, 1998). If the AB is based on task switching, the effect of SOA in dual-task trials should be stronger than in single-task trials, reflected in a significant interaction between SOA and condition (single vs. dual task). None of the fronto-parietal regions showed such a significant SOA × Condition interaction [left IPL, left PreCS, right PreCS, MFG: all $F(1, 10) < 1$; right IPL: $F(1, 10) = 3.1$, $p > .1$; cingulate: $F(1, 10) = 2$, $p > .18$]. Based on these results, a task switching explanation is unlikely. The effect of SOA in dorsolateral prefrontal regions might rather indicate increased competition between the visual stimuli, possibly driven by T2, which acts as mask for T1 in single- as well as dual-task trials, supported by previous findings (Marois et al., 2000).

DISCUSSION

The main result of our study is that the AB correlates with reduced activity in retinotopic visual regions. Reduction of activity in visual regions was strongest in V3, and to a smaller extent, was also found in V2 and V1. This finding is in line with previous studies, showing that the impact of attentional modulation in the visual cortex increases from early to late stages in visual processing (Kastner & Ungerleider, 2000; Kastner et al., 1998). The significant decrease of visual activity in dual-task trials with short SOA in which subjects failed to identify T2 paralleled the finding of impaired T2 identification at short SOA in the simultaneously collected behavioral

data. This significant behavioral AB effect was obtained without backward masking of T2, which is known to alleviate the AB effect (Vogel & Luck, 2002; Giesbrecht & Di Lollo, 1998). Our previous behavioral work has revealed AB effects without backward masking (Hein et al., 2006), but this previous study had continuous stimulation after T2, which may have acted as a mask by disrupting early sensory memory of the target. In our current experiment, T2's representation had to be very precise in order to make the required judgment. The observed AB probably reflects a fast loss of the iconic traces of these detailed representations, accounting for the observed behavioral deficit in dual-task trials. Single-task performance showed that, without attentional competition, the subjects were able to identify the targets with high accuracy, reflected in robust activation in all retinotopic regions for single-task trials.

Attentional modulation of visual activity as early as V1 has been shown in previous visual attention fMRI studies, for example, reduction of neural response for target misses (Jack et al., 2006; Ress & Heeger, 2003; Ress et al., 2000). In these previous studies, early visual responses to target misses were investigated based on detection of a single target presented with low background contrast. Low background contrast increases the perceptual difficulty, which might partly account for modulation of visual activity, at least in the higher-order visual cortex (Johnston, Shapiro, Vogels, & Roberts, 2007). Our results showed similar retinotopic effects with highly visible targets, which were missed because of attentional competition with another target. The findings of studies which combined fMRI and EEG imply that such attention effects in individual retinotopic regions are often missed in EEG signatures because EEG signals reflect the average of neural responses in a variety of different visual regions (e.g., Martinez et al., 2001). The results of our study indicate that the visual AB modulates activity in different retinotopic regions to a different extent. It is likely that the spatial resolution of the EEG signal is not sufficient to pick up these differences in retinotopic effects, which could explain why EEG studies on the visual AB failed to show modulation in early components (Sergent et al., 2005; Kranczioch et al., 2003; Vogel & Luck, 2002; Vogel et al., 1998). An alternative account is that effects in primary visual regions arise from delayed feedback from higher visual areas, as has been suggested for spatial attention effects (Noesselt et al., 2002; Martinez et al., 1999, 2001).

Although most behavioral findings indicate that the AB is most readily evoked if the task-relevant features for T1 and T2 are the same or similar (Hein et al., 2006; Awh et al., 2004; Duncan et al., 1997), it is fair to say the majority of imaging results in this domain point to limitations in central rather than lower-tier sensory capacity as the source of the AB (Kranczioch et al., 2004; Marois et al., 2000). In line with these findings, we found AB-related reduction of activity in the inferior parietal cortex. Human imaging results and monkey single-cell results indicate that the parietal lobe contains regions with predominately visual, and others with multimodal (amodal) characteristics, which can be hard to separate (Behrmann, Geng, & Shomstein, 2004). Accordingly, the observed effects in parietal lobe could either reflect modality-specific competition between visual targets, as proposed by Marois and Ivanoff (2005), or limitations in amodal parietal capacity, challenged by a variety of attention-related tasks (e.g., Corbetta & Shulman, 2002).

Exceeding previous results, our data show that early sensory processing is affected by the AB, and in fact, more so than other higher-order candidate areas in the frontal lobe. One first, possible interpretation of this result would be that sensory capacity limitations are the main source of the AB. With such an assumption, it would be difficult to integrate the results of previous AB studies, which showed evidence for a behavioral AB within the visual modality without modulation of visual activity (Shapiro et al., 2007; Kranczioch et al., 2004). Moreover, the magnitude of the AB effect in individual subjects has been shown to correlate with target-related activity in the right ventrolateral prefrontal cortex (Martens, Munneke, et al., 2006) and other frontal regions (Feinstein et al., 2004). This might support the assumption that the AB is solely based on limitations in amodal processing capacity. In this case, the observed modulation of visual activity would be driven by top–down influence of the frontal cortex, possibly together with parietal regions. Previous AB studies used complex stimuli such as faces, scenes, or letters as targets, which were mostly embedded in a stream of distractors (Martens, Munneke, et al., 2006; Feinstein et al., 2004; Kranczioch et al., 2004; Marois et al., 2000). These differences in design to our study might explain why they found strong modulations in frontal activity, whereas our results showed AB-related reduction of activity in retinotopic regions without comparable effects in multiple frontal areas. Thus, the low complexity of our stimuli might account for the absence of a frontal AB effect in this study. If the observed AB effects in retinotopic areas are related to top–down modulation, our results indicate that this is most likely driven by parietal regions.

Alternatively, our results could indicate both limitations in sensory-specific and amodal capacity. As other fMRI studies, our findings rely on sluggish hemodynamic responses and do not provide insights into the sequence of activations in different brain regions, which makes it hard to disentangle these two assumptions. Interesting complementary evidence is provided by the results of Gross et al. (2004), who investigated the AB with MEG and letters as targets. Gross et al. identified a network of frontal, parietal, and visual regions, which is involved in target letter identification, and showed that successful T2 identification depends on the temporal synchronization of neural responses in these amodal

and sensory-specific regions. The finding that amodal and sensory-specific regions need to be activated at the same time for successful target identification argues against a strict functional hierarchy (i.e., top–down modulation from the parietal to the visual cortex). It is more in line with the assumption that processing of visual targets in the AB paradigm requires the temporally synchronized support of sensory-specific and amodal regions. Attentional competition between visual targets, documented by the behavioral AB effect, might then be driven by both sensory-specific and amodal processing limitations.

We conclude that reduced sensory processing in retinotopic regions contributes to task failure in the visual AB, in addition to modulation of neural response in amodal parietal regions. This could be the neural basis for the well-known behavioral result of stronger and more robust AB effects between visual targets as compared to cross-modal designs (Arnell & Jenkins, 2004; Soto-Faraco & Spence, 2002; Duncan et al., 1997).

UNCITED REFERENCE

Shapiro, Schmitz, Martens, Hommel, & Schnitzler, 2006

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REFERENCES

Arnell, K. M., & Jenkins, R. (2004). Revisiting within-modality and cross-modality attentional blinks: Effects of target–distractor similarity. *Perception & Psychophysics, 66,* 1147–1161.

Awh, E., Serences, J., Laurey, P., Dhaliwal, H., van der Jagt, T., & Dassonville, P. (2004). Evidence against a central bottleneck during the attentional blink: Multiple channels for configural and featural processing. *Cognitive Psychology, 48,* 95–126.

Beck, D. M., & Kastner, S. (2005). Stimulus context modulates competition in human extrastriate cortex. *Nature Neuroscience, 8,* 1110–1116.

Behrmann, M., Geng, J. J., & Shomstein, S. (2004). Parietal cortex and attention. *Current Opinion in Neurobiology, 14,* 212–217.

Brefczynski, J. A., & DeYoe, E. A. (1999). A physiological correlate of the ''spotlight'' of visual attention. *Nature Neuroscience, 2,* 370–374.

Broadbent, D. E. (1958). *Perception and communication*. London: Pergamon.

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.

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience, 3,* 201–215.

Duncan, J. (1984). Selective attention and the organization of visual information. *Journal of Experimental Psychology: General, 113,* 501–517.

Duncan, J., Martens, S., & Ward, R. (1997). Restricted attentional capacity within but not between sensory modalities. *Nature, 387,* 808–810.

Feinstein, J. S., Stein, M. B., Castillo, G. N., & Paulus, M. P. (2004). From sensory processes to conscious perception. *Consciousness and Cognition, 13,* 323–335.

Giesbrecht, B., & Di Lollo, V. (1998). Beyond the attentional blink: Visual masking by object substitution. *Journal of Experimental Psychology: Human Perception and Performance, 24,* 1454–1466.

Glover, G. H. (1999). Deconvolution of impulse response in event-related BOLD fMRI. *Neuroimage, 9,* 416–429.

Green, M. F., Glahn, D., Engel, S. A., Nuechterlein, K. H., Sabb, F., Strojwas, M., et al. (2005). Regional brain activity associated with visual backward masking. *Journal of Cognitive Neuroscience, 17,* 13–23.

Gross, J., Schmitz, F., Schnitzler, I., Kessler, K., Shapiro, K., Hommel, B., et al. (2004). Modulation of long-range neural synchrony reflects temporal limitations of visual attention in humans. *Proceedings of the National Academy of Sciences, U.S.A., 101,* 13050–13055.

Hein, G., Alink, A., Kleinschmidt, A., & Müller, N. G. (2007). Neural competition between visual and auditory decisions. *PLOS ONE,* online.

Hein, G., Parr, A., & Duncan, J. (2006). Within-modality and cross-modality attentional blinks in a simple discrimination task. *Perception & Psychophysics, 68,* 54–61.

Jack, A. I., Shulman, G. L., Snyder, A. Z., McAvoy, M., & Corbetta, M. (2006). Separate modulations of human V1 associated with spatial attention and task structure. *Neuron, 51,* 137–147.

Johnston, S. J., Shapiro, K. L., Vogels, W., & Roberts, N. J. (2007). Imaging the attentional blink: Perceptual versus attentional limitations. *NeuroReport, 18,* 1475–1478.

Kastner, S., De Weerd, P., Desimone, R., & Ungerleider, L. G. (1998). Mechanisms of directed attention in the human extrastriate cortex as revealed by functional MRI. *Science, 282,* 108–111.

Kastner, S., & Ungerleider, L. G. (2000). Mechanisms of visual attention in the human cortex. *Annual Review of Neuroscience, 23,* 315–341.

Kranczioch, C., Debener, S., & Engel, A. K. (2003). Event-related potential correlates of the attentional blink phenomenon. *Cognitive Brain Research, 17,* 177–187.

Kranczioch, C., Debener, S., Maye, A., & Engel, A. K. (2007). Temporal dynamics of access to consciousness in the attentional blink. *Neuroimage, 37,* 947–955.

Kranczioch, C., Debener, S., Schwarzbach, J., Goebel, R., & Engel, A. K. (2004). Neural correlates of conscious perception in the attentional blink. *Neuroimage, 24,* 707–714.

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 & Cognition, 53,* 318–321.

Marois, R., Chun, M. M., & Gore, J. C. (2000). Neural correlates of the attentional blink. *Neuron, 28,* 299–308.
Marois, R., & Ivanoff, J. (2005). Capacity limits of information processing in the brain. *Trends in Cognitive Sciences, 9,* 296–305.
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.
Martens, S., Elmallah, K., London, R., & Johnson, A. (2006). Cuing and stimulus probability effects on the P3 and the AB. *Acta Psychologica, 123,* 204–218.
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.
Martinez, A., Anllo-Vento, L., Sereno, M. I., Frank, L. R., Buxton, R. B., Dubowitz, D. J., et al. (1999). Involvement of striate and extrastriate visual cortical areas in spatial attention. *Nature Neuroscience, 2,* 364–369.
Martinez, A., DiRusso, F., Anllo-Vento, L., Sereno, M. I., Buxton, R. B., & Hillyard, S. A. (2001). Putting spatial attention on the map: Timing and localization of stimulus selection processes in striate and extrastriate visual areas. *Vision Research, 41,* 1437–1457.
Müller, N. G., Bartelt, O. A., Donner, T. H., Villringer, A., & Brandt, S. A. (2003). A physiological correlate of the ''zoom lens'' of visual attention. *Journal of Neuroscience, 23,* 3561–3565.
Müller, N. G., & Kleinschmidt, A. (2003). Dynamic interaction of object-space-based attention in retinotopic visual areas. *Journal of Neuroscience, 23,* 9812–9816.
Müller, N. G., & Kleinschmidt, A. (2004). The attentional ''spotlight's'' penumbra: Center-surround modulation in striate cortex. *NeuroReport, 15,* 977–980.
Müller, N. G., & Kleinschmidt, A. (2007). Temporal dynamics of the attentional spotlight: Neuronal correlates of attentional capture and inhibition of return in early visual cortex. *Journal of Cognitive Neuroscience, 19,* 587–593.
Noesselt, T., Hillyard, S. A., Woldorff, M. G., Schoenfeld, A., Hagner, T., Jaencke, L., et al. (2002). Delayed striate cortical activation during spatial attention. *Neuron, 35,* 575–587.
Noguchi, Y., & Kakigi, R. (2005). Neural mechanisms of visual backward masking revealed by high temporal resolution imaging of human brain. *Neuroimage, 24,* 178–187.
O'Connor, D. H., Fukui, M. M., Pinsk, M. A., & Kastner, S. (2002). Attention modulates responses in human lateral geniculate nucleus. *Nature Neuroscience, 5,* 1203–1209.
Potter, M. C., Chun, M. M., Banks, B. S., & Muckenhoupt, M. (1998). Two attentional deficits in serial target search: The visual attentional blink and an amodal task-switch deficit. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 24,* 979–992.
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, 124,* 849–860.
Raymond, J. E., Shapiro, K. L., & Arnell, K. M. (1995). Similarity determines the attentional blink. *Journal of Experimental Psychology: Human Perception and Performance, 21,* 653–662.
Ress, D., Backus, B. T., & Heeger, D. J. (2000). Activity in primary visual cortex predicts performance in a visual detection task. *Nature Neuroscience, 3,* 940–945.
Ress, D., & Heeger, D. J. (2003). Neuronal correlates of perception in early visual cortex. *Nature Neuroscience, 6,* 414–420.
Saenz, M., Buracas, G. T., & Boyton, G. M. (2002). Global effects of feature-based attention in human visual cortex. *Nature Neuroscience, 5,* 631–632.
Sereno, M. I., Dale, A. M., Reppas, J. B., Kwong, K. K., Belliveau, J. W., Brady, T. J., et al. (1995). Borders of multiple visual areas in humans revealed by functional magnetic resonance imaging. *Science, 269,* 889–893.
Sergent, C., Baillet, S., & Dehaene, S. (2005). Timing of the brain events underlying access to consciousness during the attentional blink. *Nature Neuroscience, 8,* 1391–1400.
Shapiro, K., Schmitz, F., Martens, S., Hommel, B., & Schnitzler, A. (2006). Resource sharing in the attentional blink. *NeuroReport, 17,* 163–166.
Shapiro, K. L., Johnston, S. J., Vogels, W., Zaman, A., & Roberts, N. (2007). Increased functional magnetic resonance imaging activity during nonconscious perception in the attentional blink. *NeuroReport, 18,* 341–345.
Somers, D. C., Dale, A. M., Seiffert, A. E., & Tootell, R. B. (1999). Functional MRI reveals spatially specific attentional modulation in human primary visual cortex. *Proceedings of the National Academy of Sciences, U.S.A., 16,* 1663–1668.
Soto-Faraco, S., & Spence, C. (2002). Modality-specific auditory and visual temporal processing deficits. *Quarterly Journal of Experimental Psychology, 55,* 23–40.
Vogel, E. K., & Luck, S. J. (2002). Delayed working memory consolidation during the attentional blink. *Psychonomic Bulletin & Review, 9,* 739–743.
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.
Zaitsev, M., Hennig, J., & Speck, O. (2004). Point spread function mapping with parallel imaging techniques and high acceleration factors: Fast, robust, and flexible method for echo-planar imaging distortion correction. *Magnetic Resonance in Medicine, 52,* 1156–1166.

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