



Note

Blinks of the eye predict blinks of the mind

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ABSTRACT

The Attentional Blink (AB) – a deficit in reporting the second of two target stimuli presented in close succession in a rapid sequence of distracters – has been related to individual processing limitations of working memory. Given the known role of dopamine (DA) in working memory processes, the present experiment tested the hypothesis that DA, and in particular the DA/D1 subsystem, plays a role in the AB. We present evidence that the spontaneous eyeblink rate (EBR), a functional marker of central dopaminergic function, reliably predicts the size of AB. Thus, in line with our hypothesis, these data point to a modulatory role for DA in the AB.

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1. Introduction

The human brain is severely limited with regard to the number of events it can process at one time. A particularly convincing demonstration of this limitation is provided by the so-called Attentional Blink (AB; Raymond, Shapiro, & Arnell, 1992), which occurs if two masked (or otherwise difficult to identify) target stimuli appear in close temporal proximity, such as in RSVP (Rapid Serial Visual Presentation) tasks: Whereas the first target (T1) is commonly easy to identify and to report, performance on the second target (T2) is dramatically impaired if it follows T1 within 100–500 ms.

Many theoretical accounts of the AB assume that processing and consolidating T1 in working memory (WM) occupies limited attentional mechanisms to a degree that leaves too little available for processing and consolidating T2 if it appears before the consolidation of T1 is completed (for an overview, see Shapiro, 2001). In line with this notion, Shapiro, Schmitz, Martens, Hommel, and Schnitzler (2006) suggest a trade-off between the amount of resources devoted to T1 processing and the probability of identifying T2: Participants who showed greater attention-related brain activity while processing T1 were more likely to miss T2. Yet, the AB does not reflect a structural bottleneck in information processing (see Hommel et al., 2006), as studies have shown that T2 performance is usually very good when T2 immediately follows T1, a phenomenon called “lag-1 sparing” (Visser, Bischof, & Di Lollo,

1999). In addition, people can report even more than two targets when these targets belong to the same category and are presented in a sequence (Di Lollo, Kawahara, Ghorashi, & Enns, 2005), suggesting that distracter interference plays an important role in the AB.

Recent neuroimaging and patient studies have implicated a network of frontal, right-parietal and temporal brain areas involved in perceptual awareness in the AB (for review, see Hommel et al., 2006). Yet, little is known about the neurochemical mechanisms that may modulate the AB. In recent years, a computational theory has postulated that norepinephrine, given its role in attentional selection in the temporal domain (Aston-Jones & Cohen, 2005), is directly involved in the AB phenomenon (Nieuwenhuis, Gilzenrat, Holmes, & Cohen, 2005). Yet, a pharmacological study showed no differential effects of α 2 adrenoceptor agonist clonidine vs. placebo on AB task performance (Nieuwenhuis, Van Nieuwpoort, Veltman, & Drent, 2007). Consistent with this research, another recent study (De Martino, Strange, & Dolan, 2007) revealed no effect on T2 detection after the administration of nadolol, a peripherally acting β -adrenergic antagonist, or after the administration of 20 mg of propranolol, a centrally acting β -adrenergic antagonist. The authors did report impaired T2 detection after the administration of a higher dose (40 mg) of propranolol, but this impairment was equally strong for T2s presented within, and T2s presented outside the time window of the AB. Thus, none of the adrenergic manipulations used in these two studies affected the size of the AB. This series of null findings is inconsistent with a role for the noradrenergic system in the AB, although future psychopharmacological studies in humans are necessary to fully understand the possible role of norepinephrine in temporal attention and the AB.

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It is unlikely that any single neuromodulatory mechanism can explain the plethora of experimental factors that are known to modulate the Attentional Blink. However, dopamine (DA) represents a particularly likely candidate, given its key role in WM processes (Braver & Barch, 2002; Braver, Barch, & Cohen, 1999; Hazy, Frank, & O'Reilly, 2006). Specifically, DA projections to the prefrontal cortex (PFC) serve to gate access of context representations into active memory through simple neuromodulatory effects on processing units in the PFC (Braver et al., 1999). These effects serve both gating and learning functions, which enable the system to discover what information must be maintained for performing a given task, and to regulate when that information is updated. Thus, dopamine plays an important role in attentional selection. Notably, very recently we obtained evidence that people high in WM operation span show a smaller AB (Colzato, Spapé, Pannebakker, & Hommel, 2007), indicating that the AB is related to WM in general and attributable to operational resource limitations in particular (Dehaene, Sergent, & Changeux, 2003; Di Lollo et al., 2005; Gross et al., 2004; Hommel et al., 2006). Schizophrenic patients (Cheung, Chen, Chen, Woo, & Yee, 2002; Li et al., 2002) have been reported to show a more pronounced AB, while no differences in AB size were reported for Parkinson's patients compared to controls (Vardy, Bradshaw, & Iansek, 2003). Unfortunately, these studies have major confounds given that the schizophrenic and Parkinson's patients were taking antipsychotic drugs and L-DOPA, respectively (which both act on the dopaminergic system). The results obtained in these studies may, thus, have measured effects of medication use. Ideally, patient studies should test patients "on" and "off" medication, but for obvious ethical reasons these kind of studies are difficult to perform.

Importantly for the current study, these links between dopamine and WM on the one hand and between WM and AB on the other hand point to a possible modulatory role for DA in the AB. The major dopaminergic pathways are dominated by D1 and D2 receptors, which appear to play different, separable roles in regulating human cognition. Whereas DA/D1-dominated pathways are presumably involved in WM processes (Goldman-Rakic, Muly, & Williams, 2000; Sawaguchi & Goldman-Rakic, 1991), DA/D2-dominated pathways have been implicated in response inhibition and cognitive flexibility (Lee, Groman, London, & Jentsch, 2007). Given the link between the AB and capacity limitations of working memory, one may therefore expect that especially DA/D1 pathways are involved in the AB. Consistent with this idea, a recent study found no effect of the DA/D2 antagonist amisulpride on T2 detection (Gibbs, Naudts, Spencer, & David, 2007).

2. Purpose of study

The present experiment aimed to test the hypothesis that DA, and in particular the DA/D1 subsystem, plays a role in the AB by driving the WM processes necessary to store and consolidate targets in AB tasks. The dependent measure of interest for our purposes was the spontaneous eyeblink rate (EBR), a functional marker of central dopaminergic function (Blin, Masson, Azulay, Fondarai, & Serratrice, 1990; Karson, 1983; Kleven & Koek, 1996; Taylor et al., 1999). This measure has been shown to reliably predict behavioral performance in several cognitive task that have been associated with dopaminergic function (e.g., Dreisbach et al., 2005; Colzato, van Wouwe, & Hommel, 2007; Colzato, van den Wildenberg, & Hommel, submitted for publication-a). Further evidence that EBR reflects dopaminergic function comes from studies of patients with psychiatric and neurological disorders that are marked by abnormal dopaminergic function. For example, schizophrenic patients, who show increased dopaminergic activity in the striatum, but reduced activity in the prefrontal cortex (Davis, Kahn, Ko, & Davidson,

1991), show elevated EBRs (Freed, 1980), while EBR is reduced in Parkinson's patients (Deuschel & Goddemeier, 1998). Furthermore, recreational cocaine users, who suffer from a loss of nigrostriatal dopaminergic cells, also display lower EBRs (Colzato, van den Wildenberg, & Hommel, submitted for publication-b). Together, these findings indicate that EBR provides a reliable measure of dopaminergic function.

To examine a possible modulatory effect of dopamine on the AB, we investigated whether the size of AB can be predicted from spontaneous EBR in healthy individuals. Given the known role of DA/D1 in WM task performance (Goldman-Rakic et al., 2000; Sawaguchi & Goldman-Rakic, 1991) and based on our previous findings showing a small AB for individuals high in WM operation span (Colzato, Spapé, et al., 2007), we expected to find a negative correlation between AB size and EBR. Our specific prediction was that participants with a high EBR (i.e., high basal dopaminergic activity) would show a smaller AB.

3. Method

3.1. Participants

Twenty young healthy adults (10 women and 10 men, between 18 and 30 years old) served as participants for partial fulfillment of course credit or a financial reward. Participants were recruited via notices posted on community bulletin boards and by word of mouth. Following Colzato, van den Wildenberg, and Hommel (2007) and Colzato, Kool, and Hommel (2008) participants were selected with the Mini International Neuropsychiatric Interview (M.I.N.I.; Lecrubier et al., 1997). The following selection criteria were applied: no Axis 1 psychiatric disorder (DSM-IV), including 'substance abuse'; no clinically significant medical disease; no use of medication. Written informed consent was obtained by all participants; the protocol was approved by the local ethical committee (Leiden University, Institute for Psychological Research).

3.2. Apparatus and stimuli

The RSVP experiment was controlled by a Targa Pentium III computer. All stimuli were presented in a resolution of 800 by 600 pixels in 16 bit color on a 17" CRT refreshing at 100 Hz. Participants were seated at a viewing distance of about 50 cm. The fixation mark ("+"), as well as all RSVP items were presented centrally in black on a gray background (RGB 128, 128, 128). Each item was set in 16 point Times New Roman font. RSVP items included letters and digits. Letters were drawn randomly without replacement from the alphabet. Digits were drawn randomly from the set 1–9.

3.3. Procedure and design

The study consisted of two sessions held on the same day. In the first session, eye blink data was recorded. In the second session, participants performed the RSVP task. None of the participants needed extra training. The duration of the practice, thus, did not differ between participants. We formed two groups based on spontaneous EBR levels using a split mean: a low (10 participants, 2.40–15.16 score) and a high (10 participants, 15.30–31.80) EBR group.

3.4. Eyeblink rate

A BioSemi ActiveTwo system (BioSemi Inc., Amsterdam, The Netherlands) was used to record the EBR. Following Colzato, van Wouwe, et al. (2007), eye movements were recorded, with two vertical (one upper, one lower) and two horizontal (one left, one right) Ag–AgCl electrodes, for 6-min eyes-open segments under resting conditions. The vertical electrooculogram (EOG), which recorded the voltage difference between two electrodes placed above and below the left eye, was used to detect eye blinks. The horizontal EOG, which recorded the voltage difference between electrodes placed lateral to the external canthi, was used to measure horizontal eye movements. Given that spontaneous EBR is supposed to be stable during daytime but increases in the evening (8:30 p.m., as reported by Barbato et al., 2000), data were never collected after 5 p.m. Additionally, we asked participants to avoid alcohol and nicotine consumption and to sleep sufficiently the day before the recording. Participants were comfortably sitting in front of a blank poster with a cross in the center, located about 1 m from the participant. The participant was alone in the room and asked to look at the cross in a relaxed state.

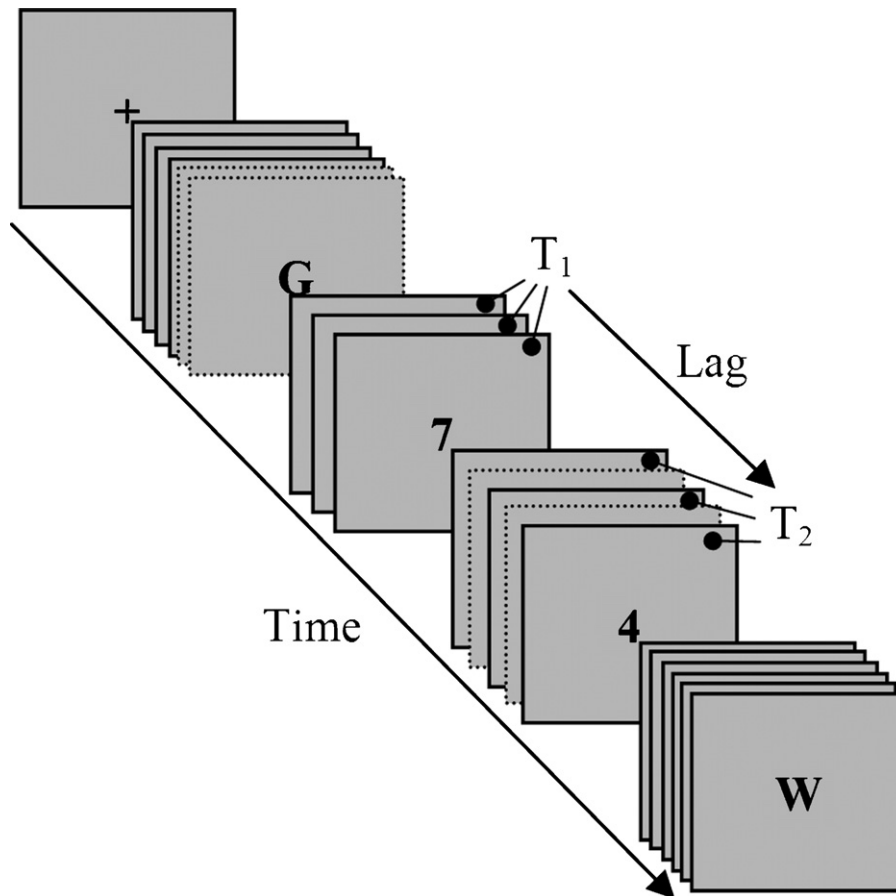


Fig. 1. Example of an RSVP trial. On every trial, 20 items were presented at the center of the screen, preceded by a 2000-ms fixation cross. Most of the items were letters, presented for 40 ms each and followed by a 40-ms blank. Participants had to detect two target numbers (T1 and T2) among the items. T1 and T2 were separated by one, three, five, or eight nontarget items, defining the *lag*. T1 was presented at position 7, 8 and 9 of the stimulus stream.

3.5. RSVP task

In the RSVP task adopted from Colzato, Spapé, et al. (2007), participants had to identify and report two digits (T1 and T2) presented in a rapid stream of letter distractors. After having read the instructions, which included a slow demonstration of the RSVP, and indicating to have fully understood the task, participants were required to go through 24 trials of training. If more than 50% of the responses were incorrect during the training, the training part was automatically restarted. A fixation plus sign, which was shown for 2000 ms, marked the beginning of each trial. After a blank interval of 250 ms, the RSVP commenced, consisting of 20 items with a duration of 40 ms each and an inter-stimulus interval of 40 ms.

The position of T1 in the stimulus stream was varied randomly between position 7, 8 and 9 in order to reduce the predictability of target onsets. T2 was presented directly thereafter (Lag 1), or after another 2, 4, or 7 distractors (Lag 3, 5, and 8 successively), see Fig. 1. Both targets were to be reported directly (order of report was not considered) after the RSVP – the question being “which two targets did you see?” – by pressing the corresponding digit keys. A full experimental session lasted 30 min and contained one block of 360 trials (3 locations of T1 \times 4 lags \times 30 repetitions).

3.6. Statistical analysis

T1 and T2 accuracy data were submitted to separate ANOVAs with lag (1, 3, 5, and 8) as a within-participants factor and EBR level (high vs. low) as between-participant factor. T2 accuracy was based only on those trials in which T1 was correctly reported (T2|T1). To test our main hypothesis that dopamine modulates the size of the AB, we ran a Pearson correlation test, which examined the association between EBR and the maximal AB (measured as T2|T1 at Lag 8 minus the minimum of T2|T1 at Lag 3 and at Lag 5). We also explored the relationship between EBR and Lag-1 sparing (measured as T2|T1 at Lag 1 minus T2|T1 at Lag 5), and between EBR and mean (i.e., averaged across lags) T1 and T2 accuracy. A significance level of $p < .05$ was adopted for all statistical tests.

4. Results

4.1. Eyeblink rate measurement

EOG data were examined using the Brain Vision Analyzer (Brain ProductsTM GmbH, Munich, Germany; www.brainproducts.com/products/analyzer/). An eyeblink was defined as a voltage change of 100 μ V in a time interval of 500 ms (Colzato, van Wouwe, et al., 2007). Our sample of participants had EBRs ranging from 2.4 to 31.8 per min (standard deviation (S.D.)=8.6), and thus represented a wide range of tonic dopaminergic functioning.

4.2. RSVP task

T1 accuracy is shown in Fig. 2. The ANOVA with lag as within-participant factor showed a significant lag effect, $F(3, 57) = 29.49$, $p < .001$. As Fig. 2 shows, this effect was due to a dip in performance at Lag 1, i.e., when T2 immediately followed T1. This pattern is often observed if T1 and T2 belong to the same category (e.g., digits) and satisfy the same selection criteria, and when the presentation rate is fast. These conditions are thought to increase the competition between T1 and T2 representations if they occur close in time, with T2 outperforming T1 more often (Colzato, Spapé, et al., 2007; Hommel & Akyürek, 2005; Potter, Staub, & O'Connor, 2002).

The ANOVA of conditional T2 accuracy (T2|T1) revealed a significant lag effect, $F(3, 57) = 33.34$, $p < .001$, indicating a marked AB with good performance at Lag 1 (Lag-1 sparing, Visser et al., 1999) and a considerable dip at Lags 3 and 5 (see Fig. 2). Interestingly,

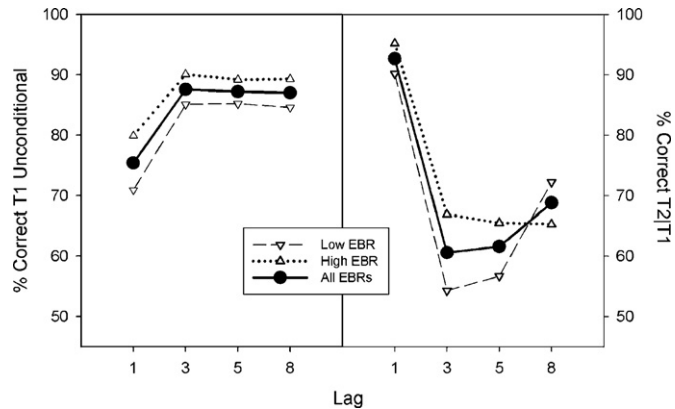


Fig. 2. T1 (unconditional) performance (left panel) and T2 performance given T1 correct (T2/T1) (right panel), shown separately for each lag and for T2/T1 for high and low spontaneous eyeblink rate (EBR).

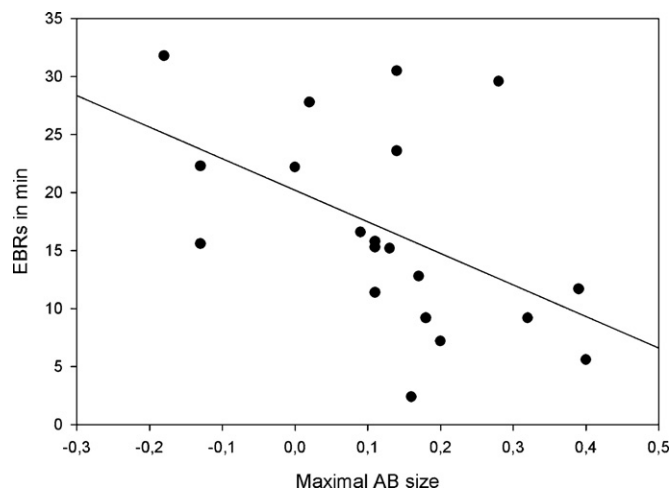


Fig. 3. Relationship between individual spontaneous eyeblink rate (EBR) in minutes (min) and maximal AB size. Note that those individuals that showed a high EBR generally showed a smaller AB.

EBR level impacted the blink size, as indicated by a two-way interaction between group and lag, $F(3, 54) = 2.81, p = .048$.¹ Participants in the high EBR group showed a smaller AB than participants in the low EBR group, thus confirming our prediction that participants with a high EBR (i.e., high basal dopaminergic activity) would show a smaller AB. Also in line with this prediction, EBR negatively correlated with AB size,² $r(20) = -.530, p = .016$. As it can be seen in Fig. 3, individuals with relatively high dopaminergic base activity (as reflected by high EBRs) generally showed a smaller AB. Importantly, EBR did not correlate significantly with Lag-1 sparing [$r(20) = .304, p = .192$], or mean T1 and T2 accuracy [respectively: $r(20) = .282, p = .228$; $r(20) = .150, p = .529$], indicating that EBR was selectively associated with AB size.

¹ This interaction was even more pronounced when the data from Lag 1 were dropped from the analysis, $F(2, 36) = 5.80, p < .01$. Separate ANOVAs showed a lag effect in low blinkers, $F(2, 18) = 11.35, p < .001$, but not in high blinkers, $F(2, 18) < 1$, with both groups showing equivalent performance at Lag 8, $p > .34$.

² Other measures of the AB deficit (e.g., $T2Lag8/T1 - T2Lag3/T1$) were also highly correlated with EBR, $r(20) = -.464, p = .039$, and even others, atypical and not of common use (e.g. $[T1Lag5 - T2Lag5] - [T1Lag8 - T2Lag8]$ and $[T1Lag3 - T2Lag3] - [T1Lag8 - T2Lag8]$), showed near significant correlations with EBR, $r(20) = -.425, p = .062$ and, $r(20) = -.385, p = .094$, respectively. These results exclude the possibility that our data depend on the specific measure of AB size used.

5. Discussion

Given that the size of the AB is predicted by individual WM capacity (Colzato, Spapé, et al., 2007) and the known link between WM and dopamine (Goldman-Rakic et al., 2000; Sawaguchi & Goldman-Rakic, 1991), we predicted that AB size varies with the individual level of dopamine production. Indeed, our findings show that spontaneous EBR, a marker of central dopaminergic activity (Blin et al., 1990; Karson, 1983; Kleven & Koek, 1996; Taylor et al., 1999), reliably predicts the size of AB, indicating a possible modulatory role for DA in the AB.³ As our participants were screened for several psychiatric disorders, we can rule out an account in terms of pre-existing psychiatric disorders (as schizophrenia, ADHD, and obsessive compulsive disorder) that have been associated with dopaminergic abnormalities (Davis et al., 1991; Pooley, Fineberg, & Harrison, 2007; Tripp & Wickens, 2007).

Even though the correlative nature of our findings does not directly speak to the underlying causal relations, the idea that DA plays an important role in the AB is supported by findings from previous behavioral studies. For example, manipulations that are thought to activate the dopaminergic system (Ashby, Isen, & Turken, 1999; Ashby, Valentin, & Turken, 2002), such as viewing pictures of positive affective content (Olivers & Nieuwenhuis, 2006) and the induction of happy states (Jefferies, Smilek, Eich, & Enns, 2008), have been shown to reduce the size of the AB. As mentioned already, people high in WM operation span, which is associated with high basal dopaminergic activity (Braver & Barch, 2002; Braver et al., 1999; Hazy et al., 2006), show a smaller AB (Colzato, Spapé, et al., 2007). Taken together, these observations support our hypothesis that the size of the AB is modulated by dopamine.

This leaves the question of how dopamine might modulate attentional processes. A major role of WM in general and, presumably, in AB-related tasks in particular is the gating of task-relevant information (Braver & Barch, 2002). Individuals low in WM capacity do not necessarily process or store fewer items than people high in capacity but, rather, are less selective with regard to what they store (Vogel, McCollough, & Machizawa, 2005). In an AB task, this would imply that individuals with lower capacity are less efficient in discriminating targets from distractors, which means that distractors are more likely to enter WM and interfere with target information. Attentional gating is thought to result from phasic increases of DA activity, which again is a multiplicative function of the individual tonic DA level (Frank, 2005; O'Reilly & Frank, 2006). Given that EBR is thought to reflect this individual tonic DA level, it makes sense to assume that higher EBRs reflected higher tonic levels, which again allowed for higher and more pronounced phasic DA peaks and, thus, more efficient gating.

Taken together, the current observations support the idea that the AB phenomenon is related to activity of the dopaminergic system, presumably that of the DA/D1 subsystem. Clearly, a more systematic investigation of this issue is necessary. Further investigations testing acute neuromodulatory effects of highly selective D1 agonists, such as SKF 38393, on the magnitude of the AB are necessary to determine the precise role of DA/D1 in the AB and in temporal attention more generally.

³ Given the pattern of T2 performance in high and low blinkers shown in Fig. 2, one may speculate that high blinkers show a shallower but longer AB, that is, it may be that at even longer lags low blinkers perform better than high blinkers. On the one hand, this is an interesting possibility that we cannot rule out and need to leave for further research. On the other hand, however, the numerical advantage for low blinkers at Lag 8 was far from significance and matches the inverse advantage on T1 for high blinkers at this lag. This may suggest that performance at Lag 8 effectively reached asymptote for both groups, who however differ slightly with regard to the amount of attentional resources devoted to T1 and T2 processing, respectively.

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