European Neuropsychopharmacology (▮▮▮▮) ▮, ▮▮▮–▮▮▮

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The DRD2 C957T polymorphism and the Attentional Blink—A genetic association study

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Received 22 June 2012; received in revised form 27 August 2012; accepted 25 September 2012

KEYWORDS
Attentional Blink;
Genetics;
Dopamine;
DRD2 C957T;
Rs6277

Abstract
The Attentional Blink phenomenon (AB) describes a transient deficit in temporally selective visual attention regarding the processing of the second of two target stimuli in a rapid serial visual presentation (RSVP) task. The AB is a very prominent paradigm in the Cognitive Neurosciences that has been extensively studied by diverse psychophysiological techniques such as EEG or fMRI. Association studies from molecular genetics are scarce although the high heritability of higher cognitive functioning is proven. Only one seminal study reported an association between AB magnitude and the dopamine receptor D2 (DRD2) C957T polymorphism (Colzato et al., 2011). This functional polymorphism influences striatal D2 receptor binding affinity and thereby the efficacy of dopaminergic neurotransmission which is important for working memory and attentional processes. Colzato et al. (2011) reported that DRD2 C957T T/T-carriers exhibit a significant smaller AB than C-allele carriers. In the present study this influence of the DRD2 SNP on the AB could not be replicated in *N*=211 healthy participants. However, a significantly larger lag 1 sparing was observed for homozygous T/T-carriers. Moreover, carriers of at least one T-allele showed a significantly poorer performance in the identification of T1. In general, these results support the notion of a role of the dopaminergic system on the AB. However, as our results do not parallel previous findings the exact nature of this influence and its dependence on task parameters will have to be examined in further genetic association studies.

1. Introduction

The AB phenomenon is an extensively studied paradigm in neuroscience. In this dual-target dual-task experimental design participants are required to detect two targets presented in close temporal proximity amongst a rapid serial visual stream of distractors (rapid serial visual presentation, RSVP).

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http://dx.doi.org/10.1016/j.euroneuro.2012.09.010

If the two targets appear in a short time window (200–500 ms) the detection accuracy of the second target is severely impaired (Raymond et al., 1992). According to cognitive theories of the AB (for review, see Dux and Marois, 2009), the AB is thought to arise from a depletion of attentional resources after the successful detection of the first target (T1), that prevents the encoding of the second target (T2) into the capacity limited working memory (WM) system. As the successful encoding of the second target (T2) in WM is a necessary prerequisite for its successful report later on, this capacity limit severely constraints task performance in the face of a rapid serial stream of distracting stimuli. Views differ with respect to the mechanism by which this attentional depletion interferes with task performance: for instance, it is still under debate whether the AB stems from a limitation of processing capacity during encoding (Chun and Potter, 1995) or of disturbed retrieval (Shapiro et al., 1994). What all theories have in common, however, is the assumption that cognitive processes at the interface between attention and WM lead to the AB (cf. Dux and Marois, 2009). Prominent theoretical accounts for WM highlight the role of attention in WM as the means by which the executive control component ("central executive") exerts control over representations in WM during encoding, maintenance and retrieval (Baddeley, 1996; Cowan, 1999; Dehaene et al., 1998). Following this logic, the control component of WM is a feasible candidate to explain performance in the AB paradigm. And indeed, high WM control has been associated with a smaller AB (Akyürek et al., 2007).

While behavioral approaches and the neurophysiological underpinnings of the AB such as for instance differences in oscillatory alpha and beta band activity and event-related potentials (ERPs) are under extensive research (see for instance Gross et al., 2004; Janson and Kranczioch, 2011; Kranczioch et al., 2003, 2007; Marois et al., 2004; Sergent et al., 2005), studies focusing on the neurochemical background including the molecular genetics are scarce. In general, the neurotransmitter dopamine plays a crucial role in complex cognitive functions such as WM and cognitive control (Brozoski et al., 1979; Cools et al., 2008; Kimberg et al., 1997; Luciana et al., 1992). Individual differences in dopaminergic genetic markers which affect dopaminergic neurotransmission such as the DRD2 gene which codes for the dopamine D2 receptor, have been linked to WM control processes (for a review, see Cools and D'Esposito, 2011).

In line with this there is only one genetic association study investigating the promising role of different dopaminergic polymorphisms in the AB performance so far (Colzato et al., 2011). In this study only the dopamine D2 receptor (DRD2) C957T polymorphism revealed significant impact on AB task performance in the way that the T-allele was associated with a better performance resulting in a smaller size of the AB. The C957T polymorphism (rs6277) of the human DRD2 gene located on chromosome 11q23 constitutes a single nucleotide polymorphism (SNP) which causes a synonymous coding C→T transition in exon 7. The SNP changes the receptor's affinity and regulates DRD2 availability *in vivo*, but its effect differs depending on the brain region under investigation (Hirvonen et al., 2004, 2009a,b). A recent PET study provides further evidence for a role of striatal dopamine in the AB (Slagter et al., 2012), even though it raises some doubt about the direction of the relationship. Here, greater D2-like receptor binding in the striatum was associated with a larger AB (ergo lower level of endogenous dopamine). Slagter et al. (2012) concluded that striatal dopamine may determine the AB by regulating the threshold for WM updating.

Genetic association studies provide considerable evidence that allelic variations in the functional DRD2 C957T influence WM. For instance, C957T interacted with COMT Val158Met in a word serial position test, where subjects with the C/C-genotype showed the poorest performance. This was further strengthened in interaction with COMT (Xu et al., 2007). The C957T was also linked to executive control, with homozygous T/T-carriers performing better on a verbal WM task (Jacobsen et al., 2006). Furthermore, dysfunctional impulsivity is enhanced in T-homozygotes measured by self-report and an experimental stop-signal paradigm (Colzato et al., 2010), and general cognitive ability measured by five cognitive tests of different domains was lower in C/C-carriers (Bolton et al., 2010). Moreover, an epistatic interaction of DRD2 C957T and CHRNA4 on visuospatial and phonological WM capacity was reported, where carriers of the DRD2 C/C-genotype showed a higher visuospatial and phonological WM capacity, but only if they were also homozygous for the T-allele of CHRNA4 rs1044396 (Markett et al., 2010, 2011). The gene CHRNA4 codes for the subunit alpha-4 of the nicotinic acetylcholine receptor and exerts pleiotropic effects among others on cognitive functions (Bellgrove and Mattingley, 2008; Markett et al., 2010, 2011). In sum, there is plenty of evidence for an association between C957T and phenotypes in the domain of WM and executive control, yet results are inconsistent with respect to the effect's direction. Carriers of the C/C-genotype show better performance on some tasks while performance on other tasks is impaired. A feasible explanation for these inconsistencies might be the non-unitarity of executive control processes (Miyake et al., 2000) and the partly opposing demands of diverse executive control tasks on cognitive stability (i.e. WM maintenance) and flexibility (i.e. WM updating) which are both thought to be mediated by dopaminergic transmission in different neural structures (Cools and D'Esposito, 2010). Therefore, it is imperative to the field of behavioral molecular genetics to apply a rigorous analysis of the cognitive task at hand and a careful psychometric characterization of the phenotype when studying the effect of polymorphisms on cognitive control (Green et al., 2008).

A further problem in the field is that in general, findings on the molecular genetics of cognitive phenotypes replicate not that easily. Given the repeated association of the DRD2 C957T polymorphism with performance in tasks that aim at WM and attentional control and the inconsistencies regarding the effect's direction, it is of major importance to assess whether independent replications of the reported results can support the initial finding. In the present study, we sought to investigate whether the reported association between the AB and the DRD2 C957T polymorphism by Colzato et al. (2011) can also be found in a large independent data set on the AB from our laboratory.

2. Experimental procedures

2.1. Participants

A total of 211 healthy subjects (n=166 women, n=45 men, mean age M=22.7 years, SD=4.7) provided buccal swaps for genotyping

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the DRD2 C957T polymorphism and gave written consent to participate in the present study. All subjects were compensated with course credits for their efforts. The high proportion of female participants reflects the gender distribution in German psychology classes. Before testing, subjects were screened for neurological and psychiatric disorders. All participants reported normal or corrected-to-normal vision. The study was carried out in accordance with the ethical principles of the Declaration of Helsinki of the World Medical Association and was approved by the local ethics committee of the University of Bonn.

2.2. Genotyping

DNA extraction and genotyping of the DRD2 C957T polymorphism (♯rs6277) was conducted as described previously (Markett et al., 2010).

2.3. Procedure

In the dual-target RSVP task target and distractor stimuli were presented in 36-point font size centrally on a 19 in. CRT monitor with a presentation frequency of 10 Hz on a white background. Distractors were black capital consonants (except F, K, Q, X, Y). The first target (T1) was a green letter, which could either be a vowel (except I) or a consonant (except F, K, Q, X, Z). The second target (T2) was a black capital X. After reading the instructions comprising two demo trials, participants underwent 13 practice trials before starting the experiment, which included three blocks of 36 trials. Each trial started with a red fixation cross for the duration of 900 to 1100 ms and was followed by 31 stimuli with 80 ms duration each and an inter-stimulus interval of 20 ms. T1 was presented after 14 to 17 distractors. In 66.67% of all trials, T1 was followed by T2, either with no (lag 1), one (lag 2) or six (lag 7) intervening distractors. After T2, 7-16 distractors followed until the trial ended. After presentation of the RSVP sequence, participants had to respond via the left or right mouse button if T1 was a vowel or not and whereas T2 (the X) appeared after the green letter.

2.4. Statistical analyses

Percentage of correct T1 and T2 identifications were computed for each lag condition. T2 accuracy includes only trials were T1 was identified correctly (T2/T1). First, we tested for possible associations between age, gender and AB task performance. Because age was negatively correlated with performance on lag 1 (lag 1: $r=-.155$, $p=.023$; lag 2: $r=-.020$, $p=.770$; lag 7: $r=-.116$, $p=.091$), it was included as covariate into all further analysis of variance (ANOVA) models. Omitting age as a covariate, however, led to virtually identical results. Separate univariate ANOVAs with gender as fixed factor and T2 accuracy on each lag, lag 1 sparing (measured as T2/T1 at lag 1 minus T2/T1 at lag 2), T1 accuracy, or AB size (measured as T2/T1 at lag 7 minus T2/T1 at lag 2) as independent variables revealed no significant associations. Second, the effect of the DRD2 C957T polymorphism on AB task performance was analyzed by repeated measures ANOVA with lag (1, 2 and 7) as within-subjects factor and genotype as between-subjects factor. Lag 1 sparing and the AB size were included in separate ANOVA models as AV and genotype as between-subjects factor. All statistical tests were conducted at a $p<.05$ threshold. All analyses were carried out using SPSS 20.0 (SPSS Inc., Chicago, IL, USA).

3. Results

3.1. Genetic analyses

The genotype frequency of the DRD2 C957T polymorphism (T/T: $n=66$, C/T: $n=100$, C/C: $n=45$) did not deviate from the Hardy–Weinberg equilibrium ($\chi^2=.38$, df=1, ns) and did not differ between gender groups ($\chi^2=.196$, df=2, ns). There were no differences in allelic distributions (men: T: 52.2%, C: 47.8%; women: T: 55.7%, C: 44.3%) between both gender groups ($\chi^2=.69$, df=1, ns).

3.2. Experimental data

The AB phenomenon could be reproduced. T2 accuracy (T2/T1) varied as a function of lag in trials where T1 was reported correctly. Mean T2 accuracy decreased from lag 1 (57.5%) to lag 2 (31.6%) and then increased again reaching 79.7% at lag 7. Accordingly, a repeated measure ANOVA with lag as within-subjects factor revealed a significant lag effect showing that at least one of the tested lag pairs differed significantly from each other ($F_{(2,418)}=18.9$; $p<.00001$; $\eta^2=.083$). We conducted three paired *T*-tests, which revealed that lag 1 differed significantly from lag 2 ($t_{(211)}=18.48$, $p<.001$), lag 2 differed significantly from lag 7 ($t_{(211)}=-24.75$, $p<.001$) and lag 1 differed significantly from lag 7 ($t_{(211)}=-10.36$, $p<.001$). Mean correct T1 identification was 93.2% when T2 appeared at lag 1, 92.1% at lag 2 and 93.7% at lag 7 ($p>.05$). To visualize the AB phenomenon the percentage values and SEM of correct T1 and T2/T1 can be seen in Figure 1.

The genetic effects analyzed by repeated measure analysis of variance revealed no significant main effect of genotype on T2 accuracy. The mean percentage correct T2/T1 as a function of DRD2 genotype and lag is illustrated in Figure 2. Furthermore, there was no evidence for an effect of allelic or genotype group on T1 performance, lag 1 sparing, or AB size. Descriptive mean values and genotype frequencies are reported in Table 1.

However, with respect to lag 1 sparing we detected a significant genetic effect on the allelic level (Figure 3). Homozygote T/T-carriers (C-) showed a significantly stronger lag 1 sparing than carriers of at least one C-allele (C+) ($F_{(1,208)}=4.71$; $p=.031$, $\eta^2=.022$). On the allelic level also a significant result was obtained for T1 accuracy. Carriers with at least one T-allele (T+) showed a significantly lower correct T1 identification ($F_{(1,208)}=5.94$; $p=.016$, $\eta^2=.028$).

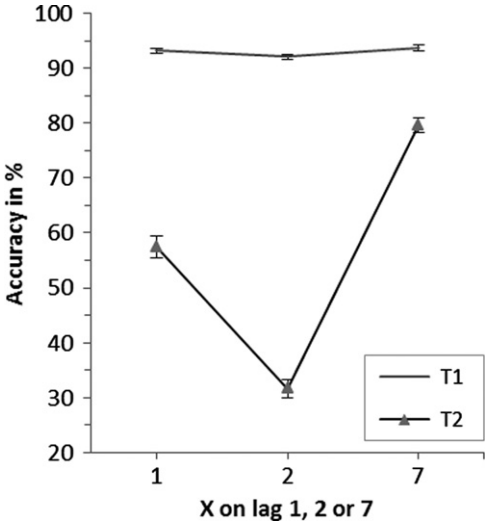

Figure 1 Mean T1 and T2 detection rate ± SEM at lags 1, 2 and 7 ($n=211$). Only trials where T1 has been identified correctly are included in T2 accuracy.

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4. Discussion

The present study investigated the association between DRD2 C957T and the AB. We could not replicate the main finding of a recently published study by Colzato et al. (2011), who observed that T/T carriers exhibited a significantly smaller AB than C/T and C/C carriers. If at all, homozygous T/T-carriers showed a somewhat larger blink compared to C-allele carriers, however only on a descriptive level. Interestingly, on the allelic level carriers of at least one T-allele exhibited a significantly reduced correct T1 identification. T/T-homozygotes showed a significantly stronger lag 1 sparing effect than C-allele carriers, which was due to a concurrent better lag 1 and worse lag 2 performance in the group of T/T-homozygotes. That is, in contrast to the results of Colzato et al. (2011) our findings indicate that the T-allele is detrimental to the ability to pick a target from an RSVP stream, and that in a dual-task dual-target context it is the C-allele, which determines how this affects the processing of a subsequent target. In conclusion, our study provides support for the finding that DRD2 C957T is associated with the AB phenomenon, while it remains unclear what aspect of the AB is influenced by the associated individual differences in the dopaminergic system.

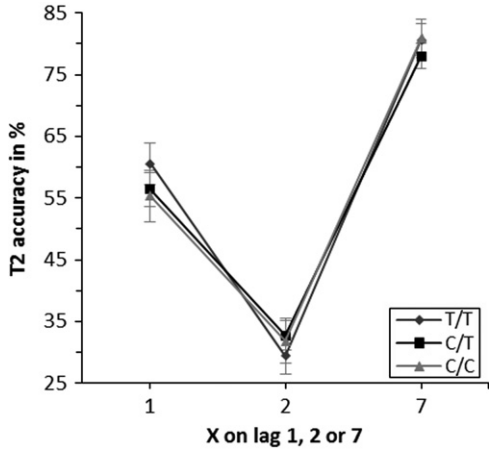

Figure 2 T2 accuracy for each lag as a function of DRD2 C957T polymorphism.

Two issues, however, warrant further discussion: first, that – compared to the Colzato et al. study – other aspects of the AB are affected by DRD2 C957T in the present study and second, that the present effects point into a different direction. With regard to the first issue, it is important to keep in mind what the two measures (AB size and lag 1 sparing) represent: lag 1 sparing constitutes the largely unimpaired recognition of the second target when both targets occur consecutively (Potter et al., 1998; Visser et al., 1999), while the size of the

Table 1 Genotype as well as allelic frequencies and respective descriptive mean values (%) of T2 accuracy (at lags 1, 2, 7), T1 accuracy, lag 1 sparing and max AB size.

DRD2 C957T	*N*	T2 accuracy			T1 accuracy	Lag 1 sparing	Max AB size
		Lag 1	Lag 2	Lag 7			
CC	45	55.28	31.85	80.66	95.06	23.43	48.81
CT	100	56.49	32.75	78.06	92.93	23.74	45.31
TT	66	60.44	29.77	81.35	92.62	30.67	51.58
C+	145	56.11	32.47	78.87	93.59	23.64*	46.40
C−	66	60.44	29.77	81.35	92.62	30.67*	51.58
T+	166	58.06	31.57	79.37	92.80*	26.49	47.80
T−	45	55.28	31.85	80.66	95.06*	23.43	48.81

*$p < .05$.

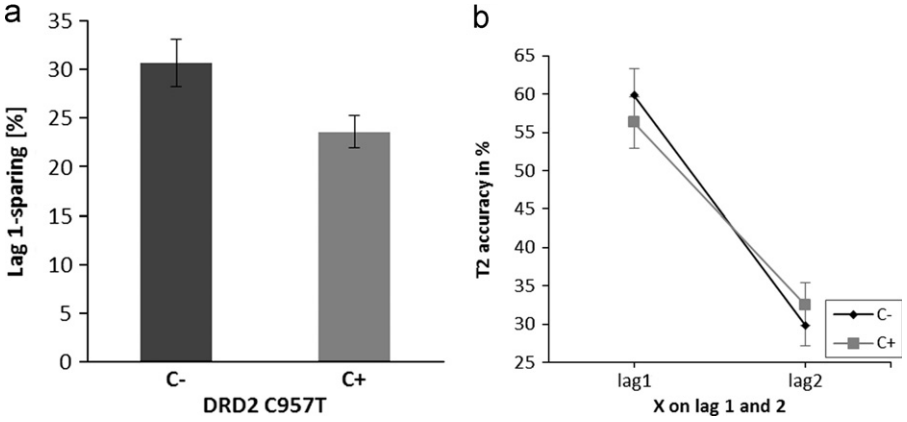

Figure 3 Means and SEMs of lag 1 sparing (a) and T2 accuracy (%) at lags 1 and 2 (b) for allelic variants C− and C+.

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AB represents the impairment of recognition of the second target when presented around 200–500 ms after the first (Raymond et al., 1992). It has been demonstrated that processes underlying lag 1 sparing and those resulting in the AB are independent from each other (Dell'Acqua et al., 2009; Livesey and Harris, 2011). Accordingly, lag 1 sparing is stimulus- and task-specific, while the AB is not (Livesey and Harris, 2011). It is therefore possible that our results differ from those of Colzato et al. because of the different nature of the tasks: whereas in the present study a so-called probe task (Kelly and Dux, 2011) was used, where participants were to detect a green letter (T1) and a black capital X (T2) amongst black letters, Colzato et al. employed a categorical task, that is, the identification of two digits amongst letters. The difference of the two tasks is for instance apparent in the substantial differences in T2 lag 1 performance: in Colzato et al.'s study it is above 95% and thus comparable to T1 performance. For the present study T2|T1 at lag 1 is at least 30% lower than T1 performance. Moreover, in the present study T1 performance was not affected by lag, whereas in the study by Colzato and colleagues T1 performance was significantly reduced when T2 was presented at lag 1. The idea that task-related differences account for at least some of the differences in the present results and the results by Colzato et al. (2011) finds further support in the finding that AB magnitude in a probe task and AB magnitude in a categorical task are not related intra-individually (Kelly and Dux, 2011, but see Dale and Arnell, 2011 for contrary findings). Kelly and Dux (2011) suggest that this might indicate that the two tasks rely on at least partially different cognitive resources.

In the present study, significant results were restricted to T1 performance and lag 1 sparing. As common in the literature, we took the difference in T2 accuracy between lag 1 and lag 2 as a measure of lag 1 sparing. For T/T-carriers this approach yielded significantly stronger lag 1 sparing, which was however due to the combined effects of a better T2 accuracy on lag 1 and a lower T2 accuracy on lag 2. Interestingly, this stronger lag 1 sparing went along with a significantly reduced T1 performance. On the other hand, C/T-carriers also showed poorer T1 performance, but in combination with smaller lag 1 sparing. Finally, for C/C-carriers a better T1 detection rate was accompanied by reduced lag 1 sparing. This pattern of results could be explained by assuming that carriers of at least one T-allele (T+) do more strongly inhibit the pre-T1 distracter stream than participant with the C/C-genotype (T−). Inhibition can spill over to T1 and would then negatively affect T1 performance. For T/T-carriers reduced T1 performance is accompanied by increased T2 lag 1 performance. This could indicate that they are able to invest additional resources to oppose the spill over of inhibition, from which T2 performance at lag 1 benefits. If the lag 1 item is a distracter however, the representation of the distracter will be strengthened, and in turn triggers a stronger inhibition of subsequent items, such as T2 at lag 2. The relevance of inhibition triggered by the T1+1 item to T2 performance at short lags has been previously proposed in the boost and bounce theory of the AB (Olivers and Meeter, 2008). C/T-carriers might not spend additional resources, thus in spite of reduced T1 performance, T2 identification at lag 1 does not improve. Similarly, T2 identification at lag 2 does not suffer. Due to their less stringent inhibition of pre-T1 distracters C/C-carriers have little difficulty to identify T1, and also they do not need to devote additional resources to oppose accidental T1 inhibition. In consequence, lag 1 sparing will not be boosted and remains smaller than in the T/T-carriers. Put in a nutshell, our findings can be explained by assuming that the T-allele is detrimental to the ability to pick a target (T1) from an RSVP stream. In a dual-task dual-target context it is however the C-allele that determines how this affects the processing of the subsequent target. Its presence appears to be linked to a relatively weaker representation of items that appear around 100 ms after the first target and, in parallel, a reduced inhibition of items that follow the first target with a somewhat longer delay.

With respect to the second issue, we would like to point out that there are mixed findings in the literature regarding the precise influence of the DRD2 C957T genetic variants on distinct cognitive processes and on the effect's direction respectively. Colzato et al.'s findings indicate that the T/T-genotype (associated with high DRD2 availability but lower levels of striatal dopamine) is linked to a reduced AB. These findings have been challenged by a recent PET study (Slagter et al., 2012), where a high D2-like receptor availability in the striatum was found to be associated with a larger AB. Our results point in the direction of the PET study as they suggest that the C-allele (associated with higher striatal dopamine levels) and in particular the C/C genotype, is beneficial for performance in RSVP tasks. The only measure where T/T carriers excelled in the present study was lag 1 sparing, but only because of a combination of relatively better T2 performance at lag 1 and relatively worse T2 performance (larger AB) at lag 2 as compared to C-allele carriers. That is, the present results can be seen as partially in line with previous findings from our group reporting a better performance of C-allele carriers in a WM task. In these two studies, however, the effect was only visible in carriers homozygous for the T/T-genotype of CHRNA4 rs1044396, implicating that the effect of C957T on WM depends on a genetic interaction (Markett et al., 2010, 2011). In the literature, however, most studies report an association between the C-allele and poorer WM performance and executive functioning (Xu et al., 2007; Jacobsen et al., 2006). This apparent contradiction might be solved by taking into consideration that recently, for the C957T genotype a counteracting effect in the regional specificity was reported: the DRD2 genotype leads to differences in binding affinity, respectively availability, in striatal versus extrastriatal regions (Hirvonen et al., 2009a). In all extrastriatal regions the C/C-genotype is associated with the highest DRD2 binding potential whereas in the striatum the C/C-genotype is associated with the lowest binding potential, respectively availability. As pointed out above recent findings from experimental psychology suggest that on first sight even slightly different AB tasks may rely on different cognitive resources (Kelly and Dux, 2011). This suggests that AB performance, that is, the cognitive phenotype, depends on at least in part different neural circuits. If this is indeed the case then the emergence of different associations between DRD2 C957T and cognitive phenotype seems plausible. While behavioral work such as the present study and the one by Colzato et al. (2011) can point out that a gene locus is of relevance for a phenotype, only the combination of a molecular genetic approach with

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neuroimaging and pharmacological challenges can conclusively delineate the exact interplay between genes, dopaminergic baseline availability and neural circuits on a complex cognitive phenomenon as the AB. Future studies are warranted, and both the present findings and the findings by Colzato at al. provide evidence that such an endeavor would be reasonable and promising.

As a final note, replication of results from molecular genetics often fails because genetically heterogeneous samples are tested or possible gene-by-environment interactions confound the results (Montag et al., 2012). In the present study and the one from Colzato et al. (2011) only Caucasian participants, who were screened for neurological and psychiatric disorders took part. Moreover, no significant gender effects were observed. Thus, we are confident that we can rule out confounding influences of these factors.

In conclusion, even though we were not able to replicate the findings by Colzato et al. (2011), our results do support the finding of modulatory role of dopamine on cognitive processes involved in the AB. The underlying genetic markers as well as the nature of the modulation have yet to be elucidated.

Role of the funding source

The study was funded by the Laboratory of Neurogenetics of the University of Bonn. No one but the six authors was by any means involved in data analysis and the development of this manuscript.

Contributors

CM, AF and MR designed the study and CK programmed the paradigm; AF, CM and NW collected and analyzed the data, AF wrote the manuscript; CM, CK, SM, NW and MR edited the manuscript.

Conflict of interest

All authors declare that they have no conflicts of interest.

References

Akyürek, E.G., Hommel, B., Jolicoeur, P., 2007. Direct evidence for a role of working memory in the attentional blink. Mem. Cognit. 35, 621–627.

Baddeley, A., 1996. The fractionation of working-memory. Proc. Natl. Acad. Sci. USA 93 (24), 13468–13472.

Bellgrove, M.A., Mattingley, J.B., 2008. Molecular genetics of attention. Ann. N Y Acad. Sci. 1129, 200–212.

Bolton, J.L., Marioni, R.E., Deary, I.J., Harris, S.E., Stewart, M.C., Murray, G.D., Fowkes, F.G., Price, J.F., 2010. Association between polymorphisms of the dopamine receptor D2 and catechol-o-methyl transferase genes and cognitive function. Behav. Genet. 40 (5), 630–638.

Brozoski, T.J., Brown, R.M., Rosvold, H.E., Goldman, P.S., 1979. Cognitive deficit caused by regional depletion of dopamine in prefrontal cortex of Rhesus monkey. Science 205, 929–932.

Chun, M.M., Potter, M.C., 1995. A two-stage model for multiple target detection in rapid serial visual presentation. J. Exp. Psychol. Hum. Percept. Perform. 21, 109–127.

Colzato, L.S., Slagter, H.A., de Rover, M., Hommel, B., 2011. Dopamine and the management of attentional resources: genetic markers of striatal D2 dopamine predict individual differences in the attentional blink. J. Cogn. Neurosci. 23 (11), 3576–3585.

Colzato, L.S., van den Wildenberg, W., van der Does, W.A.J., Hommel, B., 2010. Genetic markers of striatal dopamine predict individual differences in dysfunctional, but not functional impulsivity. Neuroscience 170, 782–788.

Cools, R., D'Esposito, M., 2010. Dopaminergic modulation of flexible cognitive control in humans. In: Björklund, A., Dunnett, S., Iversen, L., Iversen, S. (Eds.), Dopamine Handbook. Oxford University Press, Oxford, pp. 249–260.

Cools, R., D'Esposito, M., 2011. Inverted-U-shaped dopamine actions on human working memory and cognitive control. Biol. Psychiatry 69, e113–e125.

Cools, R., Gibbs, S.E., Jagust, A., D'Miyakawa, W., Esposito, M., 2008. Working memory capacity predicts dopamine synthesis capacity in the human striatum. J. Neurosci. 28 (5), 1208–1212.

Cowan, N., 1999. An embedded-process model of working memory. In: Miyake, A., Shah, P. (Eds.), Models of Working Memory. Mechanisms of Active Maintenance and Executive Control. Cambridge University Press, Cambridge, pp. 62–101.

Dale, G., Arnell, K.M., 2011. How reliable is the attentional blink? Examining the relationships within and between attentional blink tasks over time. Psychol. Res. (Epub ahead of print), http://dx.doi.org/10.1007/s00426-011-0403-y.

Dehaene, S., Kerszberg, M., Changeux, J.-P., 1998. A neuronal model of a global workspace in effortful cognitive tasks. Proc. Natl. Acad. Sci. USA 95, 4529–14534.

Dell'Acqua, R., Jolic?ur, P., Luria, R., Pluchino, P., 2009. Reevaluating encoding-capacity limitations as a cause of the attentional blink. J. Exp. Psychol.—Hum. Percept. Perform. 35, 338–351.

Dux, P.E., Marois, R., 2009. The attentional blink: a review of data and theory. Atten. Percept. Psychophys. 71, 1683–1700.

Green, A.E., Munafò, M.R., DeYoung, C.G., Fossella, J.A., Fan, J., Gray, J.R., 2008. Using genetic data in cognitive neuroscience: from growing pains to genuine insights. Nat. Rev. Neurosci. 9 (9), 710–720.

Gross, J., Schmitz, F., Schnitzler, I., Kessler, K., Shapiro, K., Hommel, B., Schnitzler, A., 2004. Modulation of long-range neural synchrony reflects temporal limitations of visual attention in humans. Proc. Natl. Acad. Sci. USA 101 (35), 13050–13055.

Hirvonen, M., Laakso, A., Nagren, K., Rinne, J.O., Pohjalainen, T., Hietala, J., 2004. C957T polymorphism of the dopamine D2 receptor (DRD2) gene affects striatal DRD2 availability in vivo. Mol. Psychiatry 9, 1060–1061.

Hirvonen, M.M., Lumme, V., Hirvonen, J., Pesonen, U., Någren, K., Vahlberg, T., Scheinin, H., Hietala, J., 2009a. C957T polymorphism of the human dopamine D2 receptor gene predicts extrastriatal dopamine receptor availability in vivo. Prog. Neuropsychopharmacol. Biol. Psychiatry 33 (4), 630–636.

Hirvonen, M.M., Laakso, A., Någren, K., Rinne, J.O., Pohjalainen, T., Hietala, J., 2009b. C957T polymorphism of dopamine D2 receptor gene affects striatal DRD2 in vivo availability by changing the receptor affinity. Synapse 63, 907–912.

Jacobsen, L.K., Pugh, K.R., Mencl, W.E., Gelernter, J., 2006. C957T polymorphism of the dopamine D2 receptor gene modulates the effect of nicotine on working memory performance and cortical processing efficiency. Psychopharmacology 188, 530–540.

Janson, J., Kranczioch, C., 2011. Good vibrations, bad vibrations: oscillatory brain activity in the attentional blink. Adv. Cogn. Psychol. 7, 92–107.

Kelly, A.J., Dux, P.E., 2011. Different attentional blink tasks reflect distinct information processing limitations: an individual differences approach. J. Exp. Psychol. Hum. Percept. Perform. 37 (6), 1867–1873.

Kimberg, D.Y., D'Esposito, M., Farah, M.J., 1997. Effects of bromocriptine on human subjects depend on working memory capacity. Neuroreport 8, 3581–3585.

Please cite this article as: Felten, A., et al., The DRD2 C957T polymorphism and the Attentional Blink—A genetic association study. European Neuropsychopharmacology (2012), http://dx.doi.org/10.1016/j.euroneuro.2012.09.010

Kranczioch, C., Debener, S., Engel, A.K., 2003. Event-related potential correlates of the attentional blink phenomenon. Brain Res. Cogn. Brain Res. 17 (1), 177–187.

Kranczioch, C., Debener, S., Maye, A., Engel, A.K., 2007. Temporal dynamics of access to consciousness in the attentional blink. NeuroImage 37 (3), 947–955.

Livesey, E.J., Harris, I.M., 2011. Target sparing effects in the attentional blink depend on type of stimulus. Atten. Percept. Psychophys. 73 (7), 2104–2123.

Luciana, M., Depue, R.A., Arbisi, P., Leon, A., 1992. Facilitation of working memory in humans by a D2 dopamine receptor agonist. J. Cogn. Neurosci. 4 (1), 58–68.

Markett, S.A., Montag, C., Reuter, M., 2010. The association between dopamine DRD2 polymorphisms and working memory capacity is modulated by a functional polymorphism on the nicotinic receptor gene CHRNA4. J. Cogn. Neurosci. 22, 1944–1954.

Markett, S., Montag, C., Walter, N.T., Reuter, M., 2011. Evidence for the modality independence of the genetic epistasis between the dopaminergic and cholinergic system on working memory capacity. Eur. Neuropsychopharmacol. 21 (2), 216–220.

Marois, R., Yi, D.J., Chun, M.M., 2004. The neural fate of consciously perceived and missed events in the attentional blink. Neuron 41 (3), 465–472.

Miyake, A., Friedman, N.P., Emerson, M.J., Witzki, A.H., Howerter, A., Wager, T.D., 2000. The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: a latent variable analysis. Cogn. Psychol. 41 (1), 49–100.

Montag, C., Jurkiewicz, M., Reuter, M., 2012. The role of the Catechol-O-Methyltransferase (COMT) gene in personality and related psychopathological disorders. CNS Neurol. Disord. Drug Targets 11 (3), 236–250.

Olivers, C.N.L., Meeter, M., 2008. A boost and bounce theory of temporal attention. Psychol. Rev. 115, 836–863.

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. J. Exp. Psychol. Learn. Mem. Cogn. 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? J. Exp. Psychol. Hum. Percept. Perform. 18, 849–860.

Sergent, C., Baillet, S., Dehaene, S., 2005. Timing of the brain events underlying access to consciousness during the attentional blink. Nat. Neurosci. 8 (10), 1391–1400.

Shapiro, K.L., Raymond, J.E., Arnell, K.M., 1994. Attention to visual pattern information produces the attentional blink in rapid serial visual presentation. J. Exp. Psychol. Hum. Percept. Perform. 20 (2), 357–371.

Slagter, H.A., Tomer, R., Christian, B.T., Fox, A.S., Colzato, L.S., King, C.R., Murali, D., Davidson, R.J., 2012. PET evidence for a role for striatal dopamine in the attentional blink: functional implications. J. Cogn. Neurosci. 24 (9), 1932–1940.

Visser, T.A.W., Zuvic, S.M., Bischof, W.F., Di Lollo, V., 1999. The attentional blink with targets in different spatial locations. Psychon. Bull. Rev. 6, 432–436.

Xu, H., Kellendonk, C.B., Simpson, E.H., Keilp, J.G., Bruder, G.E., Polan, H.J., Kandel, E.R., Gilliam, T.C., 2007. DRD2 C957T polymorphism interacts with the COMT Val158Met polymorphism in human working memory ability. Schizophr. Res. 90, 104–107.

Please cite this article as: Felten, A., et al., The DRD2 C957T polymorphism and the Attentional Blink—A genetic association study. European Neuropsychopharmacology (2012), http://dx.doi.org/10.1016/j.euroneuro.2012.09.010