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Spontaneous eye blink rate (EBR) predicts poor performance in high-stakes situations

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ABSTRACT

Although the existence of 'choking under pressure' is well-supported by research, its biological underpinnings are less clear. In this research, we examined two individual difference variables that may predict whether people are likely to perform poorly in high-incentive conditions: baseline eye blink rate (EBR; reflecting dopamine system functioning) and baseline anterior hemispheric asymmetry (an indicator of goal-directed vs. stimulus driven processing). Participants conducted a switch task under control vs. incentive conditions. People low in EBR were generally capable of improving their performance when incentives were at stake, whereas people high in EBR were not. Hemispheric asymmetry did not predict performance. These findings are consistent with the idea that suboptimal performance in high-stakes conditions may stem from the neuromodulatory effects of dopamine.

1. Introduction

During their lives, people often find themselves in situations where good performance yields immediate monetary or social rewards. Consider, for example, music auditions, sports finals, and college entrance exams. Both inside and outside science, high-stakes situations such as these are often assumed to bring out the best in people. Nevertheless, a growing body of research indicates that high-stakes situations have the potential to cause *choking under pressure*—i.e., worse-than-normal performance when pressure to perform is very high (Beilock and Carr, 2001; Baumeister, 1984). Prior psychological studies indicate that such incentive-triggered performance decrements are due to momentary impairments in working memory and attention regulation (Beilock and Carr, 2001; Beilock et al., 2004; Lewis and Linder, 1997). Yet, at present, much less is known about the biological underpinnings of suboptimal performance in high-stakes situations (Boere et al., 2016; Braver et al., 2014; Chib et al., 2012, 2014; Lee and Grafton, 2015; Mobbs et al., 2009; Silston and Mobbs, 2014). Here, we examine two candidate biological, individual differences that may make people more susceptible to such performance impairments. We consider individual differences in baseline dopamine levels in the midbrain (indicated by spontaneous Eye Blink Rate; EBR) and individual differences in baseline hemispheric asymmetry (measured with electroencephalography; EEG).

1.1. The mesolimbic and mesocortical dopamine pathways

Originating in the ventral tegmental area and the substantia nigra, the brain's ascending dopamine pathways affect a wide range of cognitive functions, such as working memory and cognitive control (Cools and D'Esposito, 2011). In particular, dopamine (DA) may be involved in maintaining a balance between cognitive flexibility and cognitive stability, which is considered important for optimal cognitive control (Cools and D'Esposito, 2011; Dreisbach et al., 2005; Jongkees and Colzato, 2016). It has been suggested that this balance depends on dopaminergic functioning in the striatum and prefrontal cortex (PFC; Cools and D'Esposito, 2011). More specifically, D1 receptor signaling in the PFC is thought to be involved in the facilitation of stable information maintenance, whereas D2 receptor signaling in the striatum is thought to serve as a gating mechanism responsible for letting through goal-relevant information and preventing distraction (Zhang et al., 2015; Cools and D'Esposito, 2011; Braver and Cohen, 1999). Interestingly, DA levels in both the striatum and PFC are thought to follow an inverted U-shape, with too high or low levels of DA impairing cognitive functioning (Arnsten, 2009; Cools and D'Esposito, 2011; Aarts et al., 2014; cf. Yerkes and Dodson, 1908), suggesting that the balance between cognitive flexibility and stability requires moderate levels of DA. Moreover, this idea implies that *choking* on performance tasks that require cognitive control can be induced by raising DA levels beyond their optimum. Given that DA is released when valuable outcomes are at stake (Howe et al., 2013; Schultz, 2007), we will test this idea by incentivizing performance on a task requiring cognitive control.

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Baseline dopamine levels can be estimated indirectly and non-invasively, by measuring spontaneous eye blink rate (EBR; see Jongkees and Colzato, 2016, for a review). DA activity and EBR are positively related (Jongkees and Colzato, 2016; Zhang et al., 2015), in the sense that higher EBR indicates stronger dopamine transmission. EBR can reflect both D1 and D2 receptor activity (Jongkees and Colzato, 2016), although it may be more strongly related to the D2 receptor system (Groman et al., 2014; Jongkees and Colzato, 2016). In particular, baseline eye blink rates measured at rest (i.e., tonic EBR) may specifically relate to D2 receptor functioning (Slagter et al., 2015).

1.2. Hemispheric asymmetry

A hallmark finding from psychological research is that incentive-triggered performance impairments often go hand in hand with the subjective experience of performance anxiety and distracting, task-unrelated thoughts (e.g., Beilock and Gray, 2007; Eysenck et al., 2007). Importantly, such subjective experiences are often suggested to be due to a disbalance between two broad attentional systems (Corbetta and Shulman, 2002; Eysenck et al., 2007): the goal-directed system (including the dorsal posterior parietal and large parts of the frontal cortex; lesions in this circuitry cause deficits in voluntarily directing attention to different locations; Halligan et al., 2003) and the stimulus-driven system (including the inferior frontal cortex and temporoparietal cortex; right lateralized; lesions in this network tend to cause spatial neglect; He et al., 2007). Performance anxiety is associated with a pronounced emphasis of the stimulus-driven system (Eysenck et al., 2007). As the stimulus-driven system is lateralized in the right hemisphere, from where it disrupts the goal-directed system, performance anxiety is thought to result in measurable hemispheric asymmetry (Harmon-Jones et al., 2010)—i.e., greater activity in the right hemisphere's frontal cortex, compared to the left hemisphere's frontal cortex.

1.3. The present research

To test our ideas, we used an incentivized task switch paradigm (adapted from Colzato et al., 2010). In this task, on each trial, people are exposed to a stimulus (in this case, a digit and a letter) on which they have to perform either of two tasks (in this case, odd/even vs. vowel/consonant judgments). In research that used this paradigm, a well-replicated finding is that people perform worse on *switch trials* (trials in which people perform a different task than on the previous trial) vs. *repeat trials* (trials in which people perform the same task as on the previous trial; Monsell, 2003).

Importantly, performance on the switch task is thought to rely on PFC functioning (Sohn et al., 2000; Gnadt and Andersen, 1988; Corbetta and Shulman, 2002). Also, previous research indicated that performance on the switch task is related to the catechol-*O*-methyltransferase (COMT) gene ($Val^{158}Met$ polymorphism), a gene that is involved in generating an enzyme that in turn affects the supply of dopamine (Colzato et al., 2010). Interestingly, having the $Val^{158}Met$ polymorphism also seems to be related to right hemisphere frontal asymmetry (Wacker et al., 2013). So, several previous findings suggest that the switch task may well respond to dopamine-related and hemispheric-asymmetry-related processes.

A novel aspect of our version of this task is that we will incentivize participants' performance in an all-or-nothing fashion. Specifically, one group of participants learns that they will lose[1] sum of money if they fail to meet a pre-specified performance criterion (see Chib et al., 2012). A control group of participants learns that they may lose money, but that this loss does not depend on their performance. So, crucially, all participants will be exposed to information regarding a potential loss and their performance; however, the potential loss is only contingent on people's performance in the experimental condition. We examine how this incentive manipulation affects performance (in general, but also on switch trials specifically) and we examine how this effect relates to EBR and hemispheric asymmetry (at baseline and during task performance).

We hypothesize that participants are more prone to performance decrements when their monetary payoff depends on performance, relative to when their payoff is not contingent on performance. Furthermore, we expect that people with high EBR are more susceptible to incentive-triggered performance impairments, compared to people with low EBR. Finally, we hypothesize that people who are inclined toward having greater activity in the right frontal cortex (i.e., at baseline) should be more prone to incentive-triggered drops in performance.

In the online Supplementary information, we present a pilot study in which we test our incentivized switch task. In the main text, we present a study that uses the same task, adding measures of EBR and hemispheric asymmetry.

2. Material and methods

2.1. Participants, design, and overview

Thirty-eight undergraduate students participated in the study (mean age = 21.5, 19 females). A priori exclusion criteria included (1) caffeine use twelve hours prior to the experiment, (2) left-handedness, (3) current substance abuse, (4) neurological conditions, and (5) mental disorders. Data from one participant was excluded due to performance below chance level on the task. Physiological data from another participant was excluded because of equipment failure. Participants were randomly assigned to the loss vs. the control condition. Participants earned €10 in exchange for their participation (see below). All participants gave written informed consent. The study was approved by the local ethics committee (Faculty of Social Sciences, Utrecht University). For a discussion of the limitations of using samples from western, educated, industrialized, rich and democrat communities, we refer the reader to Henrich et al. (2010).

2.2. Procedure

After preparing the participants for EEG data collection, participants first underwent two periods of resting state measurements. That is, participants were asked to relax with their eyes open (5 min; while we measured hemispheric asymmetry and EBR) and their eyes closed (2 min, while we measured hemispheric asymmetry). Then, the incentivized switch task was started.

Participants first familiarized themselves with the task. Specifically, they completed 2 instruction runs (34 trials; see below for a description of the trials), which included performance feedback after every trial, and 4 practice runs (68 trials), which included no feedback. Then, they learned that the experiment was about to start. Also, to make sure participants performed the task to the best of their ability, it was mentioned that they would "probably be able to perform better than they did during the practice block". Specifically, they were asked to improve their performance with 20%. Next, they performed 4 experimental runs (68 trials). Subsequently, they received the *incentive manipulation*. Specifically, they were told that they had reached their optimal performance level and they were asked to "retain their performance, or improve their performance even further" in the second block. In the loss condition, participants were told that whether they would lose their reward (€10) depended on their performance. In the control condition, participants instead learned that a lottery (after the experiment) would determine whether they would lose their payment. So, like in the

[1] Our incentive manipulation was designed to strongly increase the importance of success for participants—i.e., more strongly than typical within-subjects incentive manipulations in experimental psychology. After all, prior work (Ariely et al., 2009) suggests that stronger (vs. weaker) incentives are more likely to impair (vs. improve) performance. To strengthen our manipulation, we used an all-or-none reward schedule (i.e., participants receive nothing if they fail) and loss framing.

experimental condition, potential loss of payment was mentioned, but loss of payment was not performance-contingent. In reality, all participants received €10. Loss instructions were used (rather than gain instructions), as loss aversion may play a role in incentive-triggered drops in performance (Chib et al., 2012). After the manipulation, participants performed 4 more experimental runs (17 trials per run; 68 trials in total). The comparison of interest was performance (speed and accuracy) before vs. after the incentive manipulation.

2.2.1. Trials

On each trial, a letter and a digit were presented in a quadrant of a square on the computer screen (see Fig. 1). In the first trial of each run, these stimuli appeared in the upper left quadrant; in the following 16 trials of each run, stimuli were displayed clockwise in the next quadrant. The upper quadrants were assigned to the letter task and the lower quadrants to the digit task, so that the display location served as a task cue, and that the task changed predictably (i.e., switch and repeat trials alternated). Depending on the task, the relevant character was either a letter or a digit. The second and irrelevant character was a member of the other category. Participants were asked to indicate whether the relevant character was a vowel or consonant (upper quadrants) or whether it was odd or even (lower quadrants) by pressing the 'M' and 'Z' keys on the keyboard, respectively. Letters and digits were selected randomly from predetermined sets (GKMRAEIU and 23456789, respectively). The position of the task-relevant character was randomly determined on each trial. The next trial started immediately upon the participant's response (so, the response-stimulus interval [RSI] was zero). Between runs, there were brief breaks (10 s).

2.3. Analyses

2.3.1. Task performance

We analyzed the effect of the incentive manipulation in two ways. First, we compared performance (speed and accuracy) during the block before the manipulation (henceforth: block 1) vs. the block after the manipulation (henceforth: block 2), as a function of incentive condition. Second, we conducted the same analysis in a more fine-grained manner, analyzing data per run (rather than per block). Reaction times (RTs) that deviated more than 3SD's from the individual participants' mean RT were excluded from analysis. As the first trial of each run was neither a switch nor a repeat trial, these trials were excluded from analysis as well. We analyzed RTs only for trials on which people were accurate.

2.3.2. Eye blink rate (EBR)

A BioSemi ActiveTwo system (BioSemi Inc., the Netherlands) was used to record EBR. Eye blinks were recorded for a 5-minute time period (Colzato et al., 2009; Colzato et al., 2008). The vertical Electrooculogram (EOG) was used to detect eye blink rates, by recording the voltage difference between two electrodes placed above and below the left eye (which were applied after the skin was scrubbed with Nuprep Skin Prep Gel, Weaver and Company, Aurora, CO). Recordings did not take place after 5 p.m., because spontaneous EBR is generally stable during daytime, but increases in the evening (Barbato et al., 2000; Kowal et al., 2011). Data analysis was performed with Brain Vision Analyzer (Brain Products GmbH, Munich, Germany). Eye blinks were automatically detected using a built-in algorithm (Gratton et al., 1983). Individual EBR was calculated by dividing the total number of eye blinks during the 5-minute measurement by five.

2.3.3. EEG

EEG activity was recorded from 32 electrodes, placed according to the international 10–20 system, using the ActiveTwo system (BioSemi Inc., The Netherlands). In order to measure hemispheric asymmetry, following Harmon-Jones and Allen (1998), activity from frontal (F3, F4) and parietal (P3, P4) scalp sites was first referenced to Cz. Cz was used as a reference since it was previously shown to yield similar results to the use of both the average of all scalp electrodes and the average of the electrodes placed on the mastoids (Tomarken et al., 1992; Harmon-Jones and Allen, 1997).

After referencing to Cz, the sampling rate was transformed from 2048 Hz to 256 Hz using a spline interpolation. Data were then filtered by means of a high-pass filter (at 1 Hz) and a low-pass filter (at 30 Hz; Laufs et al., 2003). Filtering took place before segmentation in order to minimize the effect of discontinuities on the filter. Subsequently, three types of segments were created based on marker positions: (a) a 5-min segment for the eyes-open baseline measurement, (b) a 2-min segment for the eyes-closed baseline measurement, and (c) 2-min segments for measurement during task performance. For all segments, ocular correction was performed (Gratton et al., 1983). Segments were then split into two-second epochs overlapping by 75% to improve the temporal resolution (Davidson et al., 1990). Data containing artifacts were semi-automatically rejected; rejection decisions were made a priori by two trained reviewers. Power density (μV^2/Hz) in the alpha range (8–13 Hz) was estimated by means of a fast Fourier transform (FFT) on each epoch. Subsequently, the epochs were averaged for each participant and electrode. Alpha power density was then log-transformed to normalize the distribution (Davidson, 1988). Following Harmon-Jones and Allen (1998), an anterior asymmetry index (log right alpha power density – log left alpha power density) was computed as a measure of asymmetry. We computed separate anterior asymmetry indices for the eyes-open baseline and the eyes-closed baseline. These were averaged to provide one index of anterior hemispheric asymmetry (Harmon-Jones and Allen, 1998). In addition, we computed indices that reflected hemispheric asymmetry during task performance, separately for block 1 and block 2. Since alpha power is inversely related to activity (Harmon-Jones and Allen, 1998), higher scores on the indices reflect greater left-hemisphere activity.

3. Results

3.1. Descriptives

In our sample, participants blinked on average 15.1 times per minute (SD = 10.6, range 1.0 to 43.6). EBR was similar in the loss condition

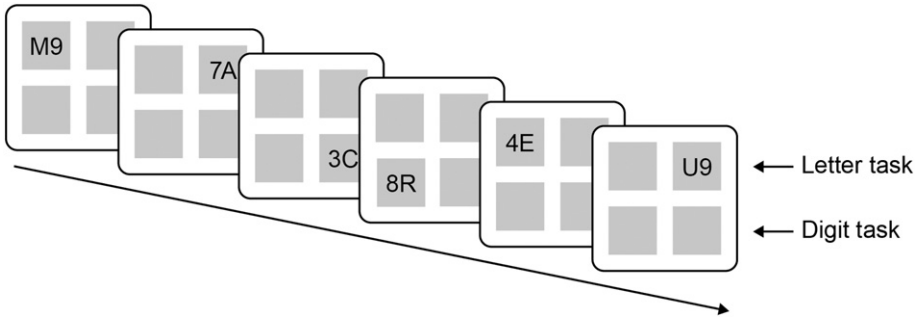

Fig. 1. Overview of the incentivized switch task.

($M = 15.5$, $SD = 12.5$, range: 1.0–43.6) and the control condition ($M = 14.8$, $SD = 8.6$, range: 2.6–32.6). Other studies that used the same methodology (EOG) in similar, healthy populations tend to report very similar values (for a systematic review, see Jongkees and Colzato, 2016).

3.2. Block level

3.2.1. Response times (RTs)

We started out by analyzing the RTs (ms) as a function of the main task variables (block, trial type, incentive condition), not yet including EBR and hemispheric asymmetry as predictors. Accordingly, we analyzed participant's RTs with a general linear model (GLM) analysis, with block (block 1 vs. block 2, within-subjects), trial type (switch vs. repeat, within-subjects), and incentive condition (loss vs. control, between-subjects) as independent variables. This analysis yielded only significant main effects of block, $F(1, 35) = 34.89$, $p < 0.01$, $\eta^2_p = 0.50$, and of trial type, $F(1, 35) = 135.44$, $p < 0.01$, $\eta^2_p = 0.79$. These effects indicated that people were faster during the second block ($M_{2nd} = 981$, $SE_{2nd} = 43$ vs. $M_{1st} = 1075$, $SE_{1st} = 46$) and on repeat trials ($M_{repeat} = 844$, $SE_{repeat} = 35$ vs. $M_{switch} = 1212$, $SE_{switch} = 55$), respectively. There was no significant main effect of incentive condition, $F(1, 35) = 0.04$, $p = 0.85$, $\eta^2_p < 0.01$, nor did incentive condition moderate any of the other effects, $Fs < 0.71$.

3.2.1.1. Eye blink rate (EBR). To examine whether EBR moderated the effects of incentive condition, we submitted RTs to the same GLM as above, now also including EBR (standardized) as a between-subjects continuous independent variable. This analysis again revealed the main effect of block, $F(1, 32) = 33.00$, $p < 0.01$, $\eta^2_p = 0.51$, and trial type, $F(1, 32) = 135.44$, $p < 0.01$, $\eta^2_p = 0.81$. There was no significant main effect[2] of EBR, $F(1, 32) = 1.90$, $p = 0.18$, $\eta^2_p = 0.056$. However, there was a block × trial type × incentive condition × EBR four-way interaction, $F(1, 32) = 4.64$, $p = 0.04$, $\eta^2_p = 0.13$. To interpret this pattern of results, we plotted the pattern of estimated means separately for participants low (−1SD) vs. high (+1SD) in EBR (Fig. 2). In turn, we analyzed changes in RT from block 1 to block 2 separately for both incentive conditions and for both types of trials, examining how these changes were different for people high vs. low in EBR.

In the control condition (Fig. 2, dotted lines), on repeat trials, participants were faster in block 2 ($M = 786$, $SE = 41$) vs. block 1 ($M = 865$, $SE = 45$), $F(1, 17) = 8.34$, $p = 0.01$, $\eta^2_p = 0.33$. This speed-up was not moderated by EBR, $F(1, 17) = 0.42$, $p = 0.53$, $\eta^2_p = 0.02$. In the control condition, on switch trials, participants were faster in block 2 ($M = 1163$, $SE = 78$) vs. block 1 ($M = 1256$, $SE = 88$), $F(1, 17) = 10.96$, $p < 0.01$, $\eta^2_p = 0.39$. Also here, this speed-up was not moderated by EBR, $F(1, 17) = 0.61$, $p = 0.44$, $\eta^2_p = 0.04$.

In the loss condition (Fig. 2, solid lines), on repeat trials, participants were faster in block 2 ($M = 791$, $SE = 53$) vs. block 1 ($M = 864$, $SE = 48$), $F(1, 15) = 14.75$, $p < 0.01$, $\eta^2_p = 0.50$. Again, this speed-up was not moderated by EBR, $F(1, 15) = 0.45$, $p = 0.51$, $\eta^2_p = 0.03$. In the loss condition, on switch trials, participants were faster in block 2 ($M = 1118$, $SE = 73$) vs. block 1 ($M = 1232$, $SE = 67$), $F(1, 15) = 14.46$, $p < 0.01$, $\eta^2_p = 0.49$. Importantly, the extent of this speedup was moderated by EBR, $F(1, 15) = 5.06$, $p = 0.04$, $\eta^2_p = 0.25$. Specifically, people low in EBR became significantly faster from block 1 ($M = 1318$, $SE = 88$) to block 2 ($M = 1146$, $SE = 95$), $F(1, 15) = 19.11$, $p < 0.01$, $\eta^2_p = 0.56$, but people high in EBR did not become significantly faster from block 1 ($M = 1146$, $SE = 89$) to block 2 ($M = 1090$, $SE = 96$), $F(1, 15) = 2.01$, $p = 0.18$, $\eta^2_p = 0.12$.

[2] We explored whether EBR was correlated with RTs before the incentive manipulation, to check consistency with earlier studies (Zhang et al., 2015). As in prior work, EBR was associated with lower RTs. This relationship was not significant in repeat trials, $r(34) = -0.15$, $p = 0.398$, and marginally significant in switch trials, $r(34) = 0.29$, $p = 0.086$.

Together, these results are consistent with the idea that incentive-triggered increases in dopamine enhance performance in people who have low levels of DA (low EBR), but not in people who have high levels (high EBR). We provide further interpretation in the Discussion.

3.2.1.2. Hemispheric asymmetry. To examine whether hemispheric asymmetry moderated the effects of incentive condition, we again analyzed participants' RTs with a general linear model (GLM) analysis, with block (block 1 vs. block 2, within-subjects), trial type (switch vs. repeat, within-subjects), and incentive condition (loss vs. control, between-subjects) as independent variables. Now, we also added baseline hemispheric asymmetry as a between-subjects, continuous predictor. As before, this analysis yielded main effects of block, $F(1, 32) = 115.94$, $p < 0.01$, $\eta^2_p = 0.78$, and trial type, $F(1, 32) = 29.49$, $p < 0.01$, $\eta^2_p = 0.48$. There was no main effect of baseline hemispheric asymmetry, $F(1, 32) = 0.35$, $p = 0.56$, $\eta^2_p = 0.01$, nor did baseline hemispheric asymmetry interact with any of the other predictors, $Fs < 3.33$.

3.2.2. Accuracy

Like we did with the RTs, we started out by analyzing accuracy scores as a function of the main task variables (block, trial type, incentive condition), not yet including EBR and hemispheric asymmetry as predictors. This initial GLM analysis revealed only a significant main effect of trial type, $F(1, 35) = 55.39$, $p < 0.01$, $\eta^2_p = 0.61$, indicating that people were more accurate on repeat trials (M = 97.6%, SE = 0.4%) than on switch trials (M = 93.7%, SE = 0.8%).

We added EBR as a predictor to the model in the previous section. This analysis again revealed the significant main effect of trial type, $F(1, 32) = 52.66$, $p < 0.01$, $\eta^2_p = 0.62$. The main effect of EBR was not significant, $F(1, 32) = 0.43$, $p = 0.52$, $\eta^2_p = 0.01$, nor did EBR interact with any of the other predictors, $Fs < 1.59$. Then, we added baseline hemispheric asymmetry as a predictor (instead of EBR). This analysis again revealed the main effect of trial type, $F(1, 32) = 48.54$, $p < 0.01$, $\eta^2_p = 0.60$. The main effect of baseline hemispheric asymmetry was not significant, $F(1, 32) = 1.00$, $p = 0.32$, $\eta^2_p = 0.03$, nor did baseline hemispheric interact with any of the other predictors, $Fs < 2.08$.

3.3. Run level

Our pilot study suggested that the effect of incentives on performance was especially pronounced directly after the manipulation, i.e., especially in the first run of the second block. Findings from the pilot study are summarized in Fig. 3 (top panels) and reported in more detail as Supplementary online information. As these pilot findings suggest that the effect of incentives was short-lived in this paradigm, it is potentially informative to also analyze task performance in a more fine-grained manner, examining performance changes right after the manipulation.

3.3.1. Response times (RTs)

To take such a fine-grained perspective, we first analyzed participants' RTs (ms) with a GLM, with run (i.e., the eight runs which together made up blocks 1 and 2, within-subjects), trial type (switch vs. repeat, within-subjects), and incentive condition (loss vs. control, between-subjects) as independent variables. This analysis yielded main effects of run, $F(7, 245) = 10.36$, $p < 0.01$, $\eta^2_p = 0.23$, and of trial type, $F(1, 35) = 132.70$, $p < 0.01$, $\eta^2_p = 0.79$. Like in the pilot study, these effects were qualified by the run × trial type × incentive condition three-way interaction, $F(7, 245) = 2.22$, $p = 0.03$, $\eta^2_p = 0.06$.

Inspection of the pattern of means (Fig. 3) suggested that RTs slowed down immediately after the incentive manipulation (i.e., the first run of the second block), in the loss condition (not in the control condition), perhaps particularly on switch trials. To estimate the extent of this slowdown at the first run of the second block, we computed a post-hoc contrast that compared the RT of that run (i.e., run 2.1 in Fig. 3) to the RTs of

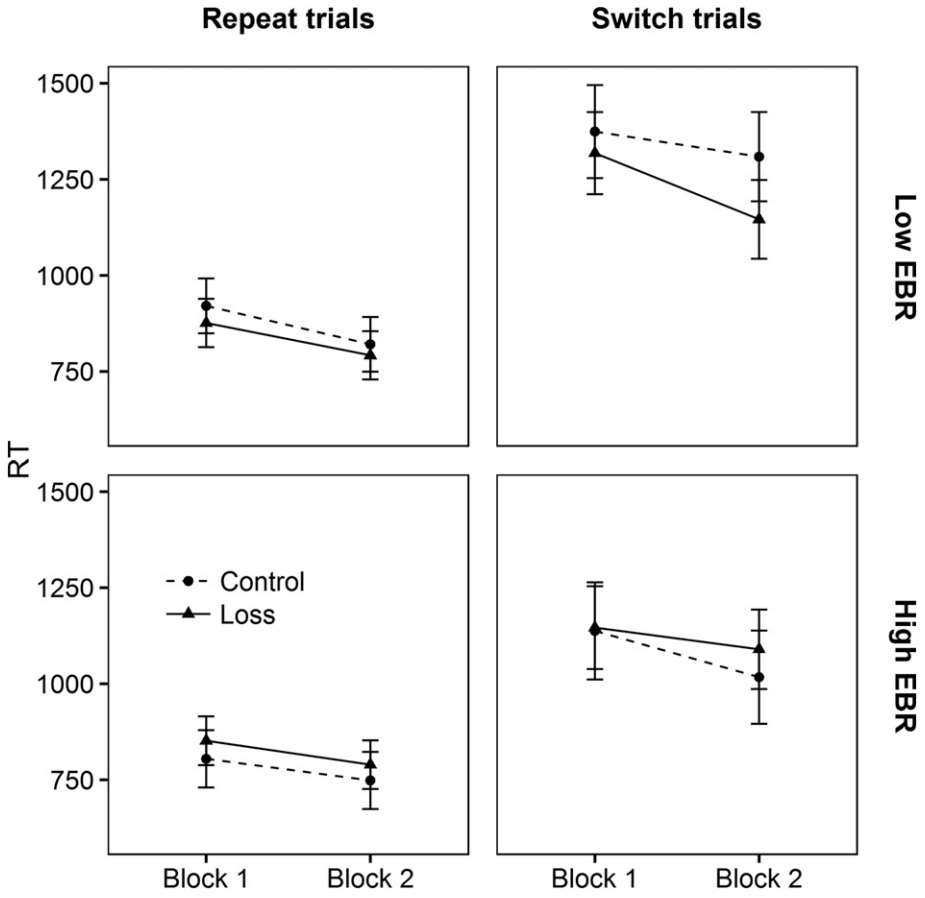

Fig. 2. Block-level reaction times (ms) as a function of eye blink rate (EBR) and incentive condition. Top panels show estimated means for low-EBR participants (−1SD); bottom panels show estimated means for high-EBR participants (+1SD). Please note that EBR was treated as a continuous variable in all analyses, including the GLM on which this plot was based. Left panels: Repeat trials. Right panels: Switch trials. Error bars reflect standard errors around the estimate.

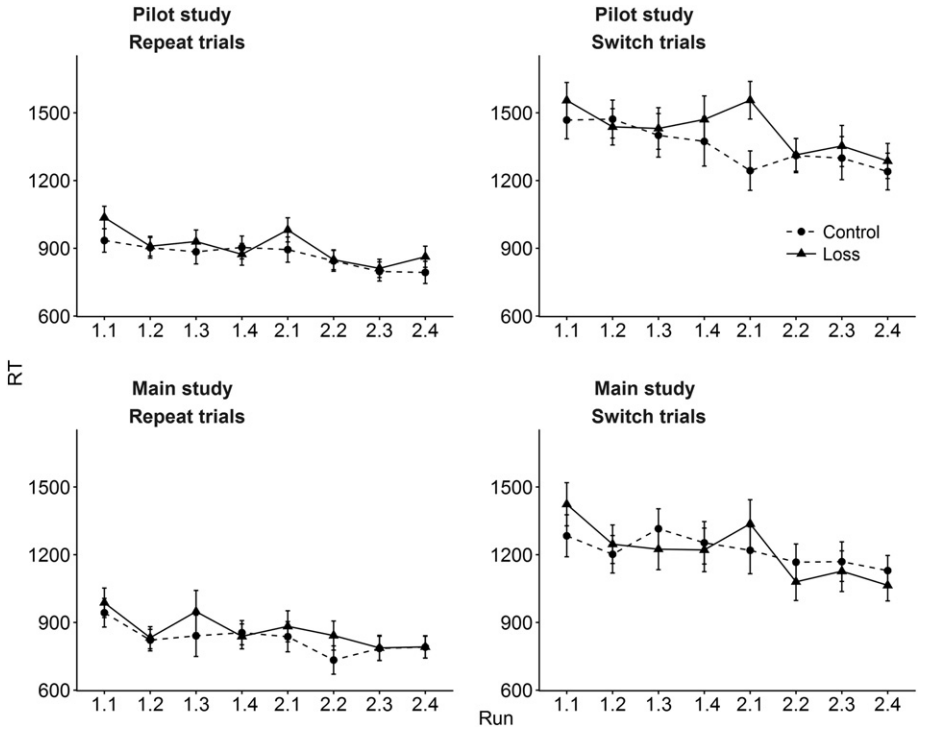

Fig. 3. Run-level reaction times (ms) as a function of incentive condition. Top panels depict estimated means found in a pilot study (N = 44), of which methods and results are reported in more detail as Supplementary online information. Bottom panels depict estimated means from the main study. While the slowdown after the manipulation (in run 2.1) on switch trials was more pronounced in the pilot study, we were still interested to examine whether this slowdown was modulated by EBR and hemispheric asymmetry. Left panels: Repeat trials. Right panels: Switch trials. Error bars reflect standard errors around the estimate.

all seven other runs. Specifically, this contrast was computed as follows: First, we computed the average RT of all runs except the first run of the second block (i.e., runs 1.1, 1.2, 1.3, 1.4, 2.2, 2.3, and 2.4 in Fig. 3). Next, we subtracted this average RT from the average RT of the run of interest (i.e., run 2.1 in Fig. 3). So, in vector notation, this contrast can be represented as [−1/7 −1/7 −1/7 −1/7 1 −1/7 −1/7 −1/7]. We computed the contrast separately for switch and repeat trials.

Then, we conducted a GLM analysis that examined whether this contrast was affected by trial type (switch vs. repeat, within-subjects) and incentive condition (loss vs. control, between-subjects). This analysis yielded no significant main effect of trial type, $F(1, 35) = 2.86$, $p = 0.10$, $\eta^2_p = 0.08$, and no significant main effect of condition, $F(1, 35) = 1.54$, $p = 0.22$, $\eta^2_p = 0.04$. However, the trial type × condition interaction approached significance, $F(1, 35) = 4.11$, $p = 0.05$, $\eta^2_p = 0.11$. So, although the slowdown right after the manipulation (on switch trials) was less pronounced than it was in the pilot study (where we found $\eta^2_p = 0.26$ for the trial type × condition interaction, see online Supplementary information), these findings suggest that the incentive manipulation may have had an effect that was short-lived, occurring directly after it was delivered. Specifically, in the loss condition, the mean value of the contrast was 139 ($SE = 54$) for switch trials and 22 ($SE = 39$) for repeat trials. This means that people were 139 ms (switch trials) and 22 ms (repeat trials) slower in the run right after the manipulation, relative to all other runs. This pattern was not present in the control condition (switch trials: $M = 2$, $SE = 53$; repeat trials: $M = 13$, $SE = 38$). In what follows, we will explore whether this contrast (i.e., the extent of the slowdown in run 2.1) was moderated by EBR and hemispheric asymmetry.

3.3.1.1. Eye blink rate (EBR). We conducted the same GLM analysis as in the previous section, which examined whether the contrast was moderated by trial type and incentive condition. However, we now also included EBR as an additional predictor. This analysis yielded no significant main effect of EBR, $F(1, 32) = 0.08$, $p = 0.78$, $\eta^2_p < 0.01$, nor did EBR interact with any of the other effects, Fs < 0.57. This analysis thus revealed no evidence that the slowdown right after the manipulation was different for people low vs. high in EBR.

3.3.1.2. Hemispheric asymmetry. We did the same analysis as in the previous section, now with hemispheric asymmetry (instead of EBR) as a predictor. There was no main effect of baseline hemispheric asymmetry, $F(1, 32) = 0.02$, $p = 0.880$, $\eta^2_p < 0.01$, nor did baseline hemispheric asymmetry interact with any of the other predictors, Fs < 0.78. So, this analysis revealed no evidence that the slowdown right after the manipulation was related to hemispheric asymmetry.

3.3.2. Accuracy

For completeness, we followed the same procedures to analyze accuracy scores on the run level. We started out by computing the contrast that compares the run after the manipulation to all other runs, now with accuracy scores rather than RTs. Then, we conducted a GLM analysis to examine whether our contrast was affected by trial type (switch vs. repeat, within-subjects) and incentive condition (loss vs. control, between-subjects). No effects were significant, Fs < 1.06. After adding EBR as a predictor, the main effect of EBR was not significant, $F(1, 32) = 0.14$, $p = 0.71$, $\eta^2_p < 0.01$, nor did EBR interact with any of the other effects, Fs < 1.59. After adding hemispheric asymmetry as a predictor (instead of EBR), the main effect of baseline hemispheric asymmetry was not significant, $F(1, 32) = 0.15$, $p = 0.71$, $\eta^2_p < 0.01$, nor did baseline hemispheric asymmetry interact with any of the other predictors, Fs < 1.16. So, like for the RTs, these analyses did not support the idea that EBR or hemispheric asymmetry was related to short-lived changes in performance.

4. Discussion

Various studies have shown that incentive-triggered performance decrements are caused by momentary impairments in working memory and attention regulation (Beilock and Carr, 2001; Beilock et al., 2004; Lewis and Linder, 1997). However, little is known about the biological underpinnings of such performance decrements. Therefore, the aim of the present research was to examine whether individual differences in baseline dopamine levels in the midbrain and in baseline hemispheric asymmetry could make people more susceptible to incentive-triggered performance decrements.

To investigate the first of these correlates, we examined whether people with high EBR (indicative of higher levels of dopamine) were more prone to performance decrements than people with low EBR (indicative of lower levels of dopamine). Given that DA levels are assumed to follow an inverted U-shape (Arnsten, 2009; Cools and D'Esposito, 2011; Aarts et al., 2010; cf. Yerkes and Dodson, 1908), we expected that incentives would more readily push dopamine levels beyond their optimum level in participants with higher EBR, thwarting performance, whereas this incentive-triggered DA release should enhance performance in participants with low EBR. Consistent with this idea, and in line with prior work (Aarts et al., 2010), findings indicated that people low in EBR were generally capable of improving their performance after the incentive manipulation, whereas people high in EBR were not.

Strictly, however, this study does not provide direct support for the idea that dopamine is involved in *choking*. After all, choking suggests a within-subjects drop in performance—not merely a failure to improve (Beilock et al., 2004; but see Bijleveld et al., 2011). Still, we believe that both phenomena (the failures to improve that we observed vs. *choking*) are closely related. After all, both reflect suboptimal performance in high-incentive situations.

In our study, EBR modulated a long-lasting effect of incentives, i.e., an effect of incentives that persisted throughout the block. Interestingly, our findings on behavioral task performance (Fig. 3) suggest that the incentive manipulation also had a separate, rather short-lived effect. In particular, people whose monetary payoff was dependent on their performance, exhibited a drop in performance on switch trials, directly after the manipulation. Although participants' performance decreased after learning about the impending loss of money, participants seemed to recover quickly (for a similar pattern of findings, see Lee and Grafton, 2015).

Our findings suggest that block-level (longer-lasting), but not run-level (shorter-lasting), changes in performance were modulated by EBR. In our view, this discrepancy is intriguing, as it overlaps with the idea that dopaminergic effects on cognition may take place on different time scales (Schultz, 2007). Indeed, phasic bursts in dopamine firing may occur very quickly, 60–100 ms after the event (e.g., receiving an unexpected reward), lasting for <200 ms (Schultz, 2007). By contrast, dopamine neurons in the striatum also have been found to fire in response to the anticipation of rewards that are further away in time (e.g., ~10 s), responding to the value of such a reward (Howe et al., 2013). Finally, stimuli that predict reward have been found to increase extra-synaptic dopamine concentrations for a prolonged period of time (in the order of minutes; Datla et al., 2002). It is thus possible that EBR reflects sustained, rather than phasic, dopaminergic processes.

Clearly, though, the exact neuromodulatory mechanisms that are involved in incentive-triggered declines in performance need further clarification. One important reason for this, is that the status of EBR as a correlate of dopamine transmission is currently under debate (Jongkees and Colzato, 2016). On the one hand, recent discoveries using Positron Emission Tomography indicate that EBR is tied to dopamine levels in the striatum, which is particularly dense in D2 receptors (Groman et al., 2014). Indeed, supra-optimal levels of striatal dopamine may harm performance, perhaps especially on switch tasks (see Aarts et al., 2014, and the present study). On the other hand, incentive-triggered performance decrements during cognitive tasks may also be caused by

supra-optimal levels of dopamine transmission in the PFC (Arnsten, 2009). As prefrontal dopamine may play an important role in harming performance in high-stakes situations, the self-report and behavioral correlates of prefrontal dopamine are currently elusive. Establishing and validating these is an important avenue for future research.

We further investigated whether the incentive-triggered effect on performance was moderated by hemispheric asymmetry. We did not find support for this hypothesis. Specifically, none of the results hinted toward the existence of a systematic relationship between greater baseline right hemispheric activity, and incentive-triggered performance decrements—as would be predicted based on attentional theories (Eysenck et al., 2007; Corbetta and Shulman, 2002). This null finding is reminiscent of a recent fMRI-study, which found no correlation between incentive-triggered drops in cognitive performance and activation in the fronto-parietal attention network (Chib et al., 2012). Together with the present study, this research suggests that incentive-triggered performance decrements may not be mediated by changes in activation in brain systems usually implicated in attentional control. Plausibly, the source of incentive-triggered drops in performance does not lie in incentive-triggered changes in activation, but rather in changes in connectivity or processing efficiency (Lee and Grafton, 2015).

Like many prior studies, our study showed that people performed worse on switch trials (vs. repeat trials). An established method to uncover the causes of these switch costs, is to manipulate the length of the response-stimulus interval (RSI). Research that used this method showed that switch costs are more pronounced when RSI is short (Rogers and Monsell, 1995), suggesting that switch costs stem from a lack of time to prepare for the upcoming task (i.e., to reconfigure the task set; Kiesel et al., 2010; Rogers and Monsell, 1995). Yet, when RSI is large, switch costs are usually still observed (Kiesel et al., 2010; Meiran, 1996; Meiran, 2000), suggesting the existence of a separate, residual component of switch costs that is not affected by preparation (Steenbergen et al., 2015). In the present study, there were no intervals between participants' responses and the subsequent stimuli. So, the switch costs we observed were likely (at least in part) due to lack of time for preparation. However, as we did not manipulate RSI, we cannot draw conclusions about a potential relation between EBR, incentives, and residual vs. preparatory switch costs. Future research may add an RSI manipulation to shed light on this issue.

4.1. Conclusion

The present study adds to emerging evidence concerning the neural underpinnings of incentive-triggered underperformance (Aarts et al., 2014; Chib et al., 2012; Chib et al., 2014; Lee and Grafton, 2015). In the current study, we show that people's failures to improve their performance (when incentives can be earned), are moderated by individual differences in eye blink rate. So, when incentives cause poor performance, this may well be the work of the ascending dopamine pathways.

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Appendix A. Supplementary information

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.ijpsycho.2017.01.009.

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