NeuroImage 103 (2014) 241–248

Contents lists available at ScienceDirect

NeuroImage

journal homepage: www.elsevier.com/locate/ynimg

Response inhibition and its relation to multidimensional impulsivity

Tilmann Wilbertz [a,*], Lorenz Deserno [a,b], Annette Horstmann [a,c,d], Jane Neumann [a,c,d], Arno Villringer [a,c,d,e,f], Hans-Jochen Heinze [a,g,h], Carsten N. Boehler [i], Florian Schlagenhauf [a,b]

[a] Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany
[b] Department of Psychiatry and Psychotherapy, Charité-Universitätsmedizin, Berlin,Germany
[c] IFB Adiposity Diseases, Leipzig University Medical Center, Germany
[d] SFB 1052 Obesity Mechanisms, Leipzig University, Germany
[e] Clinic for Cognitive Neurology, University Hospital Leipzig, Leipzig, Germany
[f] Berlin School of Mind & Brain and Mind & Brain Institute, Humboldt-University, Berlin, Germany
[g] Leibniz Institute for Neurobiology, Otto-von-Guericke University, Magdeburg, Germany
[h] Department of Neurology, Otto-von-Guericke University, Magdeburg, Germany
[i] Department of Experimental Psychology, Ghent University, Belgium

ARTICLE INFO

Article history:
Accepted 9 September 2014
Available online 18 September 2014

Keywords:
Stop signal task
Impulsivity
Urgency
IFG
Reward
fMRI

ABSTRACT

Impulsivity is a multidimensional construct that has been suggested as a vulnerability factor for several psychiatric disorders, especially addiction disorders. Poor response inhibition may constitute one facet of impulsivity. Trait impulsivity can be assessed by self-report questionnaires such as the widely used Barratt Impulsiveness Scale (BIS-11). However, regarding the multidimensionality of impulsivity different concepts have been proposed, in particular the UPPS self-report questionnaire ('Urgency', 'Lack of Premeditation', 'Lack of Perseverance', 'Sensation Seeking') that is based on a factor analytic approach. The question as to which aspects of trait impulsivity map on individual differences of the behavioral and neural correlates of response inhibition so far remains unclear.

In the present study, we investigated 52 healthy individuals that scored either very high or low on the BIS-11 and underwent a reward-modulated Stop-signal task during fMRI. Neither behavioral nor neural differences were observed with respect to high- and low-BIS groups. In contrast, UPPS subdomain *Urgency* best explained inter-individual variability in SSRT scores and was further negatively correlated to right IFG/aI activation in 'Stop > Go' trials — a key region for response inhibition. Successful response inhibition in rewarded compared to nonrewarded stop trials yielded ventral striatal (VS) activation which might represent a feedback signal. Interestingly, only participants with low *Urgency* scores were able to use this VS feedback signal for better response inhibition.

Our findings indicate that the relationship of impulsivity and response inhibition has to be treated carefully. We propose *Urgency* as an important subdomain that might be linked to response inhibition as well as to the use of reward-based neural signals. Based on the present results, further studies examining the influence of impulsivity on psychiatric disorders should take into account *Urgency* as an important modulator of behavioral adaptation.

Introduction

Impulsivity is a multidimensional construct and has been suggested as a potential endophenotype for several psychiatric disorders such as substance use disorder (Robbins et al., 2012). Poor response inhibition has been suggested as one facet of impulsivity. However, there are conflicting findings as to which aspects of trait impulsivity can directly be linked to response inhibition (Dick et al., 2011).

Response inhibition — the ability to withhold an inappropriate response — is one of the most important executive functions and is closely related to concepts of self-regulation and goal-directed behavior (Bari and Robbins, 2013). Response inhibition can be measured using a Stop-signal task, which requires individuals to rapidly suppress an ongoing, well-established response whenever a certain cue is suddenly presented. According to the horse-race model (Logan, 1984) the Stop-signal reaction time (SSRT) is an estimate of the time that an individual needs to withhold an ongoing response. Response inhibition as operationalized with such a Stop-signal Task is moderated by a network of cortical and sub-cortical regions, which suppresses stimulus-evoked behavior. Within this network the right inferior frontal gyrus (IFG) has

* Corresponding author at: Max-Planck-Institute for Human Cognitive and Brain Sciences, Stephanstraße 1a 04103 Leipzig. Fax: +49 341 2499.
E-mail address: wilbertz@cbs.mpg.de (T. Wilbertz).

http://dx.doi.org/10.1016/j.neuroimage.2014.09.021
1053-8119/

been proposed as crucial structure for response inhibition (Aron et al., 2003).

Trait impulsivity can be assessed with self-report questionnaires like the Barratt Impulsiveness Scale (BIS-11, Patton et al., 1995), which is one of the most commonly used psychometric instruments.

There is good evidence for both higher trait impulsivity and impaired response inhibition in various neuropsychiatric disorders (Verdejo-García et al., 2008). On the other hand, in healthy controls there are conflicting results regarding the relationship between self-report impulsivity measures and experimentally operationalized response inhibition (Lijffijt et al., 2004; Logan et al., 1997). On the behavioral level, in a large sample of 504 healthy individuals prepotent response inhibition (construct derived from antisaccade, Stroop, Stop-signal, and Go/No-Go tasks) was only explained to a limited extent (only 12% of variance) by psychometrically assessed impulsivity (measured with the BIS-11, Aichert et al., 2012). On the neural level, previous imaging studies revealed heterogeneous findings regarding the association between trait impulsivity and neural activation during response inhibition. Farr et al. (2012) investigated 92 healthy subjects with a Stop-signal task and found that activation for 'stop versus go' trials in the mPFC and right anterior dorsal insula correlated negatively with trait impulsivity (especially the motor subscore of the BIS-11). In a Go/No-Go task, a negative correlation with the BIS-11 and the superior frontal gyrus was observed (Horn et al., 2003). Another study found that only the middle frontal gyrus correlated negatively with the BIS-11 motor subscale in a Go/No-Go task (Asahi et al., 2004). The heterogeneity of previous results with respect to brain regions associated with trait impulsivity during inhibition tasks may be due to the use of rather coarse self-report measures of impulsivity which do not adequately account for the different subdimensions of impulsivity.

Regarding trait impulsivity, different concepts have been proposed and impulsivity can be split up into different components. Using a factor-analytic approach, four different subdomains of impulsivity as a personality trait have been identified (Whiteside and Lynam, 2001): 1) *Urgency*: inability to inhibit action impulses especially in a negative motivational state despite long-term consequences; 2) *Premeditation*: inability to anticipate the consequences of one's actions; 3) *Perseverance*: inability to continue with boring or difficult tasks and (4) *Sensation seeking*: tendency to seek novel situations.

The subdomain *Urgency* is supposed to be related to behavioral impulsivity measures as inhibition of prepotent responses (for a meta-analysis see Cyders and Coskunpinar, 2011). This points towards the so far untested hypothesis that *Urgency* may explain individual variability in the behavioral and neural correlates of a Stop-signal task.

Trait impulsivity has been linked to alterations during reward processing: especially high impulsive individuals have been proposed to be more sensitive to immediate rewards and thus show stronger delay discounting (Ainslie, 1975; Hoogman et al., 2011; Hariri et al., 2006). Activation during reward processing has been shown to be related to trait impulsivity measures (Forbes et al., 2009). In a recent review, Plichta and Scheres (2014) postulate a positive relationship of trait impulsivity and ventral striatum(VS) BOLD signal during reward processing and anticipation in healthy participants.

The influence of reward effects on response inhibition has received limited attention so far. One behavioral study found a beneficial effect of reward on response inhibition in healthy students (Boehler et al., 2012, see also Scheres et al., 2001, and Sinopoli et al., 2011). A subsequent fMRI study demonstrated elevated activation of the so-called 'inhibition network' when comparing reward- to nonreward-associated trials (Boehler et al., 2014). Based on the finding of altered reward processing in impulsive individuals (Plichta and Scheres, 2014) we asked how the relationship between trait impulsivity and response inhibition is modulated by reward. Here we investigated the influence of self-report trait impulsivity measures on response inhibition using a reward-modulated Stop-signal task (Boehler et al., 2012). A sample of high compared to low impulsive healthy individuals was preselected from the extreme ends of self-report trait impulsivity (for a similar approach studying trait aggression compare Pawliczek et al., 2013).

Given the mixed findings, we probed if high impulsive individuals show poorer response inhibition (longer SSRT) associated with lower activation of the cortical inhibition network especially the right inferior frontal gyrus (IFG). Further, we probed the relationship of multiple subdimensions of impulsivity and the behavioral and neural correlates of response inhibition which we expected to find in the ventral striatum and the prefrontal cortex, especially the anterior cingulate cortex (Boehler et al., 2014; Scheres et al., 2007; Christakou et al., 2011). In addition, we tested how different dimensions of impulsivity interact with reward and its influence on response inhibition.

Methods

Participants

From a total sample of 452 participants who completed the Barratt Impulsiveness Scale-11 (Patton et al., 1995) we selected 52 right-handed high or low scoring individuals from the upper and lower end of the range (for a distribution of the BIS-11 scores in our sample, see supplement Figure S1B). The mean BIS-score for each group fulfilled criteria for classifying subjects as high or low impulsive (Stanford et al., 2009). Subjects were matched for age and gender and screened for psychiatric disorders using the SCID-IV interview. Based on this screening, one participant was excluded because of a recent episode of major depressive disorder. A further two subjects were excluded due to malfunctioning of the buttons during fMRI scanning, leading to a total sample of 49 participants.

All participants were paid on an hourly basis and gave written informed consent to participate in the study. The study was approved by the local ethics committee. In order to compare the multiple dimensions of impulsivity, all participants additionally filled out the German version of the UPPS self-report questionnaire containing the subdimensions 'urgency', 'lack of perseverance', 'lack of premeditation' and 'sensation seeking' (Whiteside and Lynam, 2001), the NEO-FFI-30 (Körner et al., 2008), and the Sensation Seeking Scale (SSS, Zuckerman et al., 1978). To assess verbal intelligence, working memory and cognitive speed, participants underwent neuropsychological testing including a German version of the vocabulary test (Schmidt and Metzler, 1992) and the Digit Span(taken from a German version of the WAIS-III, Von Aster, Neubauer & Horn, 2006).

Paradigm

Participants performed a modified staircase-adapted Stop-signal task (Logan, 1994, see Fig. 1). In this task, subjects are instructed to respond by button press as fast as possible to a Go-signal. In a minority (33%) of trials the Go-signal is subsequently replaced by a Stop-signal prompting the subjects to withhold their response. As Go-trials form the majority of trials and the Stop-signal emerges suddenly after the Go-signal, Stop-trials force participants to cancel an already initiated prepotent response.

In order to achieve a stopping rate of approximately 50%, we introduced a staircase procedure that varied the Stop-signal-delay (SSD) after each Stop-trial. After an unsuccessful Stop-trial (US) 34 ms were subtracted from the individual SSD making it easier for the participants to inhibit their response. Accordingly, a successful Stop-trial (SS) led to an extended SSD by 34 ms, thus making it more difficult for the participants to withhold their response. Participants yielded a minimum and maximum SSD of 67 ms and 533 ms respectively. This procedure results in around 50% successful Stop-trials and enabled the computation of the Stop-signal reaction time (SSRT) as described in the following section. Go-trials were presented as traffic light symbols pointing either to the right or to the left. Participants were instructed to press the button corresponding to the direction of the symbol using their thumbs. In each

Fig. 1. Reward modulated Stop-signal task. In Go-trials participants had to indicate as fast as possible if the symbol pointed to the right or to the left side by button press. In Stop-trials the Go-signal was replaced by a Stop-signal which was either pink indicating that successful response cancellation will result in monetary gain (rewarded Stop-trials) or blue indicating no monetary consequences (nonrewarded Stop-trials). Allocation of colour to Stop-trial type was balanced over participants. Please note that no explicit feedback regarding task performance nor reward was provided.

trial, total stimuli presentation was 600 ms and was followed by an exponentially-distributed, jittered inter-trial interval with a mean of 3 s ranging from 1.5 s to 9 s.

In contrast to a classical Stop-signal design, we used two kinds of Stop-signals that differed in color (blue and pink). One color indicated a potential reward if stopping was successful and the other color was not associated with a potential reward (Boehler et al., 2012). Participants could gain 20 points for each successfully inhibited rewarded Stop-trial and points were changed into money at the end of the experiment yielding a monetary payout ranging from 4 to 5€. No feedback with regard to monetary payout was given to the participants between the runs. All participants completed a training session of 34 Go-trials and 20 Stop-trials outside the scanner. After training, participants were instructed that either the blue or the pink Stop-signal was associated with potential reward and this was counterbalanced across all participants.

The fMRI session was separated into five runs, each consisting of 64 Go-trials, 16 rewarded and 16 unrewarded Stop-trials. The task was identical to the training session with the exception that the staircase was now adapted separately for rewarded and unrewarded Stop-trials. At the beginning of the first run the start SSD was taken from the end of the training session, whereas starting SSDs for the following runs were taken from the end of the previous runs. In order to prevent slowing in Go-trials, participants were instructed between the runs to maintain a fast speed on Go-trials.

Behavioral data

The inhibition process can be quantified by computing the 'Stop Signal Reaction Time' (SSRT) using the so-called 'integration approach'. In this approach, reaction times on Go-trials are rank-ordered individually for each participant and run. The mean SSD is then subtracted from the n*th* percentile of the Go reaction time corresponding to the percentage of unsuccessful Stop-trials in the particular run, yielding the SSRT for this run. For all further analyses we used the mean SSRT across all five runs. This method aims to minimize false skewing of the SSRT that may result from continuous slowing on Go-trials (Verbruggen et al., 2013).

Behavioral data were analyzed using SPSS18. The SSRT was analyzed with a 2×2 ANOVA comprising the within-subject factor 'reward' and the between-subject factor 'group' (high vs. low BIS-11).

To characterize the influence of the multiple personality traits on response inhibition performance we performed a multiple regression analysis with SSRT as dependent variable and the four subscales of the UPPS, the five factors of the NEO-FFI-30, the SSS and the BIS-11 as independent variables.

fMRI data

Data acquisition

Imaging was performed on a 3 T Siemens Tim Trio Scanner using an echo planar imaging (EPI) sequence (TR = 2000 ms, TE = 22, flip = 90°, matrix = 64 × 64, voxel size = 3 × 3 × 3 mm, slices = 36, gap of 0.5 mm) which was tilted ~25° to the AC-PC line to reduce artifacts. Before functional scanning, field maps were collected to account for distortion (phase image: TR = 488 ms, TE = 7.38 ms, flip = 60°, matrix: 64 × 64, voxel size = 3 × 3 × 3 mm, slices = 36; magnitude image: TR = 488 ms, TE = 4.92 ms, flip = 60°, matrix: 64 × 64, voxel size = 3 × 3 × 3 mm, slices = 36). In addition, a structural scan was acquired (TR = 1300 ms, TE = 3.46 ms, flip = 10°, matrix = 240 × 256, voxel size: 1 × 1 × 1 mm, slices = 170).

Data analysis

Functional MRI data were analyzed using SPM8 (http://www.fil.ion.ucl.ac.uk/spm/). For data preprocessing, images were corrected for differences in slice time acquisition, for subject motion by realigning them to the mean volume, and simultaneously for geometric distortion based on the field map and the interaction of distortion with motion (SPM8 "realign and unwarp"). Individual anatomical T1 images were co-registered to the mean EPI and then spatially normalized to the Montreal Neurological Institute (MNI)-space using the unified segmentation approach (Ashburner and Friston, 2005). Normalization parameters were derived from segmentation of each participant's structural scan and were subsequently applied to all functional images. Images were resliced with an isotropic voxel size of 2 mm. Finally, spatial smoothing with a Gaussian smoothing kernel with 6 mm full-width at half maximum (FWHM) was performed.

For statistical analysis, we performed an event-related analysis in the context of the general linear model using SPM's two-level approach. On the single subject level, five trial types were defined and their onsets were included after convolution with the canonical hemodynamic response function: 'successful rewarded Stop-trial', 'unsuccessful rewarded Stop-trial', 'successful unrewarded Stop-trial', 'unsuccessful unrewarded Stop-trial' and 'Go-trial'. Realignment parameters were entered as additional regressors of no interest into the model in order to explain movement-related variance.

On the group level, individual contrast images for the contrast 'Stop > Go' were compared between the groups using a two-sample t-test. A full-factorial ANOVA design was used to assess the factors 'reward', 'success' and their interactions. All results are reported at $p < .05$ family wise error (FWE)-corrected for the whole brain.

Correlations between measures of self-reported impulsivity and neural activation during response inhibition and reward processing were tested in separate random effects models using the appropriate individual contrast images and including the respective measures as covariates. Based on the observation that the UPPS subscore *Urgency* best explained individual variability in inhibition performance (SSRT), we entered the UPPS subscore *Urgency* as a covariate into a one-sample t-test with the contrast 'Stop > Go' to examine the association of *Urgency* and inhibition-related brain activation. Results are reported at $p < .05$ FWE-corrected for a region of interest (ROI) of the right IFG/aI. Based on the 'Stop > Go' contrast the cluster of the right IFG/aI (voxelwise map thresholded at $p < .05$ FWE) was used to create this ROI.

To test if trait impulsivity moderates the relationship between task performance and neural correlates of success and reward processing, we conducted a moderation analysis within SPSS using the MODPROBE toolbox (Hayes and Matthes, 2009). Notably, success during a stop trial is very apparent for the participant even without receiving explicit feedback. The activated voxels of the contrast 'reward × success' (taken from peak voxel of a one-sample t-test) were integrated as a dependent

variable into a multiple regression and the variables 'SSRT', '*Urgency*' and the interaction term 'SSRT × *Urgency*' were entered as independent variables.

Results

Behavioral results

Across all participants, correct responses in Go-trials were more than 96% (Table S1). Participants successfully inhibited around 50% of the Stop-trials indicating that the staircase adaptation worked (Table S1). Although on a descriptive level the Stop-signal reaction time (SSRT) in high impulsive individuals was slightly longer compared to low impulsive individuals (high: 245.66 ± 20.67 ms; low: 238.34 ± 25.67 ms), this difference did not reach statistical significance (two-sample t-test: $t = -1.1$, $p = .28$). Using a group by reward repeated-measures ANOVA, no main effect of group ($F(1,47) = 1.199$; $p = .28$) or reward ($F(1,47) = 1.392$; $p = .24$) nor a group by reward interaction was significant ($F(1,47) = 1.096$; $p = .3$). However, behavioral data revealed a significant reward × success interaction when analyzing the Go reaction time in a sequential fashion (see supplement, Figure S4).

Regarding the multiple dimensions of impulsivity, we used the BIS-11, the UPPS sub-dimensions, the five factors of the NEO-FFI, and the SSS as independent variables in a multiple regression analysis. The SSRT was entered as the dependent variable. Only the UPPS subscale *Urgency* was significantly positively associated with the SSRT ($t = 2.092$; $p = .044$, Fig. 2 and supplement Figure S2). Although we extracted BIS-11 scores from the upper and lower end of a huge sample, the distribution of *Urgency* scores did not differ from a fitted normal distribution (Fig S1A, using a One-Sample-Kolmogorov-Smirnov Test: Test Statistics = .074, asysmptotic significance = .200)

fMRI

Neural network of response inhibition for the contrast 'Stop > Go' and impulsivity

Analyzing all subjects, a contrast of stop compared to Go-trials ('Stop > Go') showed activation of the well characterized 'inhibition network' (for a meta-analysis, see Swick et al., 2011) comprising bilateral inferior frontal gyrus/insula, dmPFC/pre SMA, bilateral middle and superior frontal gyrus, bilateral inferior parietal lobule, posterior cingulate, and subcortical areas (Thalamus) (Fig. 3A, for a full list see supplement Table S2).

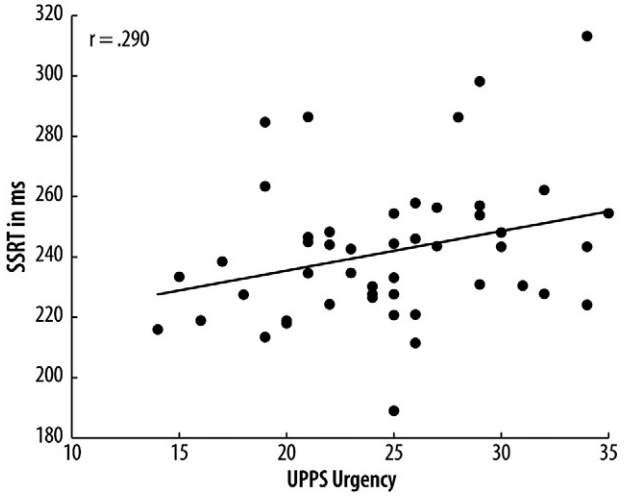

Fig. 2. Stop-signal reaction time (SSRT) and its relation to the UPPS subdomain *Urgency*. SSRT showed a positive correlation with the UPPS subdomain *Urgency* ($r = .29$; $p = .046$), indicating that individuals with higher UPPS *Urgency* scores showed reduced response inhibition abilities.

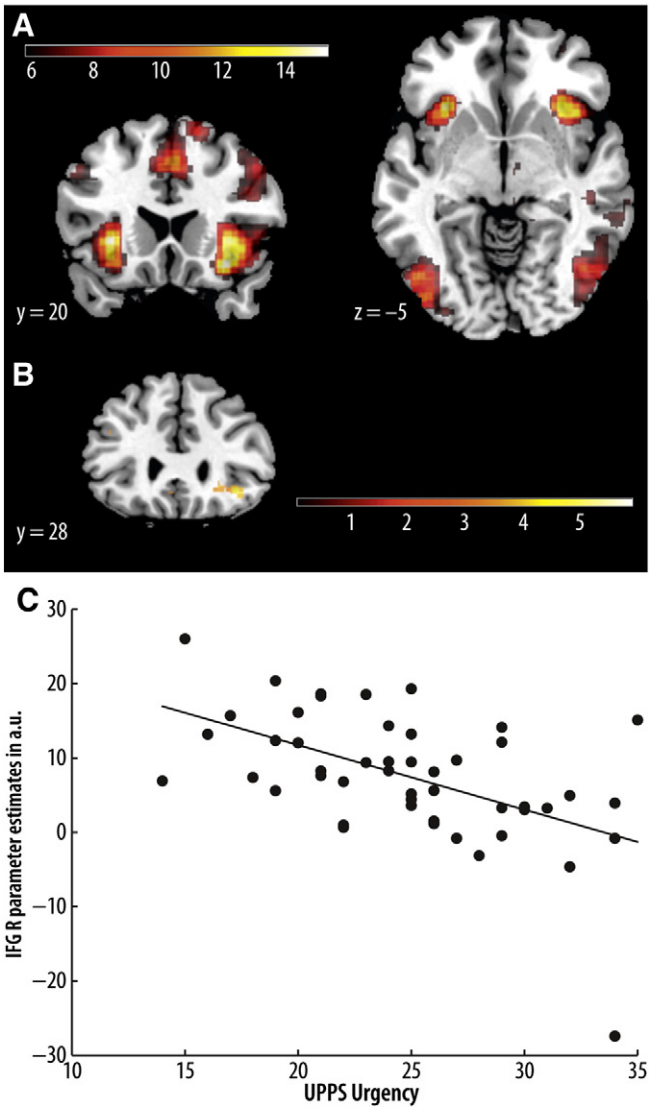

Fig. 3. Neural activation during response inhibition and its relation to impulsivity. A) The contrast 'Stop > Go' revealed a network including bilateral inferior frontal gyrus/insula and dorsal anterior cingulate (displayed at $p_{FWE\text{-corrected for the whole brain}} < .05$). B) BOLD signal activation in the right IFG/aI for the contrast 'Stop > Go' with *Urgency* scores as a covariate C) There was a negative correlation between the individual UPPS *Urgency* score and the BOLD signal in the right inferior frontal gyrus (IFG R) for the contrast 'Stop > Go' ($p_{FWE\text{-smallvolume-corrected for ifg main effect of 'Stop – Go'}} < .05$; parameter estimates extracted from peak-voxel ($x = 36$, $y = 28$, $z = -8$)).

Due to the finding that the UPPS subscale *Urgency* explained individual differences in SSRT, *Urgency* was included as a covariate in an additional SPM analysis for the contrast 'Stop > Go' including all participants. The right IFG showed a negative correlation with the UPPS *Urgency* ($t = 4.26$; $x = 36$, $y = 28$, $z = -8$; $p_{FWE\text{-smallvolume-corrected for right ifg}} = .014$), indicating that individuals with a higher *Urgency* score showed weaker BOLD response during response inhibition (Fig. 3B, supplement Figure S3). This negative correlation with *Urgency* was observed for nonrewarded ($t = 4.1$; $x = 38$, $y = 26$, $z = -10$; $p_{uncorr} < .001$) and for rewarded Stop-trials compared to Go-trials ($t = 3.05$; $x = 36$, $y = 28$, $z = -8$; $p_{uncorr} = .002$). Regarding the observed correlation of Urgency and SSRT, we intergrated SSRT as an additional covariate into the model. Even when controlling for SSRT, the negative correlation of right IFG and *Urgency* still remained significant ($t = 4.17$; $x = 38$, $y = 26$, $z = -10$; $p_{FWE\text{-smallvolume-corrected for right ifg}} = .019$).

Regarding the BIS-11 groups, no differences were found for the contrast 'Stop > Go'.

Neural effects of success and reward during response inhibition

In order to assess effects of success and reward on the activation during response inhibition, SS (successful Stop-trials) and US (unsuccessful Stop-trials) were modeled separately for rewarded and unrewarded trials using a reward × success ANOVA design. A *main effect of reward* was found in the right insula/IFG, anterior cingulate cortex and putamen ($p_{FWE\text{-corrected for the whole brain}} < .05$, see supplement Table S3) indicating stronger BOLD responses for rewarded Stop-trials compared to nonrewarded Stop-trials. Dorsal anterior cingulate cortex(dACC), anterior cingulate cortex(ACC), right and left anterior insula were identified as regions to be activated specifically for the reward condition which replicates previous findings from Boehler et al. (2014)(for a detailed analysis, see supplementary material). A main effect of success due to stronger activations for SS compared to US was present in bilateral middle frontal gyrus, anterior cingulate, medial frontal gyrus, right cingulate gyrus, right caudate head, bilateral caudate body and left precuneus ($p_{FWE\text{-corrected for the whole brain}} < .05$, for a full list see supplement Table S3). A reward by success interaction was found in the bilateral ventral striatum ($p_{FWE\ whole\ brain\ corrected} < .05$, Fig. 4A, Table S3). This interaction was due to a success effect only during rewarded but not during nonrewarded Stop-trials with a stronger BOLD signal in rewarded SS compared to rewarded US. Nonrewarded SS did not differ from nonrewarded US (Fig. 4B).

There were no significant correlations between *Urgency* or SSRT with any of these three contrasts (FWE-whole-brain-corrected or FWE-corrected for the voxels activated by the respective contrast).

For further investigation of this 'reward × success' interaction, we extracted parameter estimates from the peak voxel and computed 6 paired t-tests as a Post hoc analysis. All Post hoc tests showed significant differences (all $t \geq 2.432$, all $p \leq .02$, see Supplement Table S5), except for 'NonReward Success > Reward Unsuccess' and 'NonReward Unsuccess > NonReward Success' (all $t \leq .1602$, all $p \geq .116$, Table S5).

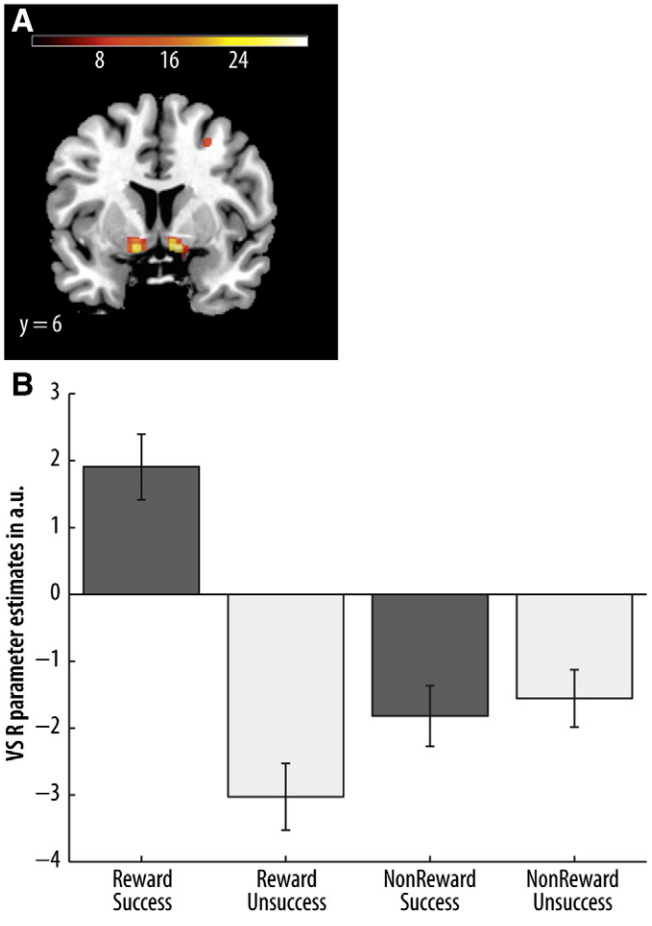

Fig. 4. Ventral striatum activated during successful compared to unsuccessful rewarded Stop-trials. A) Success by reward interaction in the ventral striatum (for display purposes $p_{uncorr} < .001$). B) Plots of the parameter estimates in the right ventral striatum peak (x = 10; y = 6; z = -12) showing stronger BOLD signal for rewarded successful Stop-trials.

Urgency moderates the relation of striatal feedback signal and response inhibition

The 'reward × success' interaction in the ventral striatum may represent a self-generated feedback signal. Such a self-generated feedback signal may act as a motivationally enhancing signal, which might subserve inidividual improvements in response inhibition. This relationship might potentially be disrupted in individuals with high levels of *Urgency*. Therefore, we investigated if *Urgency* moderates the relationship between this VS feedback signal and response inhibition measured with the SSRT. Parameter estimates of left and right ventral striatum (at $x = -12$; $y = 4$; $z = -12$ and $x = 12$; $y = 4$; $z = -12$) taken from peak-voxel activation of a one-sample t-test testing the contrast 'reward × success') were each entered into a separate regression model with 'SSRT', 'Urgency' and the interaction 'Urgency × SSRT' as independent variables. Indeed, the interaction of 'Urgency × SSRT' contributed significantly to the feedback signal for both left (change in $r^2 = .09$, beta = .312, $t = 2.157$, $p = .036$) and right ventral striatum (change in $r^2 = .089$, beta = .307, $t = 2.101$, $p = .041$) indicating that *Urgency* moderates the relation between the feedback signal and response inhibition. The factors *Urgency* (left VS: beta = .180, $t = .1227$, $p = .226$; right VS: beta = .101, $t = .684$, $p = .498$) and SSRT (left VS: beta = −.248, $t = -1.649$, $p = .106$; right VS: beta = −.232, $t = -1.520$, $p = .136$) alone did not contribute significantly to the model. In order to further explore and visualize this moderation effect, we aimed to compare individuals with high *Urgency* scores to participants with rather low and medium *Urgency* scores. Therefore, we split up the sample at the 66th percentile of the *Urgency* subdomain and found that only individuals with low or medium ($n = 32$) but not with high *Urgency* ($n = 16$) scores showed better task performance with higher VS feedback signal (Fig. 5). Notably, the significant moderation effect of the interaction term 'Urgency × SSRT' was computed dimensionally across the whole sample. The further split-up according to the *Urgency* scores was done to visualize how *Urgency* moderates the relation of SSRT and the striatal feedback signal.

Discussion

Our results show that inter-individual differences in response inhibition were best explained by *Urgency*—a subdomain of trait impulsivity. *Urgency* correlated negatively with BOLD response during reponse inhibition in the right IFG/aI, which in lesion (Aron et al., 2003) and fMRI studies have been shown to be crucial for successful response inhibition (for a review see Aron, 2007). In our task, the ventral striatal BOLD signal potentially represents a feedback signal for successful compared to unsuccessful rewarded Stop-trials. The association between this ventral striatal feedback signal and behavioral response inhibition was moderated by *Urgency*. This indicated that only participants with lower *Urgency* scores showed better response inhibition with stronger striatal error signal.

Urgency and response inhibition

Healthy high and low impulsive individuals with a high level of cognitive abilities were carefully selected from a large sample according to the widely used BIS-11. Between these two extreme BIS groups SSRT was not significantly different. While no significant difference in response inhibition (mean SSRT) was seen in these extreme groups for an overall impulsivity trait measure, the subcomponent *Urgency* did explain individual differences in response inhibition abilities. This is in line with the intended construct validity of the UPPS, where failure in

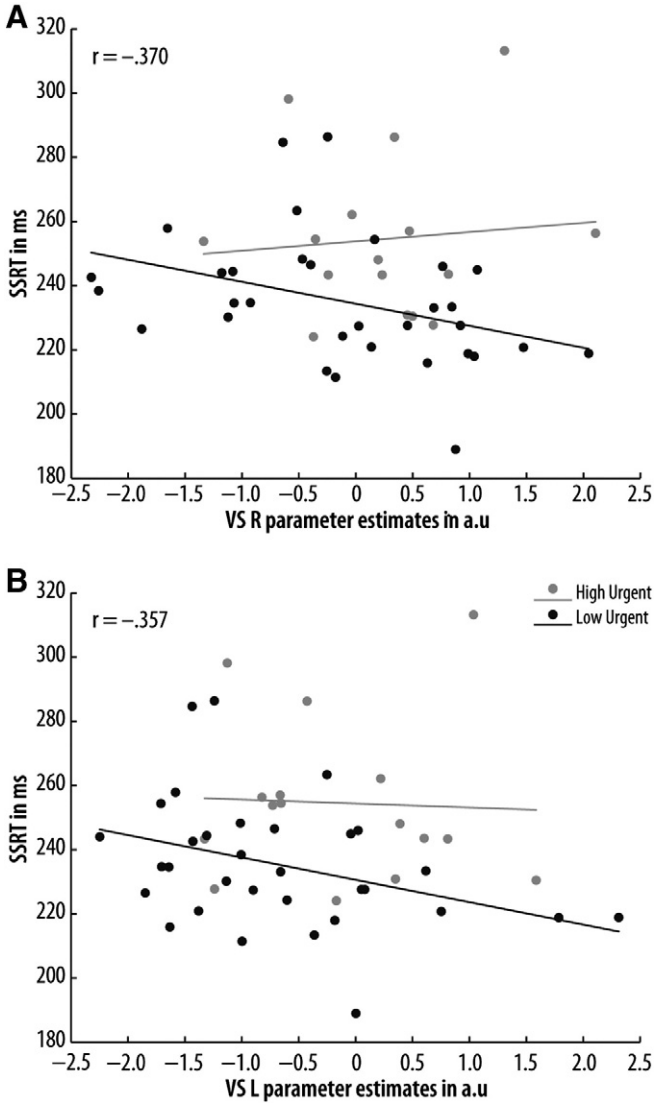

Fig. 5. Ventral striatal feedback signal and task performance in low and high urgent participants. A) Right Ventral striatum activation (VS R) correlated negatively with SSRT only in the low *Urgency* group (shown in black, r = −.37, p = .037), but not the high *Urgency* group (grey, p = .737). B) Activation of the left ventral striatum (VS L) and SSRT correlated negatively in the low *Urgency* group (black, r = −.357, p = .045), but not in the high *Urgency* group (grey, p = .875).

response inhibition should relate to the *Urgency* dimension of impulsivity (Whiteside and Lynam, 2001; Basar et al., 2010). With regard to Barratt's model of impulsivity (Patton et al., 1995) this would correspond to the motor impulsiveness although the BIS-11 subscales showed poor psychometric properties for the German version (Preuss et al., 2008). Taken together, we did not find evidence that overall trait impulsivity in high functioning healthy controls is associated with poor inhibitory motor control which is in line with previous findings (Aichert et al., 2012). Our found association of UPPS-subscore with response inhibition capacities suggests that only a certain subdomain of trait impulsivity is indeed associated with the applied behavioral measure of response inhibition.

In contrast to previous findings (Boehler et al., 2012) we did not find an overall effect of reward on the SSRT. This may be due to differences in task design and sample characteristics. Here we used a jitter of the ITI that might have disturbed subjects' automatic anticipation of the following Go-signal. Responses were given with the thumbs of the left and the right hand, which represents an additional challenge for a rapid response rather than responses of index and middle finger. Moreover, our sample involves two extreme groups, i.e., the high and low end of the BIS-11 but no intermediate group and was further different to previous samples with regard to age and gender distribution. However, besides this absent reward effect on response inhibition, sequential analysis revealed that our reward-modulation did have a behavioral effect as indicated by the shorter reaction times in Go-trials after successfully rewarded Stop-trials. Furthermore, on the neural level strong reward effects were observed.

Inhibitory IFG activation and response inhibition

Response inhibition activated a well-defined network for the contrast 'Stop > Go' comprising the inferior frontal gyrus/insula, dmPFC/pre SMA, bilateral middle and bilateral superior frontal gyrus, inferior parietal lobule, posterior cingulate, and subcortical areas (Thalamus) (Chambers et al., 2009). In our study, the IFG/aI that is crucial for response inhibition was negatively correlated with *Urgency*. Farr et al. (2012) reported a relation between anterior dorsal insula activation during the Stop-signal Task and motor BIS-11 score. They interpreted their results as reflecting higher salience attribution in high impulsive individuals. Concepts of *Urgency* and motor BIS-11 score may show a certain overlap regarding their relation to response inhibition. Here, we found that *Urgency* negatively correlated with IFG activation during rewarded as well as unrewarded stop compared to Go-trials which might indicate that this relationship is rather related to response inhibition than salience processing.

Urgency and reward-modulated activation during response inhibition

The reward-modulated Stop-signal task revealed activation during rewarded compared to nonrewarded Stop-trials in areas commonly involved in reward processing including putamen and ventromedial PFC areas (Knutson et al., 2003; Schultz, 1998). Successful compared to unsuccessful Stop-trials activated a distributed fronto-striatal network including insula, ACC and caudate. A recent fMRI study found *Urgency* to explain inter-individual variance of BOLD signal in left and right vmPFC in response to alcohol odors (Cyders et al., 2013). However, in our study activation during reward or success were not related to *Urgency,* which indicated that neural processes of reward and success effects were not explained by trait impulsivity in our sample of high functioning, healthy controls.

Activation in the ventral striatum was mainly driven by successful compared to unsuccessful rewarded Stop-trials. However rewarded unsuccessful trials showed a relative decrease in comparison to nonrewarded unsuccessful trials which might represent a negative 'dip' that is similar to a negative prediction error (Hollerman and Schultz, 1998). This ventral striatal activation showed an association with behavioral response inhibition depending on individual *Urgency* scores as indicated by the moderation analysis: Only subjects with low or medium *Urgency* scores displayed an association between VS BOLD signal and SSRT. The ventral striatum, as a main dopaminergic target region, has been implicated in impulsive personality traits before, although the effects varied with regard to fMRI tasks and impulsivity measures used (Plichta and Scheres, 2014; Forbes et al., 2009). A positive relationship between VS activation during reward processing has been postulated in healthy controls (Plichta and Scheres, 2014) while negative association was seen in patient groups associated with impulsivity (Beck et al., 2009; Ströhle et al., 2008; Scheres et al., 2007). These mixed findings might be related to differences in dopaminergic bioavailability (Hahn et al., 2011). On the other hand, a permanent overstimulation of the reward circuits might result in a downregulation of the reward system (in case of a conversion to a psychiatric disease, as e.g. addiction) and consequently in a negative correlation of VS response and impulsivity. In this case impulsivity has been suggested to more likely represent symptom severity (Plichta and Scheres, 2014). Regarding dopamine neurotransmission lower levels of presynaptic

dopaminergic regulation in the midbrain were demonstrated to mediate the positive relationship between pharmacologically-induced dopamine release in the striatum and trait impulsivity (Buckholtz et al., 2010). Also D2/D3 receptor availability in putamen and caudate correlated negatively with SSRT in a sample of healthy individuals (Ghahremani et al., 2012). In line with this, lower levels of presynaptic dopamine synthesis capacity were demonstrated to be negatively associated with the coding of reward prediction errors in ventral striatal activity, a potential surrogate of reward-induced phasic dopamine release (Schlagenhauf et al., 2013). This association appears to be disrupted in alcohol-dependent patients (Deserno et al., under review), who are also characterized by higher levels of trait impulsivity (Beck et al., 2009). Ventral striatal activation during the anticipation of a potential reward has been associated with impulsivity measured with the BIS-11 (Beck et al., 2009). Animal research strongly supports this idea of a potential regulatory mechanism within the dopaminergic system as an important contributor to impulsive and addictive behavior (Bello et al., 2011). Interestingly, impulsive ('sign-tracking') rats express phasic dopamine reward prediction errors in Nucleus accumbens, whereas low impulsive ('goal-tracking') rats do not (Flagel et al., 2011). In the present study, we did not find a direct relationship of trait impulsivity and the observed ventral striatal activation, which may potentially reflect a feedback signal comparable to a prediction error. Instead, we show for the first time that this ventral striatal feedback signal and behavioral response inhibition (SSRT) only relate depending on a certain feature of trait impulsivity, namely *Urgency*. Our finding indicates that only subjects with low *Urgency* benefit from the ventral striatal feedback signal resulting in improved response inhibition and task performance. Future studies may directly test the question as to whether high *Urgency* is indeed associated with lower levels of presynaptic dopamine.

Limitations

Our study design consisted of two extreme groups selected from the upper and lower end of a wide range of BIS-11 scores. The low impulsive group may therefore differ from a standard control group. In contrast, very low impulsive individuals may even show over-regulated behavior. Further studies should not only investigate two extreme groups but also a third intermediate group. This may help to provide an even more comprehensive dimensional analysis of trait impulsivity. However, *Urgency* scores were distributed normally in the present sample of healthy participants.

Further, the BIS-11 subscores in the German-version (Preuss et al., 2008) show poor psychometric properties. Thus, we did not analyze these subscores separately. Consequently, we could not investigate if any BIS-11 subscore might also sufficiently explain individual variability in response inhibition performance and relate to the reward-related VS feedback signal in the same way that the *Urgency* domain did.

Conclusion

Our results underline that the relationship of trait impulsivity and response inhibition has to be treated carefully. In the present study not overall trait impulsivity but the subdomain *Urgency* explained behavioral response inhibition performance. This finding was supported by a correlation of the *Urgency* subdomain and right IFG activation during response inhibition. *Urgency* seems to be related not only to response inhibition but also moderates the use of a potential feedback signal. Future studies should take the multiple dimensions of trait impulsivity and the differences between trait impulsivity and behavioral measures into account and not solely rely on one construct. Regarding the probable impact of impulsivity on psychiatric disorders such as addiction, further studies should carefully distinguish whether these findings are based on the same measurements.

Acknowledgments

The study was supported by the Max-Planck Society and in part by a grant from the German Research Foundation to FS (DFG SCHL 1968/2-1). AH and JN are supported by the German Federal Ministry of Education and Research (FKZ:01EO1001) and the German Research Foundation (SFB 1052 Obesity mechanisms).

Financial disclosures

Florian Schlagenhauf received funding from the German Research Foundation (SCHL 1969/1-1 & SCHL 1968/2-1). Hans-Jochen Heinze received funding from Novartis and MSD Sharp & Dohme.

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.neuroimage.2014.09.021.

References

Aichert, D.S., Wöstmann, N.M., Costa, A., Macare, C., Wenig, J.R., Möller, H.-J., Rubia, K., et al., 2012. Associations between trait impulsivity and prepotent response inhibition. J. Clin. Exp. Neuropsychol. 37–41 http://dx.doi.org/10.1080/13803395.2012.706261 (September).

Ainslie, G., 1975. Specious reward: a behavioral theory of impulsiveness and impulse control. Psychol. Bull. 82 (4).

Aron, A.R., 2007. The neural basis of inhibition in cognitive control. Neuroscientist 13 (3), 214–228. http://dx.doi.org/10.1177/1073858407299288.

Aron, A.R., Fletcher, P.C., Bullmore, E.T., Sahakian, B.J., Robbins, T.W., 2003. Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. Nat. Neurosci. 6 (2), 115–116. http://dx.doi.org/10.1038/nn1003.

Asahi, S., Okamoto, Y., Okada, G., Yamawaki, S., Yokota, N., 2004. Negative correlation between right prefrontal activity during response inhibition and impulsiveness: a fMRI study. Eur. Arch. Psychiatry Clin. Neurosci. 254 (4), 245–251. http://dx.doi.org/10.1007/s00406-004-0488-z.

Ashburner, J., Friston, K.J., 2005. Unified segmentation. NeuroImage 26 (3), 839–851. http://dx.doi.org/10.1016/j.neuroimage.2005.02.018.

Bari, A., Robbins, T.W., 2013. Inhibition and impulsivity: behavioral and neural basis of response control. Prog. Neurobiol. 108, 44–79. http://dx.doi.org/10.1016/j.pneurobio.2013.06.005.

Basar, K., Sesia, T., Groenewegen, H., Steinbusch, H.W.M., Visser-Vandewalle, V., Temel, Y., 2010. Nucleus accumbens and impulsivity. Prog. Neurobiol. 92 (4), 533–557. http://dx.doi.org/10.1016/j.pneurobio.2010.08.007.

Beck, A., Schlagenhauf, F., Wüstenberg, T., Hein, J., Kienast, T., Kahnt, T., Schmack, K., et al., 2009. Ventral striatal activation during reward anticipation correlates with impulsivity in alcoholics. Biol. Psychiatry 66 (8), 734–742. http://dx.doi.org/10.1016/j.biopsych.2009.04.035.

Bello, E.P., Mateo, Y., Gelman, D.M., Noaín, D., Shin, J.H., Low, M.J., Alvarez, V.A., et al., 2011. Cocaine supersensitivity and enhanced motivation for reward in mice lacking dopamine D2 autoreceptors. Nat. Neurosci. 14 (8), 1033–1038. http://dx.doi.org/10.1038/nn.2862.

Boehler, Carsten N., Hopf, J.-M., Stoppel, C.M., Krebs, R.M., 2012. Motivating inhibition — reward prospect speeds up response cancellation. Cognition 125 (3), 498–503. http://dx.doi.org/10.1016/j.cognition.2012.07.018.

Boehler, Carsten N., Schevernels, H., Hopf, J.-M., Stoppel, C.M., Krebs, R.M., 2014. Reward prospect rapidly speeds up response inhibition via reactive control. Cogn. Affect. Behav. Neurosci. http://dx.doi.org/10.3758/s13415-014-0251-5.

Buckholtz, J.W., Treadway, M.T., Cowan, R.L., Woodward, N.D., Benning, S.D., Li, R., Ansari, M.S., et al., 2010. Mesolimbic dopamine reward system hypersensitivity in individuals with psychopathic traits. Nat. Neurosci. 13 (4), 419–421. http://dx.doi.org/10.1038/nn.2510.

Chambers, C.D., Garavan, H., Bellgrove, M. a, 2009. Insights into the neural basis of response inhibition from cognitive and clinical neuroscience. Neurosci. Biobehav. Rev. 33 (5), 631–646. http://dx.doi.org/10.1016/j.neubiorev.2008.08.016.

Christakou, A., Brammer, M., Rubia, K., 2011. Maturation of limbic corticostriatal activation and connectivity associated with developmental changes in temporal discounting. NeuroImage 54 (2), 1344–1354. http://dx.doi.org/10.1016/j.neuroimage.2010.08.067.

Cyders, M. a, Coskunpinar, A., 2011. Measurement of constructs using self-report and behavioral lab tasks: Is there overlap in nomothetic span and construct representation for impulsivity? Clin. Psychol. Rev. 31 (6), 965–982. http://dx.doi.org/10.1016/j.cpr.2011.06.001.

Cyders, M. a, Dzemidzic, M., Eiler, W.J., Coskunpinar, A., Karyadi, K., Kareken, D.A., 2013. Negative urgency and ventromedial prefrontal cortex responses to alcohol cues: fMRI evidence of emotion-based impulsivity. Alcohol. Clin. Exp. Res. 1–9 http://dx.doi.org/10.1111/acer.12266.

Dick, D.M., Smith, G., Olausson, P., Mitchell, S.H., Robert, F., Malley, S.S.O., Sher, K., 2011. Understanding the construct of impulsivity and its relationship to alcohol use disorders. 15 (2), 217–226. http://dx.doi.org/10.1111/j.1369-1600.2009.00190.x.

Farr, O.M., Hu, S., Zhang, S., Li, C.-S.R., 2012. Decreased saliency processing as a neural measure of Barratt impulsivity in healthy adults. NeuroImage 63 (3), 1070–1077. http://dx.doi.org/10.1016/j.neuroimage.2012.07.049.

Flagel, S.B., Clark, J.J., Robinson, T.E., Mayo, L., Czuj, A., Willuhn, I., Akers, C.A., et al., 2011. A selective role for dopamine in stimulus-reward learning. Nature 469 (7328), 53–57. http://dx.doi.org/10.1038/nature09588.

Forbes, E.E., Brown, S.M., Kimak, M., Ferrell, R.E., Manuck, S.B., Hariri, A.R., 2009. Genetic variation in components of dopamine neurotransmission impacts ventral striatal reactivity associated with impulsivity. Mol. Psychiatry 14 (1), 60–70. http://dx.doi.org/10.1038/sj.mp.4002086.

Ghahremani, D.G., Lee, B., Robertson, C.L., Tabibnia, G., Morgan, A.T., De Shetler, N., Brown, A.K., et al., 2012. Striatal dopamine D_2/D_3 receptors mediate response inhibition and related activity in frontostriatal neural circuitry in humans. J. Neurosci. 32 (21), 7316–7324. http://dx.doi.org/10.1523/JNEUROSCI.4284-11.2012.

Hahn, T., Heinzel, S., Dresler, T., 2011. Association between reward-related activation in the ventral striatum and trait reward sensitivity is moderated by dopamine transporter genotype. Hum. Brain Mapp. 32 (10), 1557–1565. http://dx.doi.org/10.1002/hbm.21127/full.

Hariri, A.R., Brown, S.M., Williamson, D.E., Flory, J.D., De Wit, H., Manuck, S.B., 2006. Preference for immediate over delayed rewards is associated with magnitude of ventral striatal activity. J. Neurosci. 26 (51), 13213–13217. http://dx.doi.org/10.1523/JNEUROSCI.3446-06.2006.

Hayes, A.F., Matthes, J., 2009. Computational procedures for probing interactions in OLS and logistic regression: SPSS and SAS implementations. Behav. Res. Methods 41, 924–936.

Hollerman, J.R., Schultz, W., 1998. Dopamine neurons report an error in the temporal prediction of reward during learning. Nat. Neurosci. 1 (4), 304–309. http://dx.doi.org/10.1038/1124.

Hoogman, M., Aarts, E., Zwiers, M., Slaats-Willemse, D., Naber, M., Onnink, M., Cools, R., Kan, C., Buitelaar, J., Franke, B., 2011. Nitric oxide synthase genotype modulation of impulsivity and ventral striatal activity in adult ADHD patients and healthy comparison subjects. Am. J. Psychiatr. 168 (10), 1099–1107 (Retrieved from http://journals.psychiatryonline.org/article.aspx?articleid=178263).

Horn, N.R., Dolan, M., Elliott, R., Deakin, J.F.W., Woodruff, P.W.R., 2003. Response inhibition and impulsivity: an fMRI study. Neuropsychologia 41 (14), 1959–1966. http://dx.doi.org/10.1016/S0028-3932(03)00077-0.

Knutson, B., Fong, G.W., Bennett, S.M., Adams, C.M., Hommer, D., 2003. A region of mesial prefrontal cortex tracks monetarily rewarding outcomes: characterization with rapid event-related fMRI. NeuroImage 18 (2), 263–272. http://dx.doi.org/10.1016/S1053-8119(02)00057-5.

Körner, A., Geyer, M., Roth, M., 2008. Persönlichkeitsdiagnostik mit dem NEO-Fünf-Faktoren-Inventar: Die 30-Item-Kurzversion (NEO-FFI-30). Psychother. Psychosom. Med. Psychol. 58 (6), 238–245. http://dx.doi.org/10.1055/s.

Lijffijt, M., Bekker, E.M., Quik, E.H., Bakker, J., Kenemans, J.L., Verbaten, M.N., 2004. Differences between low and high trait impulsivity are not associated with differences in inhibitory motor control. J. Atten. Disord. 8 (1), 25–32. http://dx.doi.org/10.1177/108705470400800104.

Logan, G.D., 1994. On the ability to inhibit thought and action: a user's guide to the Stop-signal paradigm. In: Dagenbach, D., Carr, T.H. (Eds.), Inhibitory Processes in Attention, Memory, and Language. Academic Press, San Diego, pp. 189–239.

Logan, G.D., Schachar, R.J., Tannock, R., 1997. Impulsivity and inhibitory control. Psychol. Sci. 8 (1), 60–64. http://dx.doi.org/10.1111/j.1467-9280.1997.tb00545.x.

Patton, J.H., Stanford, M.S., Barratt, E.S., 1995. Factor structure of the Barratt impulsiveness scale. J. Clin. Psychol. 51 (6), 768–774 (Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/14551853).

Pawliczek, C.M., Derntl, B., Kellermann, T., Kohn, N., Gur, R.C., Habel, U., 2013. Inhibitory control and trait aggression: neural and behavioral insights using the emotional Stop-signal task. NeuroImage 79, 264–274. http://dx.doi.org/10.1016/j.neuroimage.2013.04.104.

Plichta, M.M., Scheres, A., 2014. Ventral-striatal responsiveness during reward anticipation in ADHD and its relation to trait impulsivity in the healthy population: A meta-analytic review of the fMRI literature. Neurosci. Biobehav. Rev. 38, 125–134. http://dx.doi.org/10.1016/j.neubiorev.2013.07.012.

Preuss, U.W., Rujescu, D., Giegling, I., Watzke, S., Koller, G., Zetzsche, T., Meisenzahl, E.M., et al., 2008. Psychometric evaluation of the German version of the Barratt Impulsiveness Scale. Nervenarzt 79 (3), 305–319. http://dx.doi.org/10.1007/s00115-007-2360-7.

Robbins, T.W., Gillan, C.M., Smith, D.G., De Wit, S., Ersche, K.D., 2012. Neurocognitive endophenotypes of impulsivity and compulsivity: towards dimensional psychiatry. Trends Cogn. Sci. 16 (1), 81–91. http://dx.doi.org/10.1016/j.tics.2011.11.009.

Scheres, A., Oosterlaan, J., Sergeant, J., 2001. Response inhibition in children with DSM-IV subtypes of AD/HD and related disruptive disorders: the role of reward. Child. Neuropsychol. 7 (3), 172–189. http://dx.doi.org/10.1076/chin.7.3.172.8746.

Scheres, A., Milham, M.P., Knutson, B., Castellanos, F.X., 2007. Ventral striatal hyporesponsiveness during reward anticipation in attention-deficit/hyperactivity disorder. Biol. Psychiatry 61 (5), 720–724. http://dx.doi.org/10.1016/j.biopsych.2006.04.042.

Schlagenhauf, F., Rapp, M.A., Huys, Q.J.M., Beck, A., Wüstenberg, T., Deserno, L., Buchholz, H.-G., et al., 2013. Ventral striatal prediction error signaling is associated with dopamine synthesis capacity and fluid intelligence. Hum. Brain Mapp. 34 (6), 1490–1499. http://dx.doi.org/10.1002/hbm.22000.

Schmidt, K., Metzler, P., 1992. Wortschatztest (WST). Beltz test, Weinheim.

Schultz, W., 1998. Predictive reward signal of dopamine neurons. J. Neurophysiol. 80 (1), 1–27 (Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9658025).

Sinopoli, K.J., Schachar, R., Dennis, M., 2011. Reward improves cancellation and restraint inhibition across childhood and adolescence. Dev. Psychol. 47 (5), 1479–1489. http://dx.doi.org/10.1037/a0024440.

Stanford, M.S., Mathias, C.W., Dougherty, D.M., Lake, S.L., Anderson, N.E., Patton, J.H., 2009. Fifty years of the Barratt Impulsiveness Scale: an update and review. Personal. Individ. Differ. 47 (5), 385–395. http://dx.doi.org/10.1016/j.paid.2009.04.008.

Ströhle, A., Stoy, M., Wrase, J., Schwarzer, S., Schlagenhauf, F., Huss, M., Hein, J., et al., 2008. Reward anticipation and outcomes in adult males with attention-deficit/hyperactivity disorder. NeuroImage 39 (3), 966–972. http://dx.doi.org/10.1016/j.neuroimage.2007.09.044.

Swick, D., Ashley, V., Turken, U., 2011. NeuroImage: Are the neural correlates of stopping and not going identical? Quantitative meta-analysis of two response inhibition tasks. NeuroImage 56 (3), 1655–1665. http://dx.doi.org/10.1016/j.neuroimage.2011.02.070.

Verbruggen, F., Chambers, C.D., Logan, G.D., 2013. Fictitious inhibitory differences: how skewness and slowing distort the estimation of stopping latencies. Psychol. Sci. 24 (3), 352–362. http://dx.doi.org/10.1177/0956797612457390.

Verdejo-García, A., Lawrence, A.J., Clark, L., 2008. Impulsivity as a vulnerability marker for substance-use disorders: review of findings from high-risk research, problem gamblers and genetic association studies. Neurosci. Biobehav. Rev. 32 (4), 777–810. http://dx.doi.org/10.1016/j.neubiorev.2007.11.003.

Von Aster, M., Neubauer, A., Horn, R., 2006. Wechsler Intelligenztest fuer Erwachsene (WIE). Harcourt Test Services, Frankfurt a. M.

Whiteside, S.P., Lynam, D.R., 2001. The Five Factor Model and impulsivity: using a structural model of personality to understand impulsivity. Personal. Individ. Differ. 30 (4), 669–689. http://dx.doi.org/10.1016/S0191-8869(00)00064-7.

Zuckerman, M., Eysenck, S., Eysenck, H.J., 1978. Sensation seeking in England and America: cross-cultural, age, and sex comparisons. J. Consult. Clin. Psychol. 46 (1), 139–149 (Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/627648).