Decreased saliency processing as a neural measure of Barratt impulsivity in healthy adults

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Cognitive control is necessary to navigating through an uncertain world. With the stop signal task (SST), we measure how cognitive control functions in a controlled environment. There has been conflicting evidence on whether trait impulsivity might reflect differences in cognitive control during the SST. While some studies find that trait impulsivity relates to measures of response inhibition, such as the stop signal reaction time (SSRT), other studies do not. Here, in 92 young adult participants (58 females; age 25±4 years), we examined whether trait impulsivity, measured by the Barratt impulsivity scale (BIS-11), is associated with differences in performance and regional brain activations for the component processes of cognitive control during the SST. Across participants, trait impulsivity showed a trend-level correlation with SSRT (F (1.90) = 3.18, p = .07; Pearson regression). In simple regressions, activation of the right anterior dorsal insula and middle frontal cortex (MFC) during stop as compared to go trials negatively correlated with motor and non-planning impulsivity score. Using the generalized form of psychophysiological interaction (gPPI), we showed that functional connectivity of the right insula and MFC with the left dorsolateral prefrontal cortex and bilateral visual areas were also negatively correlated with impulsivity. None of the other component processes of cognitive control, including response inhibition, error processing, post-error slowing, were significantly related to Barratt impulsivity. These results suggest that trait impulsivity as measured by BIS-11 may have distinct effects on saliency processing in adult individuals.

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1. Introduction

Impulsivity can be defined by the following elements: "1) decreased sensitivity to negative consequences of behavior; 2) rapid, unplanned reactions to stimuli before complete processing of information; and 3) lack of regard for long-term consequences" (Moeller et al., 2001). Impulsivity affects and sometimes defines a number of psychiatric disorders, including attention deficit hyperactivity, substance abuse, gambling, schizophrenia, borderline personality and bipolar disorders, many of which are associated with altered inhibitory control (Aron et al., 2003; Dagher and Robbins, 2009; Delisie and Braun, 2011; Goudriaan et al., 2008; Hinvest et al., 2011; Kaladjian et al., 2011; Li et al., 2007; Michalcuk et al., 2011; Odlau et al., 2011; Schuermann et al., 2011; Winstanley et al., 2006). Impulsivity is also a psychological construct that varies as a spectrum across individuals (Dimoska and Johnstone, 2006; Hinvest et al., 2011; Lijffijt et al., 2004; Manuck et al., 1998; Spinella, 2007). Thus, individuals who are otherwise healthy may engage in behavior that disposes them toward negative consequences. It would therefore be useful to understand the role that impulsivity alone plays in cognitive control outside psychiatric disorders and to understand the psychological processes and neural bases of impulsivity.

Cognitive control is widely investigated with the stop signal task (SST; Li et al., 2006, 2008a,b; Logan et al., 1984). The SST involves frequent go stimuli, which require the subject to quickly respond, interspersed with an infrequent stop stimulus that follows the go stimulus and requires subjects to stop responding. Our earlier work suggests that a number of different psychological processes are involved during performance of the SST. Using a staircase procedure in which the difficulty of the stop trials was adjusted according to participants' performance, we separated the processes of response inhibition, error processing, and post-error behavioral adjustment, which are key component processes of cognitive control (Li et al., 2006, 2008a,b). Response inhibition along with attentional monitoring can be used to describe successful stop trials when contrasted with unsuccessful stop trials, or stop errors (Li et al., 2006). Error processing describes the opposite phenomena where the subjects fail to inhibit their response during a stop trial (Li et al., 2008a). Post-error behavioral adjustment is characterized by the tendency of subjects to slow down after they make an error (Li et al., 2008b).

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The stop signal reaction time (SSRT), estimated from each subject's performance, is a measure of inhibitory control, where shorter SSRTs indicate greater capacity of inhibitory control (Logan, 1994), and vice versa. SSRT has been found to positively correlate with impulsivity in healthy adults (Avila and Parcet, 2001; Logan et al., 1997; Marsh et al., 2002). On the other hand, other studies with a similar sample size have found that impulsivity has no effect on SSRT or inhibitory control (Asahi et al., 2004; Lijffijt et al., 2004; Rodríguez-Fornells et al., 2002).

In order to better address these differences in findings and explore the relationship between trait impulsivity and other component processes of cognitive control, we sought to examine how differences in trait impulsivity might influence SST performance in a larger sample of participants. In particular, while much previous work was limited to behavioral performance, here, we investigated the neural correlates of impulsivity with functional magnetic resonance imaging (fMRI) during the SST to understand how brain activity may vary with a participant's impulsivity. To this end, we assessed 92 healthy adults with the Barratt impulsiveness scale, version 11 (BIS-11). We examined how behavioral performance and regional brain activations as well as functional connectivity varied with trait impulsivity during the SST.

2. Methods

2.1. Subjects and behavioral task

A total of 92 healthy participants (58 females; age 25 ± 4 years) participated in this study. Impulsivity was derived from scores on the Barratt impulsiveness scale, version 11 (BIS-11; Barratt and Patton, 1983; Patton et al., 1995). Three subscores of the BIS-11 were also computed for “attentional” or an inability to focus, “motor” or acting without prior thought, and “nonplanning” or not thinking and planning carefully, impulsivities (Patton et al., 1995).

We employed a simple reaction time task in this stop-signal paradigm (Fig. 1; Hu and Li, 2011; Hu et al., 2012; Ide and Li, 2011a; Li et al., 2006, 2009; Logan et al., 1984). There were two trial types: “go” and “stop,” randomly intermixed. A small dot appeared on the screen to engage attention at the beginning of a go trial. After a randomized time interval (fore-period) between 1 and 5 s, the dot turned into a circle (the “go” signal), which served as an imperative stimulus, prompting the subject to quickly press a button. The circle vanished at a button press or after 1 s had elapsed, whichever came first, and the trial terminated. A premature button press prior to the appearance of the circle also terminated the trial. About three quarters of all trials were go trials. The remaining one quarter were stop trials. In a stop trial, an additional “X,” the “stop” signal, appeared after and replaced the go signal. The subjects were told to withhold button press upon seeing the stop signal. Likewise, a trial terminated at button press or when 1 s had elapsed since the appearance of the stop signal. The stop signal delay (SSD) – the time interval between the go and stop signal – started at 200 ms and varied from one stop trial to the next according to a staircase procedure, increasing and decreasing by 64 ms each after a successful or failed stop trial (De Jong et al., 1990; Levitt, 1970). There was an inter-trial-interval of 2 s. Subjects were instructed to respond to the go signal quickly while keeping in mind that a stop signal could come up in a small number of trials. In the scanner each subject completed four 10-minute runs of the task with the SSD updated manually across runs. Depending on the actual stimulus timing (trials varied in fore-period duration) and speed of response, the total number of trials varied slightly across subjects in an experiment. With the staircase procedure, we anticipated that the subjects would succeed in withholding their response in approximately half of the stop trials. The stop signal reaction time was computed by subtracting the critical stop signal delay, or the estimated SSD required for a subject to get half of stop trials correct, from the median go reaction time (Li et al., 2008a).

2.2. Imaging protocol

Conventional T1-weighted spin echo sagittal anatomical images were acquired for slice localization using a 3T scanner (Siemens Trio). Anatomical images of the functional slice locations were next obtained with spin echo imaging in the axial plane parallel to the AC–PC line with TR = 300 ms, TE = 2.5 ms, bandwidth = 300 Hz/pixel, flip angle = 60°, field of view = 220 × 220 mm, matrix = 256 × 256, 32 slices with slice thickness = 4 mm and no gap. Functional, blood oxygenation level dependent (BOLD) signals were then acquired with a single-shot gradient echo echo-planar imaging (EPI) sequence. Thirty-two axial slices parallel to the AC–PC line covering the whole brain were acquired with TR = 2000 ms, TE = 25 ms, bandwidth = 2004 Hz/pixel, flip angle = 85°, field of view = 220 × 220 mm, matrix = 64 × 64, 32 slices with slice thickness = 4 mm and no gap. Three hundred images were acquired in each run for a total of four runs.

2.3. Data analysis and statistics

Data were analyzed with Statistical Parametric Mapping version 8 (SPM8, Wellcome Department of Imaging Neuroscience, University College London, U.K.). Images from the first five TRs at the beginning of each run were discarded to enable the signal to achieve steady-state equilibrium between RF pulsing and relaxation. Images of each individual subject were first corrected for slice timing and realigned (motion-corrected). A mean functional image volume was constructed for each subject for each run from the realigned image volumes. These mean images were normalized to an MNI (Montreal Neurological Institute) EPI template with affine registration followed by nonlinear transformation (Ashburner and Friston, 1999; Friston et al., 1995a). The normalization parameters determined for the mean functional volume were then applied to the corresponding functional image volumes for each subject. Finally, images were smoothed with a Gaussian kernel of 8 mm at full width at half maximum.

In the first general linear model (GLM), four main types of trial outcome were distinguished: go success (G), go error (F), stop success (SS), and stop error (SE) trial. The SS and SE trials were identical in stimulus condition, with SS and SE trials each involving inhibition success and failure, respectively. The contrast SS > SE thus engaged processes related to attentional monitoring and response inhibition (Duann et al., 2009; Li et al., 2006). The opposite contrast SE > SS highlights processes related to error detection (Hendrick et al., 2010; Ide and Li, 2011b). An SS or SE, together stop (S), trial involves incongruent goals between the prepotent tendency to respond and the motor intention to withhold the response. S trials are also infrequent compared to go trials, making them...
highly salient. Thus, we interpreted the contrast of S>G as reflecting saliency processing (Hendrick et al., 2011).

In a second GLM, G, F, SS, and SE trials were first distinguished. G trials were divided into those that followed a G (pG), SS (pSS), and SE (pSE) trial. Furthermore, pSE trials were divided into those that increased in RT (pSEi) and those that did not increase in RT (pSEni), to allow the isolation of neural processes involved in post-error behavioral adjustment (Li et al., 2008a). To determine whether a pSE trial increased or did not increase in RT, it was compared with the pG trials that preceded it in time during each session. The pG trials that followed the pSE trial were not included for comparison because the neural/cognitive processes associated with these pG trials occurred subsequent to and thus could not have a causal effect on the pSE trial (Li et al., 2008a). We constructed for each individual subject 2 contrasts: SS>SE, to compare with the first GLM and verify the model; and pSEi versus pSEni, to identify activations associated with post-error slowing (PES).

A statistical analytical design was constructed for each individual subject, using the general linear model (GLM) with the onsets of go signal in each of these trial types convolved with a canonical hemodynamic response function (HRF) and with the temporal derivative of the canonical HRF and entered as regressors in the model (Friston et al., 1995b). Realignment parameters in all 6 dimensions were also entered in the model. The data were high-pass filtered (1/128 Hz cutoff) to remove low-frequency signal drifts. Serial autocorrelation of the time series violated the GLM assumption of the independence of the error term and was corrected by a first-degree autoregressive or AR(1) model (Friston et al., 2000). The GLM estimated the component of variance that could be explained by each of the regressors.

The con or contrast (difference in %) images of the first-level analysis were used for the second-level group statistics (random effects analysis; Penny and Holmes, 2004). These images were correlated with the total BIS score in a simple regression across subjects and the results were reported at a threshold of p<.001, uncorrected for multiple comparisons. To explore the association with the subcomponent of Barratt impulsivity, these images were also correlated with the three subscores of BIS-11 and the results were reported at a threshold of p<.05, family wise error (FWE) corrected for the whole brain. Brain regions were identified using an atlas (Duvernoy, 1999). All templates are in Montreal Neurological Institute (MNI) space and voxel activations are presented in MNI coordinates.

2.4. Connectivity analysis: psychophysiological interaction (PPI)

PPI describes how functional connectivity between brain regions is altered as a result of psychological context or variables (Friston et al., 1997; Gitelman et al., 2003). To accommodate three task conditions (G, SS, SE), we used a generalized form of context-dependent psychophysiological interaction (gPPI, http://brainmap.wisc.edu/PPI, McLaren et al., 2008). Briefly, in gPPI, the condition onset times for G, SS, and SE are separately convolved with the canonical hemodynamic response function (HRF) for each condition, forming the psychological regressors. The interaction term is then performed psychophysiological interaction analysis (gPPI) using AR(1) model (Friston et al., 2000). The GLM estimated the component of variance that could be explained by each of the regressors.

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3. Results

3.1. Impulsivity and behavioral performance

The total BIS-11 scores of the 92 subjects ranged from 41 to 82 (mean ± SD = 60 ± 9) out of a possible 120, where a higher score indicates greater impulsivity. The average go trial response rate was 96.0 ± 1.6%, while the stop success rate was 53.4 ± 3.8%. Average go trial reaction time (GoRT) and SSRT was 563 ± 117 ms and 216 ± 44 ms, respectively. In a simple regression across subjects, trait impulsivity showed a weak, trend-level correlation with SSRT (R = .19, F(1,90) = 3.49, p = .064; Pearson regression). Motor impulsivity (range: 15–31) showed a slightly stronger albeit trend-level correlation with SSRT (R = .20, F(1,90) = 3.64, p < .059), while nonplanning (range: 15–35) and attentional (range: 8–22) impulsivity did not correlate with SSRT (R = .16, F(1,90) = 2.50, p < .11; R = .10, F(1,90) = .91, p < .34, respectively). Impulsivity also did not show a significant correlation with post-error slowing (total score: R = .14, F(1,90) = 1.73, p < .19; motor subscore: R = .14, F(1,90) = 1.88, p < .17; nonplanning subscore: R = .14, F(1,90) = 1.70, p < .19; attentional subscore: R = .08, F(1,90) = .57, p < .45).

3.2. Impulsivity and response inhibition, error processing, and post-error slowing

Regressions of the BIS-11 impulsiveness ratings with contrasts for attentional monitoring and response inhibition (SS>SE), error processing (SE>SS), and post-error slowing did not reveal any significant regional brain activations, even at a threshold of p<.005, uncorrected.

3.3. Impulsivity and saliency processing

In a one sample t-test across all subjects for the stop as compared to go contrast, we observed the typical activations of anterior cingulate cortex and supplementary motor area, insula and middle frontal cortex, inferior parietal cortex, and some subcortical structures including thalamus and posterior part of the caudate head, at p<.05, FWE corrected (Fig. 2).

In a simple regression against BIS scores, with age and gender as covariates, activations of the right anterior dorsal insula (MNI coordinates = 36, 8, 19; Z(peak) = 3.87; Extent = 24 voxels) and right middle frontal cortex (MFC; MNI coordinates = 36, 20, 28; Z(peak) = 3.57; Extent = 18 voxels) during stop as compared to go trials were negatively correlated with BIS score, at p<.001, uncorrected. The subscore of motor impulsivity negatively correlated to activation of both the right insula (MNI coordinates = 30, 8, 19; Z(peak) = 4.24; Extent = 117 voxels) and MFC (MNI coordinates = 35, 23, 28; Z(peak) = 4.05; Extent = 117 voxels) during stop as compared to go trials (p<.046, FWE corrected for the whole brain; Fig. 3a). The nonplanning impulsivity subscore inversely correlated with an activation in the right insula (MNI coordinates = 30, 8, 22; Z(peak) = 4.17; Extent = 112 voxels; p<.051, FWE corrected for the whole brain; Fig. 3b). Attentional impulsivity did not show any correlated brain activations.

3.4. Impulsivity and functional connectivity during saliency processing

Using each of the ROIs generated from the regression analysis, we then performed psychophysiological interaction analysis (gPPI) using stop as compared to go trials as the psychological regressor and each ROI separately as a physiological variable.

In a regression of the PPI term against the total BIS score, impulsivity was negatively correlated with PPI of the right insula and the left inferior precentral sulcus/precentral gyrus (MNI coordinates = −27, −7, 37; Z(peak) = 4.02; Extent = 51 voxels) and bilateral cuneus.
and PPI of the right MFC and the left DLPFC/superior frontal sulcus (MNI coordinates = −24, 14, 55; Z(peak) = 4.29; Extent = 54 voxels), precuneus (MNI coordinates = −12, −58, 46; Z(peak) = 4.80; Extent = 1127 voxels), and intraparietal sulcus/angular gyrus (MNI coordinates = −30, −70, 46; −21, −88, 7; 27, −55, 31; Z(peak) = 4.80; 4.58; Extent = 1127; 125 voxels), all at pb .05, FWE corrected (Figs. 4a and b).

4. Discussion

4.1. Barratt impulsivity and saliency processing

Barratt impulsivity is associated with decreased activation of the right anterior insula and middle frontal cortex during saliency processing. The right anterior insula and middle frontal cortex respond to both stop success and error trials in the stop signal and go-nogo tasks (Garavan et al., 1999; Hampshire et al., 2010; Kawashima et al., 1996; Rubia et al., 2003; Swainson et al., 2003). These activations have been hypothesized to signal saliency processing, a response to the infrequent stop, as compared to the frequent go signal during the stop signal task. Compared to the go signal, the stop signal is salient also because it instructs a change in act: to inhibit the prepotent go response. For this reason, some investigators have argued that a contrast between stop and go trials may implicat response inhibition, in addition to saliency processing (Dimoska et al., 2003; Pliszka et al., 2000; Rubia et al., 2001). On the other hand, studies have reported greater insular activation during stop error as compared to stop success trials (Garavan et al., 1999, 2002; Hester et al., 2004). These latter results suggest that insular activation most likely reflects saliency processing in association with the infrequency (or “oddball”) effects of stop/nogo trials or a signal of surprise associated with errors, since inhibition is in place to a greater extent in stop success than error trials. In further support of a role of the insula in saliency processing, Ramautar et al. (2006) found greater bilateral insular activation during stop error trials in a block with less frequent stop trials than one with more frequent stop trials.

Salient stimuli are abundant in the environment. Thus, the anterior insula responds to social exclusion of friends versus strangers (Meyer et al., 2012), novel and ambiguous versus unambiguous visual inputs (Jepma et al., 2012), exteroceptive versus interoceptive attention (Farb et al., 2012), and unexpected versus expected taste stimuli (Veldhuizen et al., 2011). In other studies, insula has been found to respond to salience over valuation during decision making (Litt et al., 2011), stimulus valence and cognitive demand (Gu et al., 2012), moment to moment adjustment in task set maintenance (Wilk et al., 2012), and attentional processing in a cingulo-opercular attentional network that controls goal-directed behaviors (Dosenbach et al., 2007). Additionally, the dorsal anterior insula showed greater responses to risky as compared to non-risky choices, with the responses scaling in proportion to an anxiety rating (Tang et al., 2011). Other studies showed that activations of anterior insula to threatening/emotional as compared to safe/neutral stimuli increased during an anxiety state (Ball et al., 2012; Choi et al., 2012), consistent with a bulk of earlier work on the relationship between insular functions and anxiety trait or clinical anxiety disorders (see Etkin, 2010; Holzschneider and
These findings mirror our results of decreased insular activation in impulsive individuals because anxiety and impulsivity are anti-correlated personality traits (Li and Chen, 2006).

Similarly, the middle frontal cortical (MFC) cluster, as part of the broadly defined lateral prefrontal cortex, is widely implicated in attentional processing (Bunge et al., 2005; Crone et al., 2006; Dosenbach et al., 2006; Fan et al., 2005). This MFC region increased activation during “orienting” trials where subjects had to shift their attention to a different location in space (Fan et al., 2005). This area was shown to maintain but not manipulate item information during a working memory task, suggesting that it helps sustain attention (Crone et al., 2006). The current results are also consistent with an earlier report that activation of a similar right lateral prefrontal area during nogo as compared to go trials inversely correlated with motor impulsivity during the go-nogo task (Asahi et al., 2004).

Nonetheless, we acknowledge that the contrast of stop as compared to go trials may also involve some level of response inhibition in addition to saliency processing. For instance, Chikazoe et al. (2009) reported that the insula and middle frontal regions showed greater responses to stop and to uncertain go trials as compared to certain go trials, suggesting that these regions may be involved in both preparation to stop and may mediate response inhibition and not simply saliency processing. These regions have also been linked to the preparation to inhibit, such as during the fixation period before a no-go or incongruent trial, which does not involve explicit processing of visual stimuli (Fassbender et al., 2006; Hester et al., 2004).

An additional issue is that saliency has been broadly defined across a wide array of behavioral tasks. For instance, fearful faces, painful stimulation, and negative reward evoked activations in the amygdala and midbrain in a saliency network (Berns et al., 2008; Garrido et al., 2012; Vuilleumier et al., 2003). In addition, visual search and target detection – highly salient events – are strongly linked to the frontoparietal attention network and the visual cortices (Beck et al., 2001; Corbetta and Shulman, 2002; Shulman et al., 2001). Similarly, ACC/SMA, insula, and DLPFC are known to be involved in spatial reorienting to salient stimuli, for instance after a change in feature or color, across a variety of tasks (Corbetta and Shulman, 2002; Downar et al., 2000, 2002; Peelen et al., 2004). Evidence from lesion studies shows that middle frontal cortex is particularly important to detecting and orienting attention to infrequent, or salient, events (Daffner et al., 2000; Knight and Scabini, 1998). Thus, the current findings characterize saliency processing in a broad sense, in accord with these earlier studies, but need to be contrasted with saliency related areas in behavioral tasks implicating affective and/or reward processing.

4.2. Impulsivity and functional connectivity during saliency processing

Connectivity analyses on PPI demonstrated an inverse correlation with impulsivity in functional connectivity between insula/MFC and the left superior frontal sulcus, bilateral cuneus/dorsal precuneus, and the intraparietal sulcus/angular gyrus during stop as compared to go trials. These frontal parietal cortical areas and dorsal precuneus are parts of a well-defined visuospatial attention network (Corbetta and Shulman, 2002; Dosenbach et al., 2007; Kastner et al., 1999; Nobre et al., 1997; Zhang and Li, 2012a, 2012b). Both the angular gyrus and the intraparietal sulcus are part of the inferior parietal lobe, which is known to mediate visuospatial attention (Chambers et al., 2004; Corbetta and Shulman, 2002; Egner et al., 2008). The dorsal precuneus is involved in attentional orienting across a variety of behavioral tasks (Cavanna and Trimble, 2006; Culham et al., 1998; Le et al., 1998; Nagahama et al., 1999). In particular, functional connectivity mapping showed that the dorsal precuneus along with an array of cortical and subcortical structures is involved in cognitive challenges that require visuospatial and motor attention. Similarly, cuneus increased activation to salient stimuli (Carretté et al., 2004) and modulation in value and salience during a decision-making task (Litt et al., 2011). Several imaging studies have shown that attention modulates visual cortical activation and that allocation of attention to either the peripheral or central visual field changes visual cortical activation accordingly (Brefczynski and DeYoe, 1999; Slotnick et al., 2003; Smith et al., 2000). Altogether, these results support the finding that individuals who are less impulsive have more attention-related functional connectivity between insula and MFC with visual processing areas and prefrontal cortex during saliency processing than those who are more impulsive.

The superior frontal cortex has also been implicated in a variety of cognitive control tasks (Chao et al., 2009; Greening et al., 2011; Jamadar et al., 2010; Kadota et al., 2010; Li et al., 2006; Mansouri et al., 2007; Zhang and Li, 2012a). For instance, this dorsal prefrontal region responded to nogo
trials during go/no-go tasks (Nakata et al., 2008; Rubia et al., 2001) and to inhibition of stereotyped responses in a modified rock paper scissors task (Kadota et al., 2010). Lesions of the dorsal prefrontal cortex led to deficits in cognitive control in a modified Wisconsin Card Sorting Task in non-human primates (Mansouri et al., 2007). Thus, impulsivity may impart decreased connectivity to this prefrontal structure for cognitive control during saliency processing.

### 4.3. Barratt impulsivity and other attentional functions

Impulsivity has been see to negatively affect attention in other behavioral tasks. For instance, during a visual oddball task, Russo et al. (2008) found that subjects with higher Barratt impulsivity showed lower P300 wave amplitude, a measure of stimulus evaluation and classification time, likely due to their inability to inhibit task-irrelevant behavior and maintain sustained attention. In a rapid serial visual presentation (RSVP) task, participants are required to identify targets among a series of distractors. Typically, the ability to identify a target decreases right after the detection of a target and this “attentional blink” quantifies one’s attentional capacity. Attentional blink has been found to be more severe in individuals with higher impulsivity (Li et al., 2005). Importantly, attentional processing during the RSVP task involves the middle frontal cortex, likely as part of the attentional network (Marcantonii et al., 2003; Marois et al., 2000). Altogether, this supports an association between Barratt impulsivity and impaired attentional processing in a cognitive task.

### 4.4. Barratt impulsivity and inhibitory control

We observed that SSRT only correlates to impulsivity at a trend-level of statistical significance in our cohort of 92 subjects. Along with earlier studies that showed mixed results in the association of trait impulsivity and inhibitory control (Avila and Parcet, 2001; Lijffijt et al., 2004; Logan et al., 1997; Marsh et al., 2002; Rodriguez-Fornells et al., 2002), this finding suggests that trait impulsivity could at best explain a small fraction of the variance in inhibitory control, as measured by the SSRT. We also did not observe a correlation of Barratt impulsivity total or subscore with post-error slowing (PES) or regional activations during PES. These findings suggest that Barratt impulsivity accounts for little inter-subject variability in inhibitory control and performance monitoring, as assessed by the SST.

It is important to note that there are different instruments to assess impulsivity. For instance, the commonly used version of Eysenck’s scales, the I-7, measures two broad constructs of impulsivity: “impulsiveness” and “venturesomeness” (Eysenck et al., 1985). BIS-11 and I-7 both have good test–retest reliability and often relate to behavioral measures of impulsiveness such as by the Matching Familiar Figures Test (MFFT; Asahi et al., 2004; Carillo-de-la-Pena et al., 1993; Gerbing et al., 1987; Kagan et al., 1964; Kagan et al., 1966; Luengo et al., 1991). Eysenck’s and BIS scores obtained on the same subjects correlated to each other, with the BIS motor impulsivity subscale showing the highest correlation to total impulsiveness scores on the Eysenck’s scale (Carillo-de-la-Pena et al., 1993; Luengo et al., 1991). On the other hand, neither scale appeared to fully explain behavioral measures nor teacher ratings of impulsivity (Carillo-de-la-Pena et al., 1993; Luengo et al., 1991), suggesting that there are still aspects of impulsivity that are not described by these self-report assessments.

Another popular instrument is the UPPS (urgency, premeditation, perseverance, and sensation seeking) impulsivity behavior scale, which measures four subscales: urgency, (lack of) premeditation, (lack of) perseverance, and sensation seeking (Whiteside and Lynam, 2003). Whiteside and Lynam (2003) found that the impulsiveness subscale of the Eysenck’s loaded onto their premeditation factor while venturesomeness loaded onto the sensation seeking factor. Patients with borderline personality disorder, which has impulsivity as one of the nine diagnostic criteria in the DSM-IV, scored higher than healthy controls on all three of the BIS-11 subscales, but only the impulsiveness subscale of the Eysenck’s and only the perseverance and urgency subscales of the UPPS (Jacob et al., 2010a, 2010b). These results provide further support for the multidimensionality of impulsivity. While all impulsivity scales measure impulsiveness, they vary in the dimensions of the impulsivity that they are able to capture. Future studies are required to examine whether impulsivity measured by instruments other than BIS-11 may be related to response inhibition and performance monitoring in the stop signal task.

### 4.5. Potential clinical implications

As described earlier, impulsivity is an important characteristic of many clinical conditions. Decreased insular activation during risk-taking is associated with hazardous drinking (Claus and Hutchison, 2012). Individuals with generalized anxiety disorders show greater anterior insular activation to fearful as compared to happy faces (Klumpp et al., 2012). Anterior insula increased and supplementary motor area decreased in activation during motor preparation in conversion disorder patients, suggesting that heightened arousal and limbic activity may disrupt top–down motor control (Voon et al., 2011a, 2011b). During reward tasks, increased impulsivity is related to lower ventral striatal activations in alcoholics (Beck et al., 2009). Poorer performance on the Iowa Gambling Task was also associated with increased impulsivity in MDMA users (Hanson et al., 2008). Thus, the current results may add to this literature by providing insights to cognitive deficits in individuals of impulse control disorders or other mental conditions where impulsivity represents a clinical concern.

### 4.6. Conclusions

Barratt impulsivity is associated with hypoactivation of frontal cortex and anterior insula and decreased functional connectivity with an attention network of brain regions during saliency processing. On the other hand, the current results do not appear to support a commonly assumed link between Barratt impulsivity and response inhibition or post-error slowing during cognitive control.

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### References


