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Reactive and proactive control mechanisms of response inhibition in gambling disorder

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ABSTRACT

Response inhibition, one component of cognitive control, refers to the ability to inhibit automatic responses and has been found to be impaired in gambling disorder. Recent models of cognitive control distinguish between two mechanisms: reactive control, the ability to stop in response to a stop-stimulus, and proactive control, the ability to anticipate and prepare for a stop. Previous studies have primarily focused on reactive modes of control in gambling disorder. The aim of the current study was to assess both reactive and proactive modes of response inhibition in individuals with gambling disorder (n = 27) and community controls (n = 21) using a variant of the stop-signal task. Second, the relationship between trait impulsivity, and reactive and proactive control was examined. No group differences in reactive or proactive control were found. However, premeditation, one domain of trait impulsivity, was associated with worse proactive control in the gambling group. These results suggest that difficulties with response inhibition may not be a core deficit in all forms of gambling disorder. Future research should continue to develop and test tasks that involve cognitive control processes in different presentations of gambling disorder.

1. Introduction

1.1. Gambling disorder and impulsivity

Gambling disorder affects 1–3% of the general population and is characterized by persistent and recurrent maladaptive gambling behaviors leading to social, occupational, financial, and health impairments (American Psychological Association [APA], 2013; Okuda et al., 2009). Impulsivity is a central feature implicated in the development and maintenance of gambling disorder and has been suggested to be one of the most robust characteristics associated with the disorder (Hodgins and Holub, 2015; MacKillop et al., 2014; Wareham and Potenza, 2010).

Broadly defined, impulsivity is the tendency to act rashly without adequate consideration of adverse consequences (Hodgins and Holub, 2015). Cross-sectional studies have revealed higher scores on self-report and cognitive measures of impulsivity in persons with gambling disorder compared to control groups (Ledgerwood et al., 2009; Michalczuk et al., 2011). Significant positive relationships between various facets of impulsivity and problem gambling severity have also been established in adolescents and adults (Alessi and Petry, 2003; Cyders and Smith, 2008; Secades-Villa et al., 2016; Steel and Blaszczynski, 1998). Moreover, longitudinal studies indicate a strong etiological role of impulsivity in gambling disorder (MacKillop et al., 2014; Shenassa et al., 2012; Vitaro et al., 1999). To date, however, there is a lack of consensus in the gambling research concerning the various conceptualizations and components of impulsivity.

The various conceptualizations of impulsivity can be seen in the wide range of assessment modalities used to index different components of impulsivity. Personality models define impulsivity as a stable enduring trait (trait impulsivity) measured through self-report questionnaires such as the Baratt Impulsivity Scale (BIS) (Patton et al., 1995) and the UPPS Impulsive Behavior Scale (UPPS) (Whiteside and Lynam, 2001). At a cognitive level, impulsivity is manifested via two behavioral expressions: impulsive choice and impulsive action (Brevers et al., 2012). Impulsive choice refers to the tendency to select higher-risk immediate rewards over delayed lower-risk rewards with greater benefit, and is measured using delayed discounting tasks (see Matta et al., 2012). Impulsive action is characterized as a deficit in response inhibition/inhibitory control (Conversano et al., 2012; Lawrence et al., 2009a), and is predominately assessed using laboratory-based behavioral tasks such as the stop-signal and go/no-go task.

Response inhibition refers to the ability to suppress actions that are no longer relevant or inappropriate in certain situations (Mostofsky and Simmonds, 2008). Deficits in response inhibition lead to interference in

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goal-directed behavior and may result in detrimental impulsive actions (Bari and Robbins, 2013; Verbruggen and Logan, 2008). In the context of a gambling addiction, impaired response inhibition may lead to greater engagement in gambling activities and numerous failed attempts to stop. Indeed, higher disinhibition, as measured by the stop-signal task, has been linked to more severe forms of gambling disorder (Brevers and Noel, 2013) and is associated with higher rates of relapse (Goudriaan et al., 2008). There is currently a thin body of literature that investigates response inhibition in gambling disorder and extant studies have produced inconsistent findings (Chowdhury et al., 2017).

1.2. Measures of response inhibition

As previously mentioned, the go/no-go and stop-signal tasks are the two dominant paradigms used to assess response inhibition. The standard *go/no-go tasks* consist of 'go' stimuli and 'no-go' stimuli; participants are instructed to respond as quickly as possible to go stimuli by pressing a button and are required to inhibit their responses to the no-go stimuli (Verbruggen and Logan, 2008). In this paradigm, response inhibition is measured by the ability to inhibit these responses.

Standard *stop-signal tasks* require subjects to respond to one set of stimuli during most trials. However, on a minority of trials, subjects must inhibit an *already initiated* response when they see a stop-signal (Verbruggen and Logan, 2008). As described comprehensively by Logan et al. (1984), the stop-signal paradigm is based on the "horse-race model" of stopping. The stop-signal reaction time (SSRT), an individual's stop latency, is the chief measure used to determine response inhibition deficits. Longer SSRTs are indicative of impairment in response inhibition (Chowdhury et al., 2017).

Although the stop-signal and go/no-go tasks are similar, there are important differences. First, the go/no-go paradigms are primarily a measure of inhibition failure/commission errors whereas the stop-signal task paradigms provide an index for the ability to inhibit a response that is already underway. Unlike in go/no-go tasks, stopping latency can be derived from stop-signal tasks using the horse-race model (Logan et al., 1984). Second, the no-go stimulus in standard go/no-go tasks is presented simultaneously with or in place of the go stimulus, whereas in stop-signal tasks, the stop-signal is presented after the go stimulus (Littman and Takács, 2017). Third, on a neural level, the go/no-go task reflects the mere restriction of a movement plan while the stop-signal task requires control within regions of the motor system that are already activated (Aron, 2011; Congdon et al., 2012).

1.3. Reactive and proactive processes in response inhibition

Response inhibition is considered a complex sub-process of cognitive control rather than a unitary construct (Brydges et al., 2012; Roberts et al., 2011). According to the recently developed 'dual mechanisms of control' framework, cognitive control constitutes two distinct yet complimentary modes– reactive and proactive control (Braver, 2012). Reactive control is thought of as a "late correction" mechanism involved in conflict resolution after an interfering stimulus (e.g., stop-signal) has been presented (Khng and Lee, 2014). The SSRT in the standard stop-signal task is considered a measure of reactive control (Smittenaar et al., 2015). Conversely, proactive control is considered an "early selection" mechanism involved in the anticipation and prevention of an interfering or cognitively demanding stimulus before it occurs. Aron (2011) advocates for the inclusion of proactive control in current models of response inhibition as many everyday situations (1) require selectivity, (2) involve more than solely a rapid stopping process, and (3) require goal-driven control.

The standard stop-signal task is largely a measure of reactive control, and thus, has been argued to be a limited model of response inhibition in psychiatric patients (Smittenaar et al., 2015). Past studies of reactive control in gambling disorder using the standard stop-signal task have produced mixed results. A few studies have found no significant impairment in individuals with gambling disorder (de Ruiter et al., 2012; Leppink et al., 2016; Lorains et al., 2014). Contrastingly, a majority of studies using the stop-signal task have identified significant impairments in response inhibition among gamblers (Brevers et al., 2012; Brevers and Noel, 2013; Chowdhury et al., 2017; Goudriaan et al., 2005; Goudriaan et al., 2006; Odlaug et al., 2011; Smith et al., 2014).

An important next step to further the research on response inhibition in gambling disorder is the investigation of reactive and proactive control mechanisms. The stop-signal anticipation task (SSAT) is a modified variant of the stop-signal task, which provides a measure of both reactive and proactive control (Zandbelt and Vink, 2010). In this paradigm, visual cues are used to indicate stop-signal probability as a way to manipulate the anticipation of stopping. As stop-signal likelihood increases, participants are expected to utilize these cues to proactively prepare an inhibitory response. Studies using the SSAT and other tasks with similar parameters, have observed increasing reaction times as a function of higher stop-signal probability in healthy populations (Vink et al., 2005; Zandbelt and Vink, 2010; Zandbelt et al., 2013a; 2013b). In clinical populations (e.g., schizophrenia), proactive control has been shown to be impaired (Lesh et al., 2013; Vink et al., 2006; Zandbelt et al., 2011).

Further studies are warranted in other clinical populations such as gambling disorder for a more nuanced understanding of the mechanisms of control in response inhibition (i.e. reactive and proactive control). Moreover, the relationship between self-reported impulsivity and both reactive and proactive control requires further study, as problem gamblers who obtain high scores on self-report measures of impulsivity may not necessarily display impaired control over motor responses (Leppink et al., 2016; MacKillop et al., 2014). By dissociating reactive and proactive control, it may be possible to target appropriate mechanisms of response inhibition in treatments designed for individuals with gambling disorder. Furthermore, if reactive and/or proactive control are associated with specific impulsive traits, treatments can be individualized for different subtypes of gamblers.

1.4. Objectives and hypotheses of present study

The present study had three main objectives. First, to replicate past findings of reactive control in gambling disorder, overall SSRT was examined. Consistent with previous studies, we expected gamblers would have slower SSRTs, indicating higher impulsive responding. Second, proactive control was investigated; we hypothesized gamblers relative to controls, would have more difficulty anticipating the occurrence of stop-signals. The third objective of the study was exploratory. The relationship between different mechanisms of response inhibition (proactive and reactive control) and self-reported trait impulsivity, as measured by the UPPS-P, was examined.

2. Methodology

2.1. Participants and recruitment

Twenty-seven individuals with a lifetime gambling disorder diagnosis and 21 controls were recruited from the community through media announcements, notices at local treatment agencies, and an existing registry of individuals interested in research participation. The Composite International Diagnostic Interview (CIDI) was used to identify lifetime DSM-5 gambling disorder diagnosis. For the purposes of this study, the term "gambler" will be used to refer to the individuals with gambling disorder who participated in the present study. All participants completed a pre-screening phone interview and an in-person screening visit to determine eligibility for the study.

To be eligible, all participants had to be over the age of 18 to sign consent for the study. The exclusion criteria for all participants included: (1) an intelligence quotient (IQ) less than 80 on the Wechsler

Test of Adult Reading (2) diagnosis of a past or current psychotic disorder, (3) a current or past neurological condition known to significantly influence neurocognitive function (e.g. multiple sclerosis, epilepsy), (4) uncorrectable visual problems that could interfere with study procedures, (5) color blindness, (6) history of stroke, and (7) a diagnosis of dementia, delirium, or another cognitive disorder. Community controls with a personal or family history of gambling disorder were excluded.

2.2. Procedure

The study was approved by the University of Calgary Research Ethics Board. All research participants who met inclusion criteria provided written informed consent. The Structured Clinical Interview for DSM-5 Disorders (SCID-5) was administered to assess mood disorders, schizophrenia spectrum and other psychotic disorders, substance use disorders, anxiety disorders, feeding and eating disorders, obsessive-compulsive and related disorders, trauma and stressor-related disorders, and adult attention-deficit/hyperactivity disorder. Diagnoses for all participants were assigned at case consensus meetings, which were attended by all trained interviewers and at least one principal investigator (VG or DH). Other clinical interviews and self-report measures were also collected to obtain information regarding diagnoses, medical history, past brain injury, gambling severity, impulsivity, and substance use.

The stop-signal anticipation task (SSAT) was administered amongst a larger cognitive battery. All tasks in this extensive battery were pre-ordered in a way to facilitate optimal attention and alternate between tasks measuring different cognitive domains. Participants were reimbursed for their time in the form of gift cards.

2.3. Measures

2.3.1. Clinical measures

The gambling module of the Composite International Diagnostic Interview (CIDI) was used to assess frequency of gambling, gambling history, and whether participants met criteria for lifetime gambling disorder. The CIDI is a structured clinical interview based on the diagnostic criteria outlined by the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (Kessler et al., 2008; Hodgins et al., 2012). To remain relevant to the most recent conceptualization of gambling disorder, the information from the CIDI was used to determine diagnosis based on the DSM-5 criteria.

Each participant also completed the Hamilton Depression Scale (HAM-D) to evaluate depressive symptoms experienced in the past week., as mood has been found to alter cognitive performance (Porter et al., 2008). Though not an exclusion criterion, information about past acquired brain injuries was collected using the Brain Injury Screening Questionnaire (Part One; BISQ) to account for the potential impact of brain injury on cognition. Finally, the Problem Gambling Severity Index (PGSI; Ferris and Wynn, 2001) was used to assess problem gambling severity in the past 12 months. Subjects answered nine questions on a 4-point alternative scale (0 = never, 1 = sometimes, 2 = most of the time, 3 = almost always. Scoring criteria for the PGSI outlined by Currie et al. (2013) were used to determine gambling severity.

2.3.2. Impulsivity measures

UPPS impulsive behavior scale (UPPS-P). The UPPS-P is a 59-item questionnaire designed to measure five distinct facets of personality associated with impulsive behavior: negative urgency, positive urgency, (lack of) perseverance, (lack of) premeditation, and sensation seeking (Whiteside and Lynam, 2001). Participants responded to each item of the UPPS-P on a 4-point Likert scale ranging from 1 (strongly agree) to 4 (strongly disagree). The UPPS-P was selected as it contains domains related to emotionality unlike other brief self-report measures of impulsivity (e.g., Barratt Impulsiveness Scale).

Stop-signal anticipation task (SSAT). The SSAT was developed by Zandbelt and Vink (2010) and is based on the original stop-signal paradigm (see Logan and Cowan, 1984). This modified version allows a measure of stop-signal reaction time (reactive control) and whether inhibition improves as the anticipation of a stop-signal increases (proactive control). The task was administered using Presentation® (NeuroBehavioral Systems, Albany, CA, USA), a stimulus delivery and experiment control program, on a Dell laptop with a 14′ by 7.5′ screen. The task took approximately 25 minutes to complete. Three horizontal lines displayed one above the other formed the background, which was presented to the participants throughout the task. Stimuli were presented in 3 blocks separated by two 24 s rest periods. All blocks consisted of STOP trials, which were pseudorandomly interspersed between GO trials. On every trial, a bar would rise towards the middle line at a constant duration (800 milliseconds). On a majority of trials (GO trials), participants were required to stop the bar as closely as possible to the middle line by pressing the spacebar. On a minority of trials (STOP trials), the rising bar would stop automatically before reaching the middle line. This spontaneous halt is the stop-signal and indicates that a response must be suppressed. Stop-signal likelihood was manipulated across trials and could be anticipated based on the colour of the middle line (green, 0%; yellow, 17%; amber, 20%; orange, 25%; red, 33%). See Fig. 1 for a visual depiction of the task.

The initial stop-signal onset time was set to 550 milliseconds (250 milliseconds before 800 millisecond target response time) for all stop-signal probability levels and was adjusted in real time for each participant to ensure an overall target accuracy level of 50%. If actual performance surpassed target performance on one of the trials, the stop-signal onset time was shifted 25 milliseconds towards the target response time to increase difficulty. Twenty-five milliseconds were subtracted from the stop-signal onset time if actual performance was lower than target performance. Thus, level of difficulty was kept constant to minimize differences in this variable across gamblers and controls to get a purer measure of difficulties in response inhibition. Prior to task

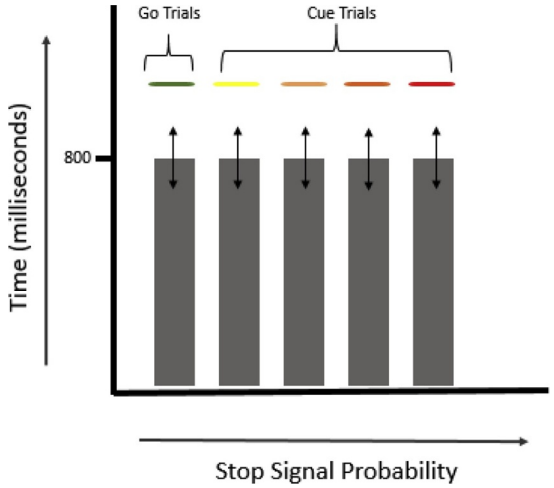

Fig. 1. Visual depiction of the stop signal anticipation task (SSAT). The task consists of 234 GO ONLY trials (green bar) and 180 GO/STOP cue trials (yellow, amber, orange, or red bar). During each trial, a bar moves at a constant speed from the bottom to the top in 800 milliseconds. On a majority of trials, participants are required to stop the bar as closely as possible to the colored bar. On a minority of trials, a stop-signal would occur (bar stops moving before reaching the color line); participants are instructed to inhibit their response when a stop-signal occurs. To measure proactive control, the stop signal probability is manipulated and varies based on the color of the response line: green (0%), yellow (17%), amber (20%), and red (33%). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

initiation, participants completed several practice blocks to familiarize themselves with the stimuli and eliminate practice effects.

2.4. Statistical analysis

SPSS for Windows, version 24.0, was used to conduct all statistical analyses. All tests were two-tailed with statistical significance set at $p < 0.05$. Differences in demographic characteristics were determined by independent sample *t*-tests for continuous variables (e.g., age, IQ), and chi-square tests for categorical variables (e.g., marital status, sex).

To examine the *first objective* of the study, the average SSRT for each individual was calculated using the integration method (Verbruggen and Logan, 2008). Research suggests that the integration method is less prone to overestimations of SSRT than the mean method (Verbruggen et al., 2013). To calculate SSRT using the integration method, the nth reaction time was selected, where n was obtained by multiplying the number of trials by the probability of responding in the stop trials. The mean SSD was subtracted from this value to obtain the SSRT.

Outliers were removed based on the method used in Zandbelt and Vink (2010). Specifically, reaction times on all GO trials throughout the task that exceeded 1.5 times the interquartile range away from the 25th and 75th percentiles of the reaction time distribution of each task condition (i.e., 4 stop-signal probability levels) were deemed as outliers. The overall mean SSRT obtained by gamblers and controls was compared using a one-way ANOVA.

To address the *second objective*, a mixed model ANOVA with GO trial condition (4 levels) as the within-subjects factor and diagnostic group (2 levels) as the between-subjects factor was conducted to test for differences in GO reaction time modulation as a function of stop-signal likelihood.

To address the *third objective* (whether specific UPPS-P personality domains predict reactive and proactive performance on the SSAT), a multiple regression analysis was conducted in each group, with reactive (SSRT) and proactive performance ([reaction time in response to red cue] – [reaction time in response to yellow cue]) on the SSAT as the dependent variables and the UPPS-P subscales as the predictor variables. Only one outlier on the perseverance domain of the UPPS-P was deemed an influential case (control participant, value = 2.90), and was removed from the analysis.

3. Results

3.1. Participant characteristics

The sample consisted of individuals with gambling disorder ($n = 27$) and community controls ($n = 21$). In the gambling group, 6 participants were identified as past gamblers (no gambling activity in the last 12 months). The severity of gambling disorder, as assessed by the DSM-5, was relatively similar in both past gamblers (2 mild, 2 moderate, 2 severe) and current gamblers (9 mild, 5 moderate, 7 severe). Age of onset for the gambling group was 30.14 ($SD = 13.50$). Demographic and clinical data, as well as statistical tests of mean differences between groups are reported in Table 1. The Welch-Satterthwaite method was applied to the following variables: HAM-D, PGSI, and UPPS-P perseverance and sensation seeking. There were no significant differences between groups in most demographic variables, with the exception that controls had higher years of mean education than gamblers, and more controls than gamblers were married. Further, there was a trend towards a higher frequency of smoking in the gambling group ($p = 0.06$). Moreover, as expected, individuals with gambling disorder scored significantly higher than controls on the HAM-D and PGSI, indicating both greater symptoms of depression in the previous week and more severe gambling problems, respectively. Of relevance, there was significant variability in the PGSI means in the gambling group (9.67 ± 7.32; score range: 0–25) and the overall mean severity level in past gamblers ($n = 6$) was low ($M = 3.00$). On the UPPS-P, there was a significant between-group difference on each subscale except sensation seeking (refer to Table 1). More specifically, individuals with gambling disorder displayed elevated scores on all subscales except negative urgency. No sex differences in either group was found, with the exception of higher scores on sensation seeking in male ($M = 2.89 \pm 0.47$) versus female ($M = 2.13 \pm 0.47$) gamblers ($t(25) = 4.01$, $p < 0.001$). Furthermore, comorbidities did not account for group differences on the UPPS-P. Finally, there were significantly higher rates of major depressive disorder ($X^2(2) = 7.63$, $p = 0.02$) and alcohol use disorder ($X^2(2) = 8.74$, $p = 0.01$) in individuals with gambling disorder than in controls (refer to Table 2).

3.2. Stop-signal anticipation task (SSAT)

Descriptive statistics for the SSAT are provided in Table 3. Two validity tests were conducted on the SSAT data to ensure participants understood the task and exerted more cognitive control during the GO ONLY trials. First, response times on GO ONLY trials (stop- signal probability of 0%) in both controls ($M = 807$ ms, $SD = 14.67$) and gamblers ($M = 810$ ms, $SD = 12.65$) were similar to the target response time of 800 ms, confirming participants understood the task and were able to perform the task accurately. Second, response times on the GO/STOP trials in both controls ($M = 825$ ms, $SD = 23.28$) and gamblers ($M = 824$ ms, $SD = 16.90$) were significantly slower than their respective response times on GO ONLY trials (control group: $t(20) = -6.63$, $p < 0.001$; gambling group: $t(26) = -6.82$, $p < 0.001$), demonstrating greater cognitive control was necessary for the GO/STOP trials.

A one-way ANOVA was conducted to test whether gamblers exhibited a slower SSRT on the SSAT, indicating impaired reactive control. However, contrary to the hypothesis, no significant difference in SSRT between groups ($F(1, 46) = 0.96$, $p = 0.33$, $\eta_p^2 = 0.02$) was found. A group (2 levels) X trial (4 levels) mixed model ANOVA was conducted to examine proactive control (hypothesis 2). There was a significant main effect of trial on reaction time ($F(3, 138) = 17.71$, $p < 0.001$) but no significant main effect of group ($F(1, 46) = 0.04$, $p = 0.85$). The trial X group interaction effect was not significant, suggesting no difference in proactive control performance between groups; Mauchly's Test of Sphericity was violated ($p < 0.05$), and thus, the Huynh–Feldt correction was applied to the *F*-statistic ($F(2.86, 131.68) = 0.10$, $p = 0.96$, $\varepsilon = 0.88$, $\eta_p^2 = 0.002$). Given, the difference in years of education between groups, the above-mentioned ANOVAs were also conducted with education added as a covariate; no changes in significance were observed on any of the findings.

3.3. Association between trait impulsivity and response inhibition

Two exploratory multiple regression analyses were conducted to identify whether trait impulsivity (UPPS-P domains) significantly predicted participants' reactive (SSRT) and proactive performance on the SSAT (refer to Table A.1 in Appendix). The overall regression models for reactive ($R^2 = 0.45$, $F(5, 19) = 2.27$, $p = 0.11$) and proactive control ($R^2 = 0.25$, $F(5, 19) = 0.91$, $p = 0.50$) were nonsignificant in controls. Similarly, the overall regression models conducted in the gambling group were nonsignificant for reactive ($R^2 = 0.06$, $F(5, 26) = 0.27$, $p = 0.93$) and proactive control ($R^2 = 0.23$, $F(5, 26) = 1.25$, $p = 0.32$). Interestingly, however, closer examination of beta weights showed premeditation was a significant predictor of performance in the gambling group ($b = -12.48$, $SE = 4.02$, $r(27) = -0.39$, $p = 0.03$), such that lower premeditation on the UPPS-P predicted worse proactive control.

Table 1
Demographic and Clinical Variables for Controls and Gamblers.

	CON (n = 21)	GD (n = 27)	Tests of group differences
Sex (% female)	48	36	$X^2(1) = 0.54$, $p = 0.46$
Mean age	46.19 ± 13.33	45.00 ± 15.31	$t(46) = 0.28$, $p = 0.78$
Mean years of education	16.05 ± 2.01	14.44 ± 2.12	$t(46) = 2.67$, $p = 0.01$
Marital status	Single (19%) Married (57%) Divorced (24%) Widowed (0%)	Single (48%) Married (19%) Divorced (26%) Widowed (7%)	$X^2(3) = 9.37$, $p = 0.03$
Mean number of children	1.14 ± 1.11	0.85 ± 1.20	$t(46) = 0.86$, $p = 0.39$
Work status	Employed (62%) Unemployed (14%) Retired (14%) Student (10%)	Employed (52%) Unemployed (15%) Retired (22%) Student (11%)	$X^2(3) = 0.64$, $p = 0.89$
Mean estimated annual salary ($)	62,190.48 ± 32,472.48	48,335.41 ± 35,526.11	$t(46) = 1.39$, $p = 0.17$
Smoking status	Current smoker (5%) Past smoker (14%) Non-smoker (81%)	Current smoker (22%) Past smoker (30%) Non-smoker (48%)	$X^2(2) = 5.72$, $p = 0.06$
Mean WTAR	103.24 ± 11.26	103.19 ± 12.01	$t(46) = 0.16$, $p = 0.98$
Mean HAM-D	1.19 ± 1.78	3.81 ± 5.39	$t(33) = -2.37$, $p = 0.04$
Mean PGSI	0.14 ± 0.48	9.67 ± 7.32	$t(26) = -6.74$, $p < 0.001$
BISQ			
Lifetime blow to head	Yes (67%) No (33%)	Yes (67%) No (33%)	–
Mean number of blows	2.14 ± 2.11	3.33 ± 2.35	$t(30) = -1.49$, $p = 0.15$
Lifetime loss of consciousness	Yes (33%) No (67%)	Yes (33%) No (67%)	–
Mean number of times unconscious	1.29 ± 0.76	2.22 ± 2.11	$t(14) = -1.11$, $p = 0.28$
Amount of time unconscious (most severe case)	*"Don't know"* (43%) *" < 1 minute"* (0%) *"1–10 mins."* (14%) *"11–20 mins."* (0%) *"21–30 mins."* (29%) *"1–23 hours"* (14%)	*"Don't know"* (11%) *" < 1 minute"* (22%) *"1–10 mins."* (44%) *"11–20 mins."* (11%) *"21–30 mins."* (0%) *"1–23 hours"* (11%)	$X^2(5) = 7.67$, $p = 0.18$
Mean UPPS-P scores			
Negative urgency	3.10 ± 0.49	2.46 ± 0.61	$t(46) = 3.92$, $p < 0.001$
(lack of) Premeditation	1.90 ± 0.31	2.26 ± 0.46	$t(46) = -3.06$, $p = 0.004$
(lack of) Perseverance	1.69 ± 0.32	2.04 ± 0.58	$t(42) = -2.66$, $p = 0.01$
Sensation seeking	2.48 ± 0.82	2.61 ± 0.59	$t(36) = -0.61$, $p = 0.55$
Positive urgency	1.77 ± 0.37	2.16 ± 0.51	$t(46) = -3.02$, $p = 0.004$

Note. CON, community controls; GD, individuals with gambling disorder; WTAR, Wechsler Test of Adult Reading; HAMD, Hamilton Depression Scale; PGSI, Problem Gambling Severity Index; BISQ, Brain Injury Screening Questionnaire.

Table 2
Comorbid mental health disorders for controls and gamblers.

Mental Health Disorder	CON (n = 21)	GD (n = 27)	Test statistic
Mood disorders, % total (n) [1]	29% (6)	56% (15)	$X^2(1) = 3.50$, $p = 0.062$
Substance use disorders, % total (n) [2]	19% (4)	52% (14)	$X^2(1) = 5.42$, $p = 0.020$
Anxiety disorders, % total (n) [3]	10% (2)	11% (3)	$X^2(1) = 0.32$, $p = 0.858$
Eating disorders, % total (n) [4]	5% (1)	4% (1)	$X^2(1) = 0.33$, $p = 0.856$
ADHD, % total (n)	0% (0)	7% (2)	$X^2(1) = 1.62$, $p = 0.203$
PTSD, % total (n)	0% (0)	11% (3)	$X^2(2) = 2.49$, $p = 0.288$

Note. CON, community controls; GD, individuals with gambling disorder;*.

[1] Mood disorders in the sample included major depressive disorder, persistent depressive disorder, substance induced depression, and premenstrual dysphoric disorder.

[2] Individuals with substance use disorder in the sample abused alcohol, cannabis, and cocaine; one person abused hallucinogens.

[3] Anxiety disorders in the sample consisted of specific phobias, panic disorder, social anxiety disorder, and agoraphobia.

[4] Eating disorders in the sample consisted of anorexia and bulimia nervosa. ADHD, Attention Deficit/Hyperactivity Disorder; PTSD, Posttraumatic Stress Disorder. The values in the chart represent the percentage of individuals in each respective group who met diagnostic criteria for the listed disorder. Values were rounded up to the nearest whole percent.

4. Discussion

Previous studies of response inhibition in gambling disorder have solely focused on reactive mechanisms of control. To further the literature, the current study sought to examine both reactive and proactive mechanisms of control using a variant of the standard stop-signal task.

4.1. Reactive and proactive control

The first aim of the present study was to add to the literature on reactive control. Current studies on reactive control in gambling disorder are both rare and have produced mixed findings. Some studies have found impaired reactive control in gamblers, indicated by slower SSRTs, while others have identified no significant differences. The findings from the present study were in accordance to past studies that

Table 3
Descriptive statistics for stop-signal anticipation task (SSAT).

Measure	CON (n = 21)	GD (n = 27)	Test Statistic
GO ONLY RT (mean % ± SD)	807 ± 14.67	811 ± 12.65	$t(46) = -0.88$, $p = 0.38$
GO/STOP RT			
SSP 17%	821 ± 26.29	820 ± 19.73	$t(46) = 0.18$, $p = 0.86$
SSP 20%	822 ± 25.95	822 ± 16.52	$t(46) = 0.04$, $p = 0.97$
SSP 25%	827 ± 22.38	825 ± 16.12	$t(46) = 0.31$, $p = 0.76$
SSP 33%	831 ± 21.00	829 ± 19.96	$t(46) = 0.22$, $p = 0.82$
Overall	825 ± 23.28	824 ± 16.90	$t(46) = 0.20$, $p = 0.84$
SSD average (ms)	568 ± 29.63	566 ± 24.98	$t(46) = 0.25$, $p = 0.81$
SSRT (mean % ± SD)			
Block 1	250 ± 20.32	256 ± 20.81	$t(46) = -1.02$, $p = 0.31$
Block 2	246 ± 22.74	247 ± 23.63	$t(46) = -0.06$, $p = 0.95$
Block 3	240 ± 22.88	248 ± 23.79	$t(46) = -1.18$, $p = 0.25$
Overall	245 ± 19.63	251 ± 19.88	$t(46) = -0.98$, $p = 0.33$
Accuracy: (mean % ± SD)			
SSP 17%	44 ± 13.48	51 ± 14.34	$t(46) = -1.71$, $p = 0.10$
SSP 20%	50 ± 8.39	50 ± 7.93	$t(46) = 0.15$, $p = 0.89$
SSP 25%	51 ± 4.78	51 ± 5.25	$t(46) = 0.13$, $p = 0.90$
SSP 33%	51 ± 3.71	50 ± 4.59	$t(46) = 0.23$, $p = 0.82$
Overall	49 ± 4.21	51 ± 5.87	$t(46) = -1.03$, $p = 0.31$
Stop Failure RT (milliseconds)	799 ± 26.42	801 ± 25.73	$t(46) = -0.31$, $p = 0.76$

Note. *. CON, community controls; GD, individuals with gambling disorder; SD, standard deviation; SSD, stop signal delay; SSRT, stop signal reaction time; SSP, stop signal probability; RT, reaction time.

have failed to find impaired reactive control in gambling disorder (de Ruiter et al., 2012; Lawrence et al., 2009b; Leppink et al., 2016; Lipszyc and Schachar, 2010; Lorains et al., 2014). SSRT values reported in studies of healthy controls on the standard stop-signal paradigm are usually between 200 and 250 milliseconds (Zandbelt and Vink, 2010); all participants' SSRTs in the current study fell within this normal range.

One implication of this finding may be that poor reactive control is not a core deficit in gambling disorder. In fact, some studies suggest impaired pre-potent motor inhibition may not be a central aspect of impulsivity in gambling disorder (e.g., Leppink et al., 2016). Nonetheless, these findings should be interpreted with caution, given the modest sample size of the study. For instance, the sample size did not allow separate analyses of gamblers with and without alcohol use disorder. Lawrence et al., (2009b) found SSRT was slower in alcohol-dependent individuals compared to controls, whereas no differences were observed between controls and problem gamblers. Therefore, one plausible explanation for intact stop-signal performance in some groups of gamblers could be the absence of the neurotoxic effects of alcohol and drug use on brain structure and chemistry, particularly in the prefrontal cortex (Potenza, 2006; Clark, 2014). Indeed, approximately half of the gamblers in the present study did not meet diagnostic criteria for any substance use disorder, and in turn, may have attenuated the SSAT outcomes. On a similar note, only two individuals in the gambling group had an ADHD diagnosis, which has consistently been shown to impair performance on the stop-signal task in children (Senderecka et al., 2012) and in adults (Lijffijt et al., 2005; Verbruggen and Logan 2008). Taken together, it is conceivable that reactive and/or proactive control are only impaired in a selection of gamblers (e.g., gamblers with co-occurring ADHD and substance use disorders). This notion is in line with the pathways model (see Blaszczynski and Nower, 2002), which suggests some subsets of gamblers (i.e. "the behaviorally conditioned gambler") endorse low levels of impulsivity. This finding also emphasizes that individuals with gambling disorder do not constitute a homogenous population.

The second aim of this study was to compare proactive control in individuals with gambling disorder and controls. Contrary to our hypothesis that gamblers would display impaired proactive control, we found no significant differences between the two groups. Further, it is important to note that although gamblers in the current study exhibited intact reactive and proactive control, they were significantly elevated on most domains of trait impulsivity, providing support for the idea that (1) high scores on self-report measures of impulsivity are not directly indicative of impaired motor control and (2) self-report and cognitive measures of impulsivity may measure different aspects of this behavior (Moeller et al., 2001). Notwithstanding, these findings should be interpreted with caution given the lack of comparative research on proactive control in the gambling literature.

4.2. The association between trait impulsivity and response inhibition

Another aim of this study was to determine the predictive capacity of the five impulsivity facets of the UPPS-P on reactive and proactive control. Group differences were observed on all domains of the UPPS-P, except sensation seeking. Consistent with previous research, gamblers in the current study scored higher on the premeditation, perseverance, and positive urgency subscales (Savvidou et al., 2017). Contrastingly, controls displayed higher levels of negative urgency than gamblers. This latter finding is paradoxical, as studies have consistently reported heightened levels of negative urgency in gambling disorder (see meta-analysis by MacLaren et al., 2011). The sample characteristics of the current study (e.g., comorbidities in control sample, past gamblers in gambling group) may, in part, explain this result. Other possibilities for this finding may include response bias and/or the unusual lack of current depressive symptoms observed in the gambling sample.

In line with past studies, no relationship between reactive control and trait impulsivity was observed in the gambling group (Lawrence et al., 2009b; Lorains et al., 2014). Regarding proactive control, although neither of the overall regressions indicated a relationship with trait impulsivity, a significant association between (lack of) premeditation and proactive control was found in the gambling group.

The unique association found between premeditation and proactive control fits well with the conceptual overlap of these two constructs, as both require some level of anticipation (either of a consequence or an interfering stimulus in the environment, respectively). Lack of premeditation has been strongly linked to gambling behavior (Blain et al., 2014; Michalczuk et al., 2011; Whiteside et al., 2001), higher gambling severity (Haw, 2015), co-occurring mood disorders (Lister et al., 2015), and illicit substance use (Mitchell and Potenza, 2014). Based on the data from the current study, it seems that lack of premeditation is also significantly associated with lower levels of proactive control in gambling disorder. That is, gamblers who self-report difficulty planning out

actions and anticipating consequences show a lower capacity to use informative contextual cues to prepare for inhibition (i.e. slowing responses for greater likelihood of a successful stop).

As such, impairments in proactive control may be uniquely related to certain impulsive traits in individuals with gambling disorder. This finding not only adds to the understanding of the cognitive correlates of trait impulsivity, but also has practical relevance for treatment development. For example, the UPPS-P may possess utility as a screener used in clinical settings to develop more accurate profiles of treatment seeking gamblers. From the current findings, one might speculate that a gambler elevated on the premeditation subscale of the UPPS-P may benefit from cognitive remediation as part of their care (e.g., response inhibition training; see Verbruggen et al., 2012). Despite an apparent association between premeditation and proactive control, directionality and causation cannot be inferred from the current findings.

4.3. Limitations and future directions

A number of limitations are acknowledged in this study. First, the sample size of the current study was modest, reducing the power of the statistical analyses. Smaller samples sizes may attenuate differences in SSRT between participants (Lienissen et al., 2017). Therefore, the findings from this research should be considered preliminary and are subject to replication. Second, the sample size of the gambling group did not permit examination of the outcome variables with gamblers parsed based on preferred mode of gambling (e.g., strategic versus non-strategic), current gambling status, or comorbid conditions. Considering the heterogeneity of the gambling disorder population, we recommend that future studies account for these within-group differences. Third, the SSAT may be argued to lack a sufficient level of reward/punishment saliency to appropriately map onto gambling behavior. Thus, prospective studies using stop-signal tasks with greater ecological validity may be superior in capturing the true essence of response inhibition deficits in gambling disorder.

Finally, different variants of the stop-signal task should be used in future studies to investigate reactive and proactive control in gambling disorder. For example, the standard stop-signal task with simpler or more complex manipulations can be implemented to measure proactive control. Furthermore, some studies have used separate tasks biased towards either reactive or proactive control to measure each concept separately (e.g., see Lesh et al., 2013). By the same token, as the 'dual mechanisms of control' theory continues to develop, it is important to identify a smaller subset of "gold standard" or optimal tasks to evaluate reactive and proactive control. The heterogeneity of features across stop-signal tasks in the literature makes it difficult to compare results across studies and needs to be addressed in future works.

4.4. Conclusion

The present study provides new information regarding the mechanisms of response inhibition in individuals diagnosed with gambling disorder. No group differences in reactive and proactive control were detected in this study, suggesting impaired response inhibition may not be a central feature of all individuals with gambling disorder. Furthermore, there was preliminary evidence to suggest low levels of premeditation are associated with worse proactive control in gamblers, which supports a large body of studies that have reported unique relationships between self-reported and behavioral measures of impulsivity. To determine which aspects of impulse control should be targeted in treatment, studies should continue to decompose impulsivity into lower-order components.

Declarations of interest

none (applies to all three authors)

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Supplementary materials

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