

## Relapse in pathological gamblers: A pilot study on the predictive value of different impulsivity measures

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*Backgrounds and aims:* Pathological gambling, a common psychiatric disorder, has many similarities with substance use disorders. Relapse, an important element in addictive disorders, however, has seldom been studied in pathological gambling. Hence, in analogy with previous research studies examining the role of self-report and neurocognitive measures on relapse in substance dependent patients, the present pilot study was executed. *Methods:* Twenty-two pathological gamblers and 31 healthy controls took part in this research. They filled in self-report questionnaires measuring impulsive personality (Barratt Impulsiveness Scale, Sensitivity to Punishment and Sensitivity to Reward Questionnaires) and performed neurocognitive tasks measuring impulsivity, decision-making and attentional bias (Iowa Gambling Task, Delay Discounting Task, Stroop Gambling Task). Twelve months later gambling activity was re-examined. *Results:* Analyses showed that PGs who relapsed ( $n = 13$ ) did not differ on self-report and neurocognitive measures of impulsivity with PGs who did not relapse ( $n = 9$ ). However, both groups did differ in age at onset. Finally, healthy controls and PGs differed in some (Barratt Impulsiveness Scale, Stroop Gambling Task), but not all impulsivity measures (Delay Discounting Task, Iowa Gambling Task, Sensitivity to Punishment and Sensitivity to Reward Questionnaires). *Conclusions:* One-year relapse in pathological gamblers is not predicted by self-report and or neurocognitive measures of impulsivity and decision-making. The similarities in performances between pathological gamblers and healthy controls illustrate the relative health of the examined pathological gamblers. This last finding supports the idea that subtypes of pathological gamblers exist so that different treatment strategies might be necessary.

**Keywords:** pathological gamblers, impulsivity, relapse

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### INTRODUCTION

With lifetime prevalence between 0.8% and 1.6% in the adult population pathological gambling (PG) is a relatively common psychiatric disorder that is associated with severe socio-legal problems and frequent comorbidity with other psychiatric disorders (Ledgerwood & Petry, 2006a). In spite of these high prevalence rates and severe consequences, few studies have explored the processes that contribute to the continuation and relapse of pathological gambling.

Pathological gambling is currently categorized in the Diagnostic and statistical manual (DSM-IV text revision, American Psychiatric Association, 2000) as an impulse control disorder, and thus hypothesized to lie among an impulse–compulsive spectrum, also representing obsessive–compulsive spectrum disorders (e.g., Brewer & Potenza, 2008). Although individuals with impulse control disorders engage in repetitive behaviors with great urges, these behaviors are egosyntonic (e.g., Andrade & Petry, 2012; Brewer & Potenza, 2008), whereas repetitive behaviors or rituals in obsessive–compulsive disorders are generally egodystonic (e.g., Brewer & Potenza, 2008). Furthermore, on a phenotypical and pathological level there are striking similarities with substance use disorders (SUD), even though there is no administration of an exogenous substance to

cause harmful effects in the brain (e.g., Potenza, 2001). Impairments in self-regulatory behavior and underlying brain processes for instance are hypothesized to be central in the development and maintenance of both pathological gambling and SUD (Alvarez-Moya et al., 2010; Koob and Volkow, 2010; Leeman & Potenza, 2012). These and other similarities have given ground for the suggestion that gambling disorders should be reclassified within the upcoming DSM-V within the category substance use and addictive disorders (American Psychiatric Association, 2012, www.DSM5.org).

Relapse is a central phenomenon characterizing these disorders. Recent research findings in substance dependent patients have shown that impairments in neurocognitive self-regulatory processes are associated with an individual's vulnerability to relapse and can differentiate between those patients who do relapse and those who remain abstinent after treatment. Specifically, tasks measuring risk/reward decision-making like the Iowa Gambling Task (IGT, Bechara,

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Damasio, Damasio & Anderson, 1994) have proven promising within this context (e.g., Bowden-Jones, McPhillips, Rogers, Hutton & Joyce, 2005; De Wilde, Sabbe, Hulstijn & Dom, 2013; Passeti, Clark, Mehta, Joyce & King, 2008). Since it has been suggested that these neurocognitive performance deficits are reflective of central underlying neurobiological vulnerabilities or changes (e.g., Verdejo-Garcia, Lawrence & Clark, 2008), it is remarkable that, contrary to chemical addictions, studies exploring neurocognitive performance on impulsivity and decision-making and their association with treatment outcome or relapse within pathological gamblers (PGs) have been limited until now. Álvarez-Moya et al. (2011) recently explored possible associations between decision-making (Iowa Gambling Task, ABCD & EFGH versions) and self-reported impulsivity (Temperament and Character Inventory–Revised) on the one hand and treatment outcome on the other. Goudriaan, Oosterlaan, de Beurs and van den Brink (2008) examined the role of self-reported (impulsivity–reward sensitivity) versus neurocognitive (disinhibition [Stop Signal Reaction Time] – decision-making [Card Playing Task]) measures in the prediction of one-year relapse in PGs. Both research groups concluded that self-reported measures and neurocognitive measures of cognitive flexibility/involuntary attention (Stroop) did not affect outcome measures (Álvarez-Moya et al., 2011: relapse during treatment; Goudriaan et al., 2008: one-year relapse). Neurocognitive indicators of disinhibition together with longer duration of the disorder predicted one-year relapse (Goudriaan et al., 2008). Poor decision-making finally predicted dropout (Álvarez-Moya et al., 2011) and one-year relapse (Goudriaan et al., 2008) but not relapse during treatment (Álvarez-Moya et al., 2011).

Reasons for these inconsistent findings may be numerous. First, the processes controlling vulnerability to relapse may be different during treatment (Álvarez-Moya et al., 2011) and at follow-up one year after treatment (Goudriaan et al., 2008). Second, the nature of the examined neurocognitive measures assessing decision-making might be different. Third, the heterogeneity of the PGs group might have affected research findings. Indeed, PG is frequently associated with other Axis I and II psychiatric comorbid disorders. Substance use disorders (SUD) and personality disorders (PD) are often associated with pathological gambling (e.g., Petry, 2006; Wareham & Potenza, 2010). Of importance, earlier studies indicate that these disorders in themselves are associated with changes in impulsivity measures (and underlying neurobiological processes). Thus, comorbidity within PGs may confound both relapse risk, the changes found on the neurocognitive level, and their association.

We hence decided to do a pilot study examining the role of different self-report and neurocognitive measures on one-year relapse of pathological pathological gamblers. We focused upon the Iowa Gambling Task (IGT, Bechara et al., 1994), a measure known to predict relapse in substance dependent patients (e.g. Bowden-Jones et al., 2005; De Wilde et al., 2013; Passeti et al., 2008) and included the Delay Discounting Task (DDT, Richards, Zhang, Mitchell & de Wit, 1999), a measure related to immediate reward. Both neurocognitive measures were found to differentiate between PGs and healthy controls (e.g., Petry, 2001). We finally included a Stroop Gambling Task, measuring attentional bias specifically for gambling stimuli. All neurocognitive measures together with self-report measures of impulsivity were completed by a small group of PGs without

manifest other psychiatric disorders, enrolling in a longitudinal (12-month follow-up) outcome study. In line of the earlier research in substance use patients and PGs, we hypothesized that performance deficits on neurocognitive impulsivity measures but not impulsivity on self-report measures of impulsivity would relate to an increase in relapse risk.

## MATERIAL AND METHODS

### *Participants*

Twenty-two outpatient lifetime pathological gamblers (PGs, slot players) and 31 healthy controls (HCs) participated in this pilot study. The principal researchers (BDW and GD) informed regional addiction counselors and chairmen of two self-help groups about the present research (mainly about the hypothesis and procedure). They asked them to transmit this information to their patients and to motivate them to participate. Contact data of interested pathological gamblers were then given to the principal researchers who then got in touch with the patients. Seventeen patients were found through local addiction counselors and thus in active treatment, five patients through self-help groups. Two of them were in full remission when they signed the informed consent. They all were slot machine players, frequenting bars and casinos. The HCs responded to an ad in a local newspaper. Participants were excluded if they demonstrated signs of lifetime substance use disorders (with the exception of caffeine or nicotine abuse or dependence,  $n = 1$ ; Structured Clinical Interview for the DSM-IV disorders, axis I disorders; First, Spitzer, Gibbon & Williams, 1996), psychotic disorders ( $n = 0$ ; Structured Clinical Interview for the DSM-IV disorders, axis I disorders; First et al., 1996), organic deterioration or amnesic disorders ( $n = 0$ ; Structured Clinical Interview for the DSM-IV disorders, axis I disorders; First et al., 1996), physical handicaps ( $n = 0$ ; medical examination), severe somatic disorders ( $n = 0$ ; medical examination) or illiteracy ( $n = 0$ ; Revised National Adult Reading Test; Nelson & Willison, 1991). Healthy controls were excluded when they showed signs of pathological gambling ( $n = 0$ ; South Oaks Gambling Screen; Lesieur & Blume, 1987).

### *Measures*

*Gambling.* Local addiction counselors and researchers (BDW) used the Structured Clinical Interview for the DSM-IV disorders, Axis I disorders (SCID-I; First et al., 1996) and the South Oaks Gambling Screen (SOGS, Lesieur & Blume, 1987) to identify pathological gamblers. The SCID-I is a semi-structured interview for making the major DSM-IV Axis I diagnoses (First et al., 1996). The SOGS (Lesieur & Blume, 1987) is a questionnaire containing twenty questions examining lifetime gambling behavior. Participants scoring five or more are generally seen as ‘probable pathological gamblers’.

*Substance use disorders.* The CAGE (Ewing, 1984) and the Drug Abuse Screening Test (DAST-10) (Skinner, 1982) were used to detect substance use disorders. The CAGE (Ewing, 1984) is a short four-question screening instrument for lifetime alcoholism. The DAST-10 (Skinner, 1982) is a self-report questionnaire that holds ten questions concerning information about patients’ potential involvement with

drugs excluding alcohol and tobacco during the past year. Participants were included if none of the questions on the CAGE (alcohol use disorders) and the DAST-10 (substance use disorders) led to a positive answer.

*Self-report measures of impulsive personality.* The Barratt Impulsiveness Scale (BIS, Patton, Stanford & Barratt, 1995), a self-report questionnaire (30 items), measured total, attentional, motor, and non-planning facets of trait impulsivity. The Sensitivity to Punishment and Sensitivity to Reward Questionnaires (SPSRQ, Torrubia, Ávila, Moltó & Caseras, 2001) measured personality traits associated with the behavioral activation or appetitive system (sensitivity to reward) and the behavioral inhibition system (sensitivity to punishment).

*Cognitive measures of impulsivity and decision-making.* Delay Discounting Task (DDT—computerized version, Richards et al., 1999). Participants answered 100 questions, such as the following: ‘Would you rather have 10€ in 30 days or 2€ now?’ A random adjusting amount procedure was used, so that the amount of immediate money was adjusted across trials until reaching an amount equivalent to a delayed reward, as determined by the participant’s choice. These indifference points were determined for all reward values (10€, 30€, 100€) and delays (2, 30, 180, 360, 720 days). Outcome measures were the mean logarithms of these delays for  $k$  at 10€, 30€, and 100€.

*Iowa Gambling Task.* The Iowa Gambling Task (IGT, Bechara et al., 1994) required participants to choose 100 cards from four card decks (A’, B’, C’, D’). Each deck held sixty cards with identical backs. Participants were instructed to select cards to earn as much money as possible. Unknown to them, card selections came with different pay-offs: good decks (C’, D’) combined modest wins with small losses (net gains), while bad decks (A’, B’) combined large wins with even larger losses (net losses). Good decks gave net gains while bad decks led to net losses. Outcome measures were the mathematical differences between the number of cards picked from the advantageous decks and the number of cards picked from the disadvantageous decks, calculated for blocks of twenty cards. The net score is the total sum of all blocks.

*Stroop Task.* Finally, a computerized version of the Stroop Color Word Task (SCWT) was used. The SCWT consisted of eight blocks of 48 words from different categories (GAMBL: words related to gambling – NEUTR: neutral words). All words were printed in one of the following colors: yellow, red, green, or blue. Participants were asked to look at the word’s color and to push the button with the same color as the word. Outcome measures included response time and number of errors.

*Personality disorders.* The Assessment of DSM-IV Personality disorders (ADP-IV, Schotte & De Doncker, 1994, 1996) is a self-report questionnaire comprising 94 phrases. Each phrase represents a DSM-IV axis-II criterion. Each phrase is measured on a seven-point Likert scale to form a trait score (How much do you agree with this statement about yourself? Answers: 1 = totally disagree; 2 = disagree; 3 = tend to disagree; 4 = agree nor disagree; 5 = tend to agree; 6 = agree; 7 = fully agree). Typical criteria (Trait score  $\geq 5$ ) are further judged on a three-point Distress scale (Has this trait ever caused you or others any suffering or problems? Answers: 1 = not at all; 2 = to a degree; 3 = definitely). The T<sup>5</sup> (Trait score  $\geq 5$ ) and D<sup>1</sup> (Distress score  $\geq 1$ ) categorical diagnostic evaluation algorithm was used to state the presence of DSM-IV axis-II criteria. The diagnoses were made based on the presence of the DSM-IV criteria.

*Additional measures.* The Revised National Adult Reading Test (NART, Nelson & Willison, 1991) and the Raven Progressive Matrices (Raven PM, Raven, 1936) were used to assess participants’ intelligence. The first test is a reading test: patients read fifty words at loud and get points for correct pronunciation. The total score stands for participants’ premorbid intelligence. The second test is a nonverbal test made of sixty multiple choice questions, listed in order of difficulty. It is designed to measure current reasoning ability (general intelligence).

### Procedure

Pathological gamblers and HCs were seen over two appointments. During the first appointment, participants were asked about their substance use and gambling behavior. Additionally, we administered the NART and the Raven PM (intelligence) during the first session. A week later, at the second appointment, we administered tests to obtain information on neurocognitive measures of decision-making and impulsivity (DDT, IGT, SCWT). Additionally, participants returned the completed self-report questionnaires on impulsivity and personality disorders (ADP-IV, BIS, SPSRQ). One year after this last appointment, PGs were questioned about their gambling activities over the past year. Relapse was defined as the presence of any gambling behavior (Ledgerwood & Petry, 2006b) and coded as a binary variable (abstinent/non-abstinent). Participants were asked questions from the SCID-I and the SOGS to determine abstinence.

All participants gave written informed consent prior to study entrance. The research protocol was approved by the Ethical Committee of the Antwerp University.

### Design and statistical analyses

Differences in demographic, personality and additional variables were analysed by means of  $\chi^2$ -tests (gender) or univariate analyses of variance (other variables). *T*-tests were used to examine differences in pathological gambling. Multivariate analyses were used to measure differences in impulsive personality between groups. GLM repeated measures (DDT, IGT, SCWT) were used to examine differences in neurocognitive measures of decision-making and impulsivity. Helmert contrasts were used to clarify possible differences between the HCs and the PGs and later on between the abstinent and non-abstinent PGs. Estimates of effect sizes were added to the tables.

## RESULTS

### Demographic and addiction variables

When they signed the informed consent, there were no differences in demographic variables and or addiction variables between the pathological gamblers in formal treatment ( $n = 17$ ) and those in self-help ( $n = 5$ ). The PGs in the first group were as old as the PGs in the latter group ( $t(20) = -0.291, p = 0.774$ ). Age of onset respectively was  $18.47 \pm 5.90$  and  $23.40 \pm 13.32$  years ( $t(20) = -1.218, p = 0.237$ ), meaning that they had been gambling for  $13.18 \pm 9.95$  and  $10.00 \pm 6.93$  years ( $t(20) = 0.662, p = 0.515$ ). There also was no difference in gambling severity as assessed by the SOGS ( $t(20) = -0.286, p = 0.778$ ).

Table 1. Demographic, personality, addiction and additional variables

	Data			Group effects		Group contrasts	
	HCs (N = 31)	APGs (N = 9)	NAPGs (N = 13)	T, F or $\chi^2$	P	HCs vs. PGs	APGs vs. NAPGs
Gender	27♂ – 4♀	8♂ – 1♀	12♂ – 1♀	0.25	0.883		
Age	28.06 ± 7.79	37.00 ± 10.98	31.08 ± 7.66	4.06	0.023	0.014	0.108
NART	107.13 ± 7.62	108.83 ± 8.68	104.00 ± 6.57	1.00	0.378	0.762	0.213
Cluster A presence	0 present	0 present	0 present				
Cluster B presence	3 present	1 present	3 present	3.08	0.214		
Cluster C presence	2 present	0 present	2 present	2.99	0.224		
Age of onset		24.22 ± 10.92	16.38 ± 2.57	2.52	0.021		
Duration		9.22 ± 8.00	14.69 ± 9.77	0.29	0.773		
SOGS		11.44 ± 4.39	10.92 ± 3.93	1.39	0.181		

APGs: abstinent pathological gamblers; HCs: Healthy controls; NAPGs: non-abstinent pathological gamblers; NART: Revised National Adult Reading Test; PGs: pathological gamblers; SOGS: South-Oaks Gambling Screen. The cluster A, B & C presences were assessed by means of the ADP-IV (The Assessment of DSM-IV Personality Disorders questionnaire). Group contrast was only mentioned when group effects were significant.

Table 2. Correlation matrix impulsivity measures at follow-up, nine PGs were abstinent, thirteen PGs were relapsed

Part A: Healthy controls (N = 31)

	Age	SOGS	BIS_Att	BIS_Mot	BIS_NP	BIS_Tot	SPSRQ_SP	SPSRQ_SR	DDT_logk10	DDT_logk30	DDT_logk100	IGT_Netscore	SCWT_RT_N	SCWT_RT_G
Age	1	-0.12	-0.10	0.02	-0.08	-0.09	0.07	-0.36	0.28	0.26	0.17	-0.11	-0.03	-0.25
SOGS		1	-0.19	-0.04	-0.25	-0.21	-0.14	0.19	0.13	0.28	0.10	-0.24	0.25	0.27
BIS_Att			1	0.25	<b>0.55**</b>	<b>0.78**</b>	<b>0.51*</b>	0.07	-0.12	0.16	-0.23	-0.03	<b>-0.47*</b>	<b>-0.54**</b>
BIS_Mot				1	<b>0.64**</b>	<b>0.74**</b>	0.14	0.24	0.13	<b>0.41*</b>	0.36	0.17	0.08	-0.01
BIS_NP					1	<b>0.91**</b>	0.30	0.09	0.01	0.11	0.15	0.22	-0.03	-0.11
BIS_Tot						1	<b>0.42*</b>	0.17	-0.01	0.25	0.09	0.15	-0.19	-0.28
SPSRQ_SP							1	0.10	-0.20	0.15	-0.01	-0.00	-0.27	<b>-0.41*</b>
SPSRQ_SR								1	0.15	0.27	0.19	0.26	-0.07	0.11
DDT_logk10									1	<b>0.51**</b>	<b>0.64**</b>	0.07	0.10	0.12
DDT_logk30										1	<b>0.50**</b>	-0.20	-0.20	-0.18
DDT_logk100											1	<b>0.40*</b>	0.17	0.13
IGT_Netscore												1	0.01	0.15
SCWT_RT_N													1	<b>0.77**</b>
SCWT_RT_G														1

Part B: Pathological gamblers (N = 22)

	Age	Age of onset	Duration	SOGS	BIS_Att	BIS_Mot	BIS_NP	BIS_Tot	SPSRQ_SP	SPSRQ_SR	DDT_logk10	DDT_logk30	DDT_logk100	IGT_Netscore	SCWT_RT_N	SCWT_RT_G
Age	1	0.39	<b>0.52*</b>	-0.14	-0.01	-0.21	-0.22	-0.19	-0.20	-0.45	0.24	0.32	0.28	-0.17	<b>0.53*</b>	<b>0.54*</b>
Age of onset		1	<b>-0.45*</b>	-0.19	<b>-0.48*</b>	-0.44	-0.40	<b>-0.54*</b>	-0.18	-0.37	0.36	<b>0.55*</b>	0.41	-0.12	0.03	-0.00
Duration			1	0.16	0.21	0.17	0.17	0.22	-0.00	-0.02	0.04	-0.07	0.01	-0.11	0.37	0.36
SOGS				1	0.46	0.38	0.34	0.31	0.21	<b>0.53*</b>	0.04	0.19	0.12	0.20	0.29	0.30
BIS_Att					1	0.35	<b>0.58*</b>	<b>0.78**</b>	-0.06	0.09	-0.35	-0.29	-0.32	-0.06	0.05	0.08
BIS_Mot						1	<b>0.57*</b>	<b>0.79**</b>	-0.10	0.37	0.06	0.03	-0.01	-0.20	0.09	0.14
BIS_NP							1	<b>0.88**</b>	0.04	0.14	-0.24	-0.12	-0.24	-0.16	0.16	0.24
BIS_Tot								1	-0.06	0.26	-0.20	-0.15	-0.22	-0.17	0.12	0.19
SPSRQ_SP									1	0.34	-0.02	0.03	0.12	0.39	0.42	0.36
SPSRQ_SR										1	-0.04	-0.26	-0.26	0.26	0.16	0.07
DDT_logk10											1	<b>0.81**</b>	<b>0.91**</b>	-0.34	0.01	-0.01
DDT_logk30												1	<b>0.95**</b>	-0.31	0.16	0.14
DDT_logk100													1	-0.34	0.15	0.10
IGT_Netscore														1	0.14	0.16
SCWT_RT_N															1	<b>0.97**</b>
SCWT_RT_G																1

\* $p < 0.05$ ; \*\* $p < 0.005$ . BIS: Barratt Impulsiveness Scale; BIS\_Att: BIS Attentional; BIS\_Mot: Bis Motor; BIS\_NP: BIS Non-Planning; BIS\_Tot: BIS Total; SPSRQ: Sensitivity to Punishment and Sensitivity to Reward; DDT: Delay Discounting Task; IGT: Iowa Gambling Task; SCWT\_RT\_G: Stroop Color Word Test\_Reaction Time\_Gambling words; SCWT\_RT\_N: Stroop Color Word Test\_Reaction Time\_Neutral words; SPSRQ\_SP: SPSRQ\_Sensitivity to Punishment; SPSRQ\_SR: SPSRQ\_Sensitivity to Reward.

At follow-up, nine PGs were abstinent, thirteen PGs were relapsed. The HCs ( $n = 31$ ), abstinent ( $n = 9$ ) and non-abstinent ( $n = 13$ ) PGs groups did not differ in gender ( $\chi^2(2) = 0.248, p = 0.883$ ). They did differ in age ( $F(2, 52) = 4.06, p = 0.023$ ). Both abstinent and non-abstinent PGs groups indeed were older than the HC group (see Table 1). As seen in Table 2, age was correlated with self-report measures of impulsivity. We used this variable as a covariant in the analyses. Results were not affected hereby.

Abstinent and non-abstinent PGs groups did not differ in demographic variables (gender–age). They did differ in gambling history: PGs who were abstinent at follow up were older when they started to gamble than PGs who relapsed ( $t(20) = 2.515, p = 0.021$ ). Both groups, however, did not differ in gambling involvement ( $t(20) = 0.292, p = 0.773$ ) and severity ( $t(20) = -1.386, p = 0.181$ ) (Table 1). As seen in Table 2, gambling involvement affected neurocognitive measures of impulsivity. We used this variable as a covariant in the analyses. Results were not affected hereby.

*Impulsivity measures*

*Self-report questionnaires.* As can be seen in Table 3, both PGs groups were more impulsive than the HCs on the BIS (Total, Motor and Non Planning subscales) but not on the BIS Attentional subscale and the SPSRQ. There thus were

no differences in impulsivity self-report questionnaires for PGs who remained abstinent and PGs who were non-abstinent at follow-up.

*Neurocognitive measures.* Decision-making. GLM repeated measures analyses of variance with Block (Block 1 to 5) as within subjects factor and Group (HCs vs. PGs – abstinent PGs vs. non-abstinent PGs) as between-subjects factor showed that HCs and PGs on the one hand and abstinent and non-abstinent PGs on the other did not differ in IGT performances ( $F(2, 50) = 1.12, p = .335$  – effect size: 0.043). IGT performances changed over Blocks ( $F(4, 48) = 6.183; p < 0.001$ ). There was no significant Block\*Group interaction effect ( $F(8, 48) = 1.206; p = 0.297$ ) (Figure 1).

GLM repeated measures analyses of variance with amount (10\$, 30\$, 100\$) as within-subjects factor and group (HCs vs. PGs – abstinent PGs vs. non-abstinent PGs) as between-subjects factors showed that HCs and PGs on the one hand and abstinent and non-abstinent PGs on the other did not differ in DDT ( $F(2, 46) = 1.57, p = .219$  – effect size: 0.064). There was a significant amount ( $F(2, 46) = 13.082; p < 0.001$ ) but no amount\*group interaction effect ( $F(4, 44) = 0.807; p = 0.524$ ) (Table 4).

*Impulsivity.* As shown in Table 4, both PGs groups were as slow and significantly slower on the SCWT than HCs. Word class did not affect reaction times.

Table 3. Impulsivity as measured by self-report questionnaires

	Data			Group effects		Group contrasts	
	HCs (N = 31)	APGs (N = 9)	NAPGs (N = 13)	F	P	HCs vs. PGs	APGs vs. NAPGs
BIS_Tot	54.13 ± 9.04	68.67 ± 11.09	70.91 ± 9.85	16.34	< 0.001	< 0.001	0.606
Bis_Att	14.83 ± 3.77	17.89 ± 5.47	17.27 ± 2.65	2.95	0.062		
Bis_Mot	18.63 ± 2.93	24.22 ± 4.44	24.36 ± 4.43	15.02	< 0.001	< 0.001	0.930
BIS_NP	20.73 ± 4.29	26.56 ± 3.81	29.27 ± 4.17	19.36	< 0.001	< 0.001	0.155
SPSRQ_SP	11.25 ± 5.90	7.00 ± 5.00	9.44 ± 6.06	1.91	0.161		
SPSRQ_SR	10.36 ± 3.74	10.78 ± 6.92	10.00 ± 3.39	0.07	0.934		

APGs: abstinent pathological gamblers; BIS: Barratt Impulsiveness Scale; BIS\_Att: BIS Attentional; BIS\_Mot: Bis Motor; BIS\_NP: BIS Non Planning; BIS\_Tot: BIS Total; HCs: Healthy controls; NAPGs: non abstinent pathological gamblers; PGs: pathological gamblers; SPSRQ: Sensitivity to Punishment and Sensitivity to Reward; SPSRQ\_SP: SPSRQ\_Sensitivity to Punishment; SPSRQ\_SR: SPSRQ\_Sensitivity to Reward. Group contrast was only mentioned when group effects were significant.

Table 4. Impulsivity as measured by neurocognitive measures

	Data			Group effects		Group contrasts		$\eta^2$	Power
	HCs (N = 31)	APGs (N = 9)	NAPGs (N = 13)	F	P	HCs vs. PGs	APGs vs. NAPGs		
IGT				1.12	0.335			0.043	0.236
IGT_Block 1	-2.19 ± 8.40	1.56 ± 5.55	-2.77 ± 6.25						
IGT_Block 2	4.39 ± 9.68	1.78 ± 5.52	-2.77 ± 6.25						
IGT_Block 3	6.13 ± 9.48	1.56 ± 8.59	4.31 ± 11.46						
IGT_Block 4	5.65 ± 9.38	4.22 ± 7.17	4.46 ± 11.02						
IGT_Block 5	8.58 ± 10.34	4.44 ± 9.20	5.08 ± 7.51						
DDT				1.57	0.219			0.064	0.316
DDT_logk10	-1.77 ± 0.59	-1.45 ± 1.12	-1.47 ± 0.63						
DDT_logk30	-2.00 ± 0.54	-1.39 ± 0.89	-1.58 ± 0.67						
DDT_logk100	-2.19 ± 0.77	-1.95 ± 1.26	-1.94 ± 0.90						
SCWT_RT				38.20	<0.001	<0.001	0.914	0.614	1.000
SCWT_RT_Neutral	523.19 ± 47.33	702.08 ± 98.99	689.88 ± 100.65						
SCWT_RT_Gambling	531.35 ± 37.60	700.18 ± 115.85	696.57 ± 89.04						

APGs: abstinent pathological gamblers; DDT: Delay Discounting Task; HCs: Healthy controls; IGT: Iowa Gambling Task; NAPGs: non-abstinent pathological gamblers; PGs: pathological gamblers; SCWT\_RT: Stroop Color Word Test\_Reaction Time. Group contrast was only mentioned when group effects were significant.

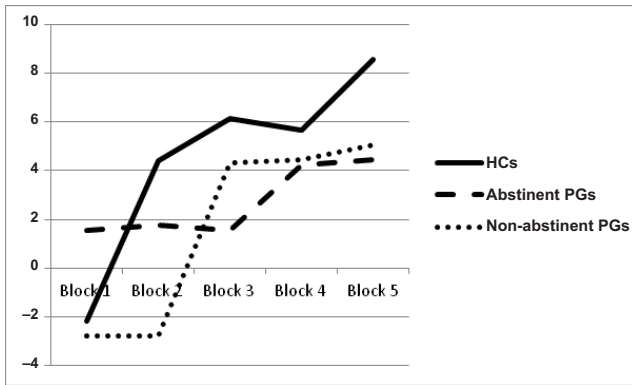


Figure 1. IGT block scores in healthy controls (HCs;  $N = 31$ ), abstinent (Abstinent PGs;  $N = 9$ ) and non-abstinent pathological gamblers (Non-abstinent PGs;  $N = 13$ )

### Personality and additional measures

The presence of personality disorders was rare. Results showed that neither HCs and PGs nor abstinent PGs and non-abstinent PGs differed in personality disorders (Table 1). There were no differences in IQ as measured with the NART (Table 1).

### Correlation analysis

There proved to be weak, non-significant correlations between both self-report questionnaires of impulsive personality (BIS, SPSRQ). In addition, weak, non-significant correlations were found between all neurocognitive measures of decision-making and impulsivity (DDT, IGT, SCWT). Age of onset was associated with BIS and DDT scores indicating that younger participants scored higher on self-report measures of impulsivity and were more inclined to save their money. Gambling severity (SOGS-scores) finally was positively associated with sensitivity to reward (SPSRQ\_SR), indicating that participants with more severe gambling problems were more inclined to respond to rewards (Table 2).

## DISCUSSION

Contrary to our initial hypothesis, PGs who relapsed did not differ on self-report and neurocognitive measures of impulsivity with PGs who did not relapse. However, both groups did differ in age at onset. Finally, healthy controls and PGs differed in some (BIS, SCWT), but not all impulsivity measures (DDT, IGT, SPSRQ).

Contrary to our hypothesis, neither self-reported impulsivity nor neurocognitive measures of decision-making and impulsivity were associated with relapse risk. This result is partially in agreement with earlier research findings documenting possible risk factors for relapse in pathological gambling. Indeed, both Goudriaan et al. (2008) and Álvarez-Moya et al. (2011) found weak, non-significant associations with self-reported impulsivity or Stroop interference scores and relapse risk. In addition and in further agreement with our data, no relations were found between measures of decision-making (IGT) and relapse risk. In these studies in contrast a significant relation was found with a measure of response inhibition (SST, Goudriaan et al., 2008) and an alternative measure of decision-making, the Card Playing Task (Álvarez-Moya et al., 2011; Goudriaan

et al., 2008). Goudriaan et al. (2008) suggested that differences between the IGT and the CPT may explain this particular finding. They suggest that a simple task as the CPT may tap into separate aspects of executive functioning, while a more complex task as the IGT may rely upon a mix of cognitive demands diluting the predicting power of an aspect such as “disinhibition”. However, it remains a remarkable finding that IGT performance does not relate to relapse within PGs. This is in clear contrast with the growing literature relating decision-making impairments with an increased risk on relapse after treatment (or drop out during treatment) in substance abusing patients (e.g., Bowden-Jones et al., 2005; Passetti et al., 2008). These differences in neurocognitive functioning and its consequences between pathological gamblers and substance abusers need to be explored. One possible explanation is that risk/reward decision-making is more impaired in pathological gamblers compared to substance abusing patients and an overall characteristic of pathological gamblers, leaving less room to differentially impact relapse risk. Indeed, in a recent review Leeman and Potenza (2012) suggested that PGs versus substance dependent patients, were characterized by less impairments in basic executive functions (i.e. working memory and attention) but by more severe impairments in reward/risk decision-making.

In contrast to the neurocognitive measures age of onset proved to be significantly associated with relapse. This finding is in accordance with the current literature on relapse in pathological gambling, showing that measures of gambling severity, including age of onset and years of gambling experience, were associated with successful abstinence. Pathological gamblers that began gambling at a younger age showed higher relapse risks than PGs that started gambling at a later age (e.g., Blaszczynski & Nower, 2002; Dowling, 2009; Goudriaan et al., 2008; McCormick & Taber, 1991). This finding further is consistent with studies within substance abusing samples, where age at onset is a significant marker associated with the severity and prognosis of the disorder (Dom, Hulstijn & Sabbe, 2006).

Finally and most remarkably, our PG and HC groups did not differ on self-report measures or on neurocognitive measures of impulsivity, with the exception of higher BIS scores in PGs compared to HCs. This finding is inconsistent with earlier studies demonstrating impairments in decision-making (DDT and IGT) in PGs versus controls (Goudriaan, Oosterlaan, de Beurs & van den Brink, 2005; Petry, 2001; for a review see Leeman and Potenza, 2012). However, this discrepancy may have resulted from our strict sample selection. Indeed, we selected a group of pathological gamblers specifically excluding psychiatric co-morbidity in order to avoid confounding effects on our measures by co-morbid disorders. Indeed, frequent co-morbid disorders in PGs samples such as SUD and (cluster B) personality disorders are themselves associated with higher self-reported and neurocognitive impulsivity measures. Overall it seems that our sample is reflective of a subgroup of non-impulsive PGs. This finding is in accordance with recent data published by Dannon, Shoenfeld, Rosenberg, Kertzman and Kotler (2010) indicating that PGs were no more impulsive than HCs, or even less impulsive in some instances. Overall, an increasing number of authors currently suggests the existence of different subtypes of pathological gambling (Álvarez-Moya et al., 2010; Blaszczynski & Nower, 2002; Shead, Callan and Hodgins, 2008). Our PG sample best resembles the conditioned or emotionally vulnerable problem

gamblers as defined by Blaszczynski and Nower (2002) or type IV or high-functioning problem gamblers described by Alvarez-Moya et al. (2010). All of these PGs subgroups are defined as having a more adaptive personality profile, lower levels of substance use, and fewer psychopathological disturbances (Alvarez-Moya et al., 2010; Blaszczynski & Nower, 2002) and thus fewer impulsivity deficits. In contrast, the other subtypes as defined by these authors are characterized by a higher prevalence of (externalizing) psychiatric co-morbidity such as SUD and (impulsive) personality types (cluster B). It is an interesting hypothesis to be explored in future research, whether those subgroups (based on clinical variables) can be differentiated based upon their underlying neurocognitive profile. Our data suggest at least, that a non-impulsive subgroup exists and is characterized by normal performance on measures of inhibition and decision-making.

The limited sample size is undoubtedly the most serious limitation of the current study, warranting replication within larger samples. Main strengths, however, are the homogeneous sample of PGs without psychiatric co-morbidity, the long-term follow-up of 12 months and the very low number lost to follow-up.

Taken together, the results of our pilot study show that one-year relapse in a small group of PGs without comorbid other psychiatric disorders is not predicted by self-report or neurocognitive measures of impulsivity and decision-making. The absence of differences on self-report and neurocognitive measures of impulsivity and decision-making between HCs and PGs illustrates the relative health of the examined PGs group, regardless of their pathological gambling. This particular finding emphasizes the need to further look into the differences between subtypes of pathological gamblers in future studies exploring neurocognitive mechanisms underlying the pathogenesis and chronicity of this disorder.

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