Effects of impulsivity, reinforcement sensitivity, and cognitive style on Pathological Gambling symptoms among frequent slot machine players

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A B S T R A C T

Pathological Gambling (PG) is the inability to resist recurrent urges to gamble excessively despite harmful consequences to the gambler or others. A cognitive-behavioral Pathways Model of PG (Blaszczynski & Nower, 2002) suggests individual differences in rash impulsivity and reward sensitivity, together with a cognitive style that promotes poor decision making, as risk factors. These individual differences were examined in a community sample of experienced slot machine players (N = 100), who were classified into Low, Moderate, and Problem gambling groups according to the Problem Gambling Severity Index (Ferris & Wynne, 2001). There were significant group differences on rash impulsivity as measured by the Eysenck Impulsivity scale, and on reward sensitivity as measured by the BIS/BAS Drive scale. For cognitive style, there were differences on Actively Openminded Thinking (AOT), but not the Rational Experiential Inventory. Hierarchical regression analyses found that impulsivity and AOT predicted severity of PG, but that AOT mediated the effect of BAS Drive. A thinking style that promotes erroneous cognition may correlate with PG, but individual differences in rash impulsivity and reward-seeking play a more critical role in the etiology of PG. The individual characteristics of Pathological Gamblers are similar to those of people with Substance Use Disorders.

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1. Introduction

Pathological Gambling (PG) is the inability to resist recurrent urges to gamble excessively despite harmful consequences to the gambler or others. The most influential account of the phenomenon is the Pathways Model of Problem and Pathological Gambling (Blaszczynski & Nower, 2002). According to this theory, “Operant conditioning occurs when intermittent wins delivered on a variable ratio produce states of arousal often described as equivalent to a drug induced high.”, and once the habit of gambling is acquired, “Attempts to resist completing the habit provoke a state of aversive arousal experienced as a drive, compulsion or urge to carry out the behavior.” (p. 491). This learning mechanism implies that clinical PG symptoms should be increased among people who gamble frequently, who are motivated strongly by the excitement of winning, and who have weak control over their compulsion to gamble excessively. Disinhibition of the gambling habit involves both reward seeking and rash impulsivity, and in this regard the Pathways Model is consistent with the 2-Component Approach to Reinforcing Substances model of addiction (Gullo, Ward, Dawe, Powell, & Jackson, 2011).

The Pathways Model also proposes a complex set of cognitive mechanisms that promote persistent gambling despite recurring losses. This is due to faulty decision making that leads to desperate “chasing” of lost money:

As the frequency of gambling increases, strong biased and distorted cognitive schemas appear. These schemas shape beliefs surrounding attribution, personal skill and control over outcome, biased evaluations, erroneous perceptions, superstitious thinking and probability theory. The potency and pervasiveness of distorted and irrational cognitive belief structures strengthen with increasing levels of involvement in gambling (p. 491).

Thus, the Pathways Model proposes two distinct phases of PG: an acquisition phase marked by habit formation through conditioning, and a maintenance phase characterized by irrational decision-making. The focus of the present paper is on individual differences in personality and cognitive style that may elevate vulnerability to clinical PG symptoms among people who gamble frequently. It was predicted that PG symptoms would be positively correlated with reward sensitivity and impulsivity because those traits should facilitate acquisition, and be negatively correlated with a rational cognitive style necessary for effective decision-making.

1.1. The personality of Pathological Gamblers

The personality of Pathological Gamblers is characterized by Negative Affective and Disinhibitory traits, including facets of
impulsivity (MacLaren, Fugelsang, Harrigan, & Dixon, 2011). This combination may form an externalizing dimension of psychopathology (Markon, Krueger, & Watson, 2005; Widiger, 2011) that may be expressed as PG, as Substance Use Disorders (Kotov, Gámez, Schmidt, & Wilson, 2010), and in other behaviors that reflect Antisocial or Borderline features (Samuel & Widiger, 2008). These meta-analytic findings are consistent with the Pathways Model in its assertion of a specific subtype of “Antisocial Impulsivist” Pathological Gambler with elevated risk compared to those who are merely “Behaviorally Conditioned” but without personality pathology (Milosevic & Ledgerwood, 2010).

Impulsive antisociality is a dimension of psychopathy (Ross, Benning, Patrick, Thompson, & Thirston, 2009), which reflects the maladjustment of brain systems that control learning to seek rewards and to fear punishments (Corr, 2010; Fowles, 1988; Lykken, 1955). According to the Reinforcement Sensitivity Theory of personality (RST; Gray & McNaughton, 2000), the neuropsychological substrate of excessive reward seeking is the Behavioral Approach System (BAS), which consists of the mesolimbic and mesocortical dopaminergic systems (Gray & McNaughton, 2000). The Fight–Flight–Freeze and Behavioral Inhibition Systems (BIS) mediate affective and behavioral responses to threat and response conflict, and include the amygdala and septo-hippocampal structures. Exposure to unusually powerful sources of reward (e.g. winning large prizes) may sensitize the BAS and feedback positively into a compulsive pattern of addictive behavior (Koob & Le Moal, 2008; Robinson & Berridge, 2000).

Reward sensitivity is a critical component of disinhibition in PG. In student samples, BAS Drive (Carver & White, 1994) correlates positively with time and money spent gambling (O’Connor, Stewart, & Watt, 2009), and Sensitivity to Reward (Torrubia, Avila, Moltó, & Caseras, 2001) correlates positively with PG symptoms (Mercer & Eastwood, 2010). Clinical studies of Pathological Gamblers have found higher BAS (Goudriaan, Oosterlaan, deBeurs, & van den Brink, 2006), and Sensitivity to Reward (Loxton, Nguyen, Casey, & Dawe, 2008) compared to nonpathological gamblers. In experimental studies, high BAS predicts risky decision-making (Franken & Muris, 2005; Suhr & Tsa nadis, 2007) in the Iowa Gambling Task (Bechara, Damasio, Damasio, & Lee, 1999) and on a computerized dice game (Kim & Lee, 2011), as well as higher wagers on simulated slot machines (Brunborg, Johnsen, Mentzoni, Molde, & Pallesen, 2011; Demaree, DeDonno, Burns, & Everhart, 2008).

1.2. The cognition of PathologicalGamblers

Distorted cognitions are common among Pathological Gamblers (Joukhador, MacCallum, & Blaszczynski, 2003). They may easily rec all wins because of an availability heuristic (Tversky & Kahneman, 1974a), they may fail to deliberately weigh the probability of winning against the risk of losing (Fletcher, Marks, & Hine, 2011), and they may misattribute winning to personal skill because they have an illusion of control (Langer, 1975). Reliance on such cognitive heuristics and biases is a trait-like characteristic (Epstein, Pacini, Denes-Raj, & Heier, 1996) that is separate from general cognitive ability (West, Toplak, & Stanovich, 2008). Cognitive style ranges from a rational, logical and fact-based thinking style to one that is intuitive, emotional and heuristic. Cognitive style can be measured using the Rational Experiential Inventory (REI; Pacini & Epstein, 1999), or the Actively Openminded Thinking test (AOT; Stanovich & West, 2008).

The role played by cognitive style in PG has been directly tested in two studies. Emond and Marmurek (2010) found that Pathological Gamblers had higher scores on a measure of erroneous gambling cognitions and lower scores on the REI Rational scale than nonproblem gamblers. Toplak, Liu, MacPherson, Toneatto, and Stanovich (2007) found lower scores among Pathological Gamblers than nonproblem gamblers on a subset of externalizing measures of psychopathology.
those with a score of 3–7 as ‘Moderate’ gamblers, and those with a score of 8–27 as ‘Problem’ gamblers. Cronbach’s α was .90.

2.3.2. Eysenck Impulsivity questionnaire

The Eysenck Impulsivity scale, version 7 (Eysenck et al., 1985) is a widely used measure of rash impulsiveness. The 19 items were endorsed by circling Yes or No. Total scores were divided by the number of scored items to eliminate missing values (0.3% of questions). Cronbach’s α was .85.

2.3.3. BIS/BAS scales

The BIS/BAS scales (Carver & White, 1994) measure the sensitivity of behavioral approach and avoidance systems. The BAS items measure Reward Responsiveness (5 items), Drive (4 items), and Funseeking (4 items). In keeping with revised RST (McNaughton & Corr, 2008), the BIS scale was divided into separate subscales measuring Fear (4 items) and Anxiety (3 items) following Heym, Ferguson, and Lawrence (2008). Items were rated on a 4-point Likert scale: (1) strongly disagree, (2) disagree, (3) agree, (4) strongly agree. Total scores were divided by the number of scored items to eliminate missing values (1.4% of questions). Cronbach’s α for Reward, Drive, Funseeking, Fear and Anxiety was .82, .85, .91, .59, and .64, respectively.

2.3.4. Rational Experiential Inventory

The REI (Paciﬁ & Epstein, 1999) measures analytical-rational and intuitive-experiential cognitive styles. The 20 items of each dimension were rated on 5-point Likert scales: (1) definitely not true of myself, (2) somewhat not true of myself, (3) may be true or not true of me, (4) somewhat true of myself, (5) definitely true of myself. Total scores were divided by the number of scored items to eliminate missing values (2.2% of questions). Cronbach’s α of the Rational and Experiential scales was .92 and .95 respectively.

2.3.5. Actively Openminded Thinking test

The AOT (Stanovich & West, 2008) measures the tendency toward novel versus conservative thinking and is conceptually similar to the REI Rational scale. The 41 items were rated on 6-point Likert scales ranging from (1) definitely not to (6) strongly agree. Total scores were divided by the number of scored items to eliminate missing values (1.3% of questions). Cronbach’s α was .91.

2.3.6. Consideration of future consequences scale

The CFC (Strathman et al., 1994) is a self-report measure of delay nondiscounting in which respondents consider distant versus immediate consequences of potential behaviors. The 12 items were rated on a 5-point Likert scale: (1) extremely uncharacteristic of me, (2) uncharacteristic of me, (3) somewhat characteristic of me, (4) characteristic of me, (5) extremely characteristic of me. Total scores were divided by the number of scored items to eliminate missing values (0.8% of questions). Cronbach’s α was .84.

3. Results

As shown in Table 1, the Problem group had higher I7 and lower AOT scores than the Low and Moderate groups. The Problem group also had higher BAS Drive than the Low group. Importantly, the groups did not differ on the REI Rational scale. As shown in Table 2, continuous PGSI scores were correlated positively with I7, BAS Drive and Funseeking, and BIS Fear. PGSI scores were also correlated negatively with CFC and AOT, but not with REI Rational. The null results for REI Rational are contrary to predictions from the Pathways Model.

There were complex intercorrelations among the personality and cognitive style scales. To examine their unique contributions to PG severity, the continuous PGSI scores were regressed onto age and sex, followed by the 10 scales. The overall regression equation was significant, $F(12,81) = 4.38$, $p < .001$, $R^2 = .39$, with I7 ($β = .43, t = 3.51, p = .001$), and AOT ($β = -.32, t = 3.33, p = .001$) accounting for unique variance. The only other predictor that approached significance was BAS Drive ($β = .21, t = 1.81, p = .074$). Because of the high degree of intercorrelation among the measures of personality and cognitive style, two secondary regression analyses were carried out to test for mediation effects.

To examine the possible mediation of other predictors by AOT, we repeated the above regression analysis, but did not include AOT as a predictor. PGSI was regressed onto age and sex, followed by the remaining 9 scales. This second regression equation was significant, $F(11,82) = 3.35, p = .001$, $R^2 = .31$, with I7 ($β = .48, t = 3.66, p < .001$) and BAS Drive ($β = .28, t = 2.38, p = .020$) accounting for unique variance. Because both Drive and AOT correlate significantly with each other and with PGSI, and because Drive was a significant predictor of PGSI only when AOT was not simultaneously entered, it appears that the nonsignificant effect of Drive on PGSI in the first regression was due to mediation of its effect by AOT (Baron & Kenny, 1986). Together with the null effects of REI Rational, this pattern indicates that the hypothesized contribution of cognitive style to PG is not supported.

We similarly tested for mediation effects involving impulsivity. Here PGSI was regressed onto age and sex, followed by the remaining 9 scales. This third equation was significant, $F(11,82) = 3.21, p = .001$, $R^2 = .30$, with only AOT ($β = -.36, t = 3.49, p = .001$) accounting for unique variance. The only other predictor that approached significance was BAS Drive ($β = .23, t = 1.89, p = .062$). This shows that the effect of I7 on PGSI in the first regression did not mediate the effect of any of the other variables.

4. Discussion

Individual differences in traits representing approach motivation (i.e. BAS Drive) and inhibitory control (i.e. I7) systems were the best predictors of clinical PG symptoms among frequent slot machine gamblers. This is consistent with the 2-CARS model of personality characteristics (Gullo et al., 2011) that may reﬂect neurobiological mechanisms of addiction (George & Koob, 2010). The Negative Affective and Disinhibitory traits of Pathological Gamblers (MacLaren et al., 2011) are similar to those who have Substance Use Disorders (Kotov et al., 2010), and there appear to be common biological substrates that support these addictive disorders (van Holst, van den Brink, Veltman, & Goudriaan, 2010; Zack & Poulos, 2009).

The Pathways Model suggests a central role for poor critical thinking (i.e. low REI Rational or AOT scores) in the maintenance of PG behavior, but the results of the present study call this crucial element of the theory into question. We found null effects of REI Rational, and a spurious effect of AOT that was due to its mediation of BAS Drive. As for delay nondiscounting, the CFC scale appears to have tapped a well-established role of impulsivity in PG, since it was negatively correlated with I7 and those scales had an inverse pattern of correlations with other measures. The lack of an effect of REI Rational, which is contrary to the ﬁndings of Emond and Marmurek (2010) and Toplak et al. (2007), might have been due to range restriction caused by the inclusion of only high frequency slot machine gamblers in our sample. The correlation between REI Rational and PG might also be attenuated because REI Rational correlates negatively with Neuroticism (Paciﬁ & Epstein, 1999), which is elevated in PG (MacLaren et al., 2011) and is conceptually related to BIS (Smillie, Pickering, & Jackson, 2006). In our sample, REI Rational
was negatively correlated with both subscales of BIS and with I7, which may have contributed to the null effects of REI Rational on PGSI. Sensitivity to both reward and punishment may be important in PG, as gamblers derive arousal not only from the anticipation of winning, but also from the fear of losing (McNaughton & Corr, 2009).

Pathological Gamblers often have a bizarre understanding of the games they play and why they play them. These cognitions are often situational and individual gamblers may simultaneously hold beliefs that are not logically coherent. For instance, a gambler might continue betting after a series of losing outcomes and accept the gambler’s fallacy, the belief that a winning outcome must be imminent because the preceding series of losses is unlikely to continue even though the outcomes are independent (Tversky & Kahneman, 1974b). Alternatively, a gambler might continue betting on a variable reinforcement schedule typical of slot machines, where the occurrence and magnitude of wins are both unpredictable, is likely to be persistent no matter what gamblers may think (Skinner, 1953). To our knowledge, there is insufficient evidence that erroneous gambling cognitions precede and cause Pathological Gambling. The present study has shown that Pathological Gamblers do not inherently tend toward faulty thinking. Happily, the lack of a deficiency in cognitive style among Pathological Gamblers suggests that cognitive-behavioral interventions should be no less effective for PG than for other forms of psychopathology.

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References


