A pilot study of impulsivity and compulsivity in pathological gambling

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Abstract

We examined the relationship between gambling severity, impulsivity and obsessionality/compulsivity in thirty-eight pathological gamblers, comprising the complete Minnesota sample of a randomized, placebo-controlled clinical trial of paroxetine for the treatment of Pathological Gambling (PG), using Pearson correlations and linear regression models at baseline and treatment endpoint. At baseline, Pathological Gambling Modification of the Yale-Brown Obsessive-Compulsive Scale (PG-YBOCS) scores correlated significantly with those of the Eysenck Impulsiveness Questionnaire (EIQ) Impulsiveness subscale and Padua Inventory (PI) factors I and IV (corresponding to impaired control over mental and motor activities, respectively). None of the associations between PI factors and the PG-YBOCS were significant after adjusting for Impulsiveness scores. There were no differences in changes in the PG-YBOCS between the paroxetine and placebo group. Changes in PG-YBOCS scores after treatment correlated with changes in Impulsiveness scores. These changes appeared independent of paroxetine treatment. These results suggest that, although PG exhibits features of both obsessionality/compulsivity and impulsivity and elements of both decrease with treatment, impulsivity predominates and changes in gambling severity are most associated with changes in impulsivity.

Keywords

Pathological Gambling; Impulsivity; Compulsivity; Impulse control disorder; Behavior Addiction

1.) Introduction

Impulsivity transcends multiple psychiatric disorders (Moeller et al. 2001) and is thought to be central to impulse control disorders such as pathological gambling (PG) (Blaszczynski et

al., 1997; Petry and Casarella, 1999; Petry, 2001a; Potenza et al., 2001). The relationship between impulsivity and obsessionality/compulsivity is relatively poorly understood, particularly as related to specific psychiatric disorders and their treatments. Multiple studies have reported that pathological gamblers score higher than healthy volunteers on measures of impulsivity (Blaszczynski et al., 1986; Blanco et al., 1996), and one report observed pathological gamblers scoring higher than social gamblers on obsessionality/compulsivity (Blaszczynski 1999). Although pharmacological approaches to PG have been based on the postulate that the studied medications target features of impulsivity or compulsivity, such hypotheses have not been formally tested. A more precise knowledge of the clinical features targeted by these medications may help improve our understanding of the neurobiology of pathological gambling and guide future treatment research.

Rationales for the study of serotonin reuptake inhibitors (SSRIs) in the treatment of PG have been based on their efficacy in treating obsessive-compulsive disorder and/or the relationship between serotonin and impulsivity (Grant et al. 2003a). SSRIs are considered first-line treatments for obsessive-compulsive disorder. At the same time, a number of studies have documented the relationship between abnormalities in the serotonergic system and different disorders related to impulsivity. Prior to the conduct of this study, several clinical trials had suggested that SSRIs, including paroxetine (Hollander et al., 1998; Blanco et al., 2002; Kim et al., 2002) might be useful in the treatment of PG, although they had also documented high placebo response rates.

We sought to examine the extent to which PG symptom severity correlated with obsessionality/compulsivity and impulsivity at baseline, and whether changes in PG symptomatology during treatment with paroxetine were associated with changes in obsessionality/compulsivity and impulsivity. We hypothesized that: 1) consistent with its diagnostic classification as an impulse control disorder, PG severity would correlate with impulsivity rather than with obsessionality/compulsivity; 2) decreases in gambling behavior would correlate with decreases in impulsivity, and, 3) patients treated with paroxetine would have greater decreases in impulsivity and gambling behavior than those treated with placebo.

2.) Methods

2.1 Subjects

This study was conducted as an ancillary study in one of the sites (Minnesota) participating in a larger, multicenter placebo-controlled study of paroxetine for pathological gambling (Grant et al. 2003b). Subjects from the other sites were not administered the impulsivity and compulsivity measures described below and, therefore, could not be included in this study. The University of Minnesota's institutional review board approved the study. The study was carried out in accordance with Good Clinical Practice Guidelines and the Declaration of Helskinki. All participants provided written informed consent for this study. Subjects were 18 years and older with a primary DSM-IV diagnosis of PG and no current axis I comorbidity (except possibly nicotine dependence) or lifetime history of bipolar or psychotic disorders. Patients in the study were not allowed to receive any other interventions (including Gamblers Anonymous) during the study. After a one-week placebo run-in, eligible patients were randomized using a computer-generated table of random numbers to 16 weeks of placebo (n=20) or paroxetine (n=18) up to 60 mg/day.

2.2 Assessments

Structured Clinical Interviews for DSM-IV (SCID) (First 1995) and Pathological Gambling (SCI-PG) (Grant et al., 2004; Pallanti et al., 2005) were used for diagnostic evaluation. At

study entry and termination (week 16 or earlier), patients were administered the Yale-Brown Obsessive-Compulsive Scale modified for PG (PG-YBOCS), the Padua Inventory (PI), and the Eysenck Impulsiveness Questionnaire (EIQ).

The PG-YBOCS, a valid and reliable measure of PG symptomatology, is a 10-item, 5-point severity, clinician-administered scale (Hollander et al. 1998). The first five items of the PG-YBOCS comprise the gambling urge/thought subscale (time occupied with urges/thoughts; interference and distress due to urges/thoughts; resistance against and control over urges/thoughts), and items 6-10 comprise the gambling behavior subscale (time spent gambling and amount of gambling; interference and distress due to gambling; ability to resist and control gambling behavior). Each item is rated from 0 to 4, with higher scores reflecting greater severity, and the total score thus ranges from 0 to 40. It has shown excellent inter-rater reliability, and convergent validity with the Clinical Global Impression Scale and the South Oaks Gambling Screen (Stinchfield 2003).

The PI is a reliable and valid 60-item, 5-point severity self-report inventory that measures obsessions and compulsions and contains four factors: 1) Impaired control over mental activities, which assesses ruminations and exaggerated doubts; 2) Fear of contamination; 3) Checking; and, 4) Impaired control over motor activities which measures urges and worries related to motor behavior, such as violent impulses (Sanavio 1988). It has been used in clinical samples of individuals with PG (Blaszczynski 1999) obsessive-compulsive disorder (van Balkom et al. 1998), polysubstance abusers (Sumnall et al. 2004), and in general population samples (Mancini et al. 2002). It has shown high internal consistency, test-retest reliability and good convergence validity with other measures of obsessionality-compulsivity (Sanavio 1988).

The EIQ is a commonly used measure of impulsivity. It consists of three subscales: Impulsiveness, Venturesomeness, and Empathy. Impulsiveness assesses the tendency to act without forethought, Venturesomeness measures sensation-seeking, and Empathy quantifies sociability. The EIQ was developed through several iterations in non-clinical samples (Eysenck and Eysenck, 1978; Eysenck and McGurk, 1980; Eysenck et al., 1985). It has good test-retest reliability, and convergence validity with other measures of impulsivity (Eysenck et al., 1985; Dickman, 1990). Previous studies have demonstrated elevated EIQ scores in pathological gamblers (Blaszczynski et al., 1997; Clarke, 2004).

2.3 Data Analysis

Prior to statistical testing, variables were examined for normality. Most variables did not follow a normal distribution. To circumvent the problem of heteroskedasticity, baseline values of all variables were log-transformed prior to statistical analysis (Neter 1996). To measure changes over time, analyses of covariance, using the baseline score as covariate and group treatment (paroxetine or placebo) as the predictor, were used to compare changes over treatment between the paroxetine and placebo groups in PG-YBOCS, PI and EIQ and their subscales.

Pearson product-moment correlations of the log-transformed variables were used to investigate the associations between scores on the PG-YBOCS, PI, and EIQ at baseline. Pearson correlations were also used to investigate whether change in PG-YBOCS scores from baseline to endpoint were associated with concurrent changes in scores on the PI and EIQ. Pearson correlations were also calculated between the PG-YBOCS and each of the PI and EIQ subscales at baseline and over time to more precisely investigate the components of obsessionality/compulsivity and impulsivity that may be related to PG.

Baseline variables whose correlation coefficients with PG-YBOCS scores were significant at $\alpha \leq 0.1$ were entered as predictors into a linear regression model with baseline PG-YBOCS scores as the outcome variable to examine the strength of the association of each predictor after adjusting for all other predictors. An $\alpha \leq 0.1$ rather than $\alpha \leq 0.05$ was selected as entry criterion in the regression models to avoid premature elimination of variables that might have not reached conventional levels of statistical significance in the bivariate analysis, but could become significant after adjusting for additional covariates (Neter et al., 1996). Similar linear models were constructed using the change variables (e.g. change in log-transformed pre-post Impulsiveness scores) as predictors of change in PG-YBOCS. To avoid multicollinearity (which can result in unstable estimates of the regression coefficients), when both a factor or subscale and the overall scale scores had a statistically significant correlation with the PG-YBOCS score in the bivariate analysis, two separate regression models were fit: one using the factors or subscales as predictors, and another using the overall scale as a predictor. The degree of multicollinearity was assessed using the variance inflation factor (VIF) (Neter 1996) Significance values reported correspond to two-tailed analyses.

3.) Results

Thirty-eight individuals, twenty men (52.6%) and eighteen women (47.4%), mean age 46.6 years (SD=9.0), comprising the complete sample of the Minnesota site of the multicenter placebo-controlled trial of paroxetine participated in the study (i.e., no participant in the treatment study declined participation in this investigation). Twenty-seven patients (71.1%) were married and twenty-eight (73.7%) had at least some college education. Only six (15.8%) were unemployed. All subjects in the sample were non-Hispanics (Table 1).

Data were obtained for all patients at baseline and termination. Ten subjects in the paroxetine and 12 in the placebo group completed all 16 weeks of the clinical trial. Eight subjects in the paroxetine group terminated the clinical trial prematurely due to noncompliance with study schedule and adverse events. Eight subjects in the placebo group terminated the clinical trial prematurely due to noncompliance with study schedule, lack of efficacy, and loss to follow-up. None of the placebo subjects terminated treatment prematurely due to adverse events. Overall, the men time to discontinuation among nonc-completers was 6.8 (SD=2.9) weeks.

Mean and standard deviations of the baseline and endpoint score for the full sample are presented in their original units in Table 2. Paired t-tests for the full sample showed significant decreases in gambling severity, the overall score in the Padua Inventory and its factor I, as well as with the total score of the Eysenck Impulsiveness Scale and its Venturosomeness subscale. Change in the Impulsiveness subscale approached but did not reach significance.

Analyses of covariance (ANCOVA) indicated that patients on placebo had higher decreases than patients on paroxetine on Factor I of the Padua Inventory as well as the total score of the Eysenck Impulsiveness Questionnaire. There were no other differences between paroxetine and placebo (see Supplementary Table).

Correlations of the scores on the PG-YBOCS with the Padua Inventory and the Eysenck Impulsiveness Scale (and their subscales) are presented in Table 3 for baseline and change values.

At baseline, PG-YBOCS scores had statistically significant correlations (P<.05) with scores on factors I and IV of the PI and the Impulsiveness scales of the EIQ, as well as the overall EIQ score. Change in the PG-YBOCS was significantly correlated with change in the

Impulsiveness and Venturesomeness scores of the EIQ, as well as the overall EIQ score (Table 3).

At baseline, the correlation between the Impulsiveness subscale of the EIQ and Padua factor I of $r=0.39$ (df=36, $P=.02$), while the correlation between Impulsiveness scale of the EIQ and Padua factor IV was $r=0.38$ (df=36, $P=.02$). The correlation between change in Impulsiveness subscale of the EIQ and change in Padua factor I was $r=0.21$ (df=36, $P=0.2$), while the correlation between change in Impulsiveness subscale of the EIQ and change in Padua factor IV was $r=0.02$ (df=36, $P=0.9$).

To examine the strength of the association of each predictor after adjusting for all other predictors, we fitted a regression model using PG-YBOCS baseline score as the outcome variable and used baseline scores on factors I and IV of the PI and the Impulsiveness subscale of the EIQ. These variables were selected as predictors in the regression equation because they were the only ones whose correlation coefficients with baseline PG-YBOCS were significant at $\alpha \leq 0.1$. This model identified the baseline Impulsiveness score as the only statistically significant predictor ($\beta=0.21$, standard error [SE]=0.07, $P=0.003$) and had an $R^2=0.37$, suggesting that this model explained a relatively high proportion of the variance in the PG-YBOCS scores at baseline. Maximum VIF for this model was 1.2, suggesting no significant multicollinearity (i.e., the predictors in the regression were not highly intercorrelated), and indicating that the estimates of the regression coefficients of the model were stable. Regression analyses using the overall PI score as the predictor variable instead of those from factors I and IV, or overall EIQ score instead of that of Impulsiveness yielded similar results (not shown).

A regression model using change in PG-YBOCS score as the outcome variable and changes in Impulsiveness and Venturesomeness scores as predictors identified Impulsiveness as the only significant predictor ($\beta=0.75$, SE=0.34, $P=0.03$), and had an $R^2=0.29$. Removal of the change in Venturesomeness variable from this full model resulted in a simplified model with also $R^2=0.29$, indicating that deletion of the change in Venturesomeness variable did not decrease the explanatory power of the regression model. Because the adjusted R^2 (a measure that balances the explanatory power of the regression model and its number of predictors) of this simplified model was 0.27, compared to an $R^2=0.25$ in the model including change in Venturesomeness, the simplified model including only change in Impulsiveness as predictor was preferred on the basis of parsimony.

4.) Discussion

This is the first study to examine simultaneously in a group of pathological gamblers measures of obsessionality/compulsivity and impulsivity. We found that treatment of PG was associated with decreased scores on measures of gambling, obsessionality/compulsivity, and impulsivity. Paroxetine treatment did not appear to influence the relationship between gambling, obsessionality/compulsivity, and impulsivity and treatment outcome. Although changes occurred in all domains (gambling, obsessionality/compulsivity, and impulsivity), the relationships among the domains were complex and provide insight into specific psychological dimensions and their association with treatment response in PG.

At baseline, PG symptom severity was associated with features of impulsivity and obsessionality/compulsivity. During treatment, overall scores on measures of impulsivity and compulsivity/obsessionality diminished, with significant decreases seen in factor I of the PI (impaired control over mental activities) and the venturesomeness and impulsiveness (trend) subscales of the EIQ. It is possible that factor I may be related to the anxiety symptoms that are common among pathological gamblers (Black and Moyer, 1998; Ibanez

et al., 2001; Petry et al., 2005), and may improve with treatment. However, improvement in symptoms of PG was only associated with decreases in impulsivity and not obsessionality/ compulsivity. More specifically, improvement in PG symptoms was associated with changes in aspects of impulsivity related to acting without forethought, rather than other impulsivity dimensions such as novelty- or sensation-seeking. Our findings, although preliminary, confirm and extend prior investigations on the relationship between PG, impulsivity and obsessionality/compulsivity (Blaszczynski et al., 1997; Blaszczynski, 1999).

The finding of a positive correlation between the level of impulsivity and PG symptom severity is consistent with prior work (Blaszczynski et al. 1997). Our findings on PI scores at baseline also replicate prior work, and extend it by documenting a correlation between baseline scores on factors I and IV with PG-YBOCS scores at baseline (Blaszczynski et al. 1997). These results reflect difficulties in controlling gambling-related thoughts and episodic loss of control over gambling, commonly reported by pathological gamblers.

Our study further extends prior findings by including and modeling concurrent measures of obsessionality/compulsivity. The results of our regression analysis suggest that the obsessive-compulsive dimensions of PG at baseline may be subsumed in the impulsive dimensions of the disorder. Furthermore, our results also indicate a relationship between treatment-related changes in PG symptoms and impulsivity, suggesting that changes in impulsivity might mediate changes in gambling symptoms. Although prior studies have documented elevated scores in several measures of impulsivity in pathological gamblers (Petry, 2001a; 2001b) and proposed neurobiological substrates for such increased impulsivity (Blanco et al., 1996; Potenza et al., 2003a, 2003b), this study is the first to relate changes in impulsivity with changes in PG symptomatology. Recent work in borderline personality disorder have also recently documented changes in impulsivity related to treatment (Hollander et al. 2005b) These findings suggest that changes in impulsivity may be achieved with relatively brief interventions. Future research should investigate the temporal stability of these changes.

Contrary to our hypothesis, there were no significant differences between the paroxetine- and placebo-treated groups regarding the changes in impulsivity or gambling behavior, suggesting that the changes in impulsivity were not due to a specific pharmacological effect. However, the small sample size limits the ability to exclude this possibility. The neurobiological changes underlying changes in impulsivity and PG symptomatology remain to be determined. An important direction for future research involves investigating whether changes in gambling behavior following psychotherapy are also associated with changes in impulsivity.

Our findings have treatment implications. Although improvement in PG symptoms was associated with changes in impulsivity, there were no differences between treatment groups, suggesting that those changes were not due to the effect of paroxetine. Serotonin reuptake inhibitors have demonstrated inconsistent efficacy in the treatment of PG (Grant et al. (2003a). Alternative treatments that target impaired impulse control and have shown initial promise in the treatment of PG, such as cognitive-behavioral relapse prevention (Sylvain et al., 1997; Petry, 2002), mood stabilizers (Pallanti et al., 2002; Hollander et al., 2005a) or mu-opioid receptor antagonists (Kim and Grant, 2001; Kim et al., 2001), should be further investigated. Mounting evidence indicates that impulsivity is a heterogeneous construct and different pharmacological and behavioral interventions may be effective for subtypes of impulsivity (Moeller et al. 2001). A more refined understanding of the different impulsivity domains, their underlying neurobiological substrates, and how they relate to PG may help develop better-targeted, theoretically-driven treatment strategies.

Our study has several limitations. First, all participants were pathological gamblers without axis I comorbidity (except possibly nicotine dependence) seeking pharmacological treatment in Minnesota. The results may not generalize to treatment-seeking pathological gamblers in other geographical locations or to the general population of pathological gamblers. Second, although we collected measures over time, we did not include PI and EIQ assessments at times other than baseline and termination, precluding the exploration of causality. Third, our assessment was based on self-reports, limiting the dimensions of impulsivity and obsessionality/compulsivity that could be examined. Future studies should include other measures of impulsivity, such as the Barratt Impulsivity Scale or performance-based measures of impulsivity (Dougherty et al. 2004) that continue to delineate the dimensions of impulsivity that are associated with PG. Fourth, imbalance in the allocation of males and females to paroxetine and placebo as a result of the randomization may have influenced the results of the study. However, there were no changes in the results when gender was included in the ANCOVA analyses as a covariate, while the correlations were calculated for the medication and placebo together. Fifth, the sample size was small, limiting the ability to identify possible clinically relevant relationships; e.g., between paroxetine and behavioral measures. Finally, we did not re-assess subjects beyond study completion. Therefore, we do not know the extent to which the relationship between impulsivity and gambling symptomatology is maintained over time. In particular, we do not know whether worsening of PG symptoms is associated with increases in impulsivity.

In conclusion, we observed that PG has aspects of obsessionality/compulsivity and impulsivity, that these measures decrease during treatment, and that changes in gambling severity correlate with changes in impulsivity. Strategies that target specific aspects of impulsivity deserve further investigation in the treatment of PG. A challenge for future research involves the identification of the neurobiological substrates of the different dimensions of impulsivity, and their relationship to treatment outcome.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Table 1

Demographics Characteristics of 38 Subjects with Pathological Gambling

Variable	Paroxetine (n=18)	Placebo (n=20)	χ^2	df	P
Age					
Mean (± SD), years	48.1 (9.5)	45.4 (8.6)	0.9[a]	36	0.4
Gender, n (%)					
Female	13 (72.2%)	5 (27.8%)	8.5	1	0.004
Male	5 (25.0%)	15 (75.0%)			
Ethnicity, n (%)[b]					
Not Hispanic/Caucasian	18 (100%)	20 (100%)			1.00
Marital Status, n (%)					
Single	1 (5.6%)	3 (15.0%)	7.81	2	0.020
Married	10 (55.6%)	14 (70.0%)			
Widow/Separated/Divorced	7 (38.9%)	3 (15.0%)			
Education, n (%)					
High school grad or less	3 (16.7%)	5 (25.0%)	7.09	3	0.069
Some college/trade school	11 (61.1%)	7 (35.0%)			
College graduate	4 (22.2%)	4 (20.0%)			
Post-college education	0 (0%)	4 (20.0%)			
Unemployed, n (%)[b]	1 (5.6%)	0 (0%)			1.00

[a] T-test rather than χ^2-test used.

[b] Fisher exact test, rather than χ^2-test used.

Table 2

Baseline and endpoint scores on the Pathological Gambling Modification of the Yale-Brown Obsessive-Compulsive Scale (PG-YBOCS), the Padua Inventory (PI), and the Eysenck Impulsiveness Questionnaire (EIQ)

Variable[a]	Mean (SD)		t[b,c]	P
	Baseline[a]	Endpoint[a]		
PG-YBOCS	19.2 (5.0)	13.9 (9.6)	2.6	0.01
Padua Inventory				
Total	30.1 (26.6)	18.3 (17.4)	2.6	0.01
Factors				
I	9.8 (9.9)	6.6 (7.7)	2.5	0.02
II	5.7 (6.5)	3.8 (3.8)	0.8	0.4
III	3.6 (4.7)	3.5 (4.4)	0.9	0.4
IV	0.9 (1.4)	1.2 (1.98)	.07	0.9
Eysenck Impulsiveness Questionnaire				
Total	30.0 (7.1)	26.0 (11.1)	2.6	0.01
Subscales.				
Impulsiveness	9.4 (4.3)	8.2 (4.8)	1.7	0.06
Venturesomeness	7.9 (3.9)	6.6 (4.2)	2.1	0.03
Empathy	12.7 (3.2)	11.2 (4.8)	2.8	0.1

[a] In original (i.e., non-log-transformed) units.

[b] All df=37.

[c] All tests based on log-transformed variables (see text for explanation).

Table 3

Correlation coefficients of scores on the Pathological Gambling Modification of the Yale-Brown Obsessive-Compulsive Scale (PG-YBOCS) with those on the Padua Inventory (PI) and Eysenck Impulsiveness Questionnaire (EIQ)

Variable	Correlations at Baseline[a,b]		Correlations between Change Scores[a,c]	
	$r^{e,f}$	P	r^g	P
Padua Inventory				
Total	0.08	0.6	0.18	0.3
Factors				
I	0.34	0.04	0.21	0.2
II	-0.02	0.9	0.18	0.3
III	-0.07	0.7	-0.24	0.3
IV	0.36	0.03	0.001	.99
Eysenck Impulsiveness Questionnaire				
Total	0.51	0.001	0.49	0.002
Subscales				
Impulsiveness	0.58	<0.001	0.54	<0.001
Venturesomeness	0.24	0.1	0.44	0.006
Empathy	0.03	0.9	-0.14	0.4

[a] All df=36.

[b] Correlations between log-transformed PG-YBOCS baseline scores and log-transformed PI and EIQ baseline scores.

[c] Correlations between log-transformed PG-YBOCS change scores and log-transformed PI and EIQ change scores.

[e] Correlation between PG-YBOCS at baseline and the remaining variables in the table. The correlation is not present for PG-YBOCS with itself because by definition it is identically 1.

[f] Calculated by subtracting the post- from the pre-treatment score.

[g] Correlation between change in PG-YBOCS and the change in the remaining variables in the table. Correlation of change PG-YBOCS with itself is not presented because by definition it is identically 1.