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Gender-Related Clinical and Neurocognitive Differences in Individuals Seeking Treatment for Pathological Gambling

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Abstract

Objectives—Understanding variations in disease presentation in men and women is clinically important as differences may reflect biological and sociocultural factors and have implications for selecting appropriate prevention and treatment strategies. The aim of this study was to investigate clinical and cognitive differences in treatment-seeking people with pathological gambling as a function of gender.

Materials and methods—501 adult subjects (n=274 [54.7%] females) with DSM-IV pathological gambling presenting for various clinical research trials over a 9-year period were assessed in terms of sociodemographics and clinical characteristics. A subset (n=77) had also undertaken neuropsychological assessment with the Stop-signal and set-shift tasks.

Results—PG in females was associated with significantly worse disease severity, elevated mood and anxiety scores, and history of affective disorders, later age of study presentation, later age of disease onset, and elevated risk of having a first-degree relative with gambling or alcohol problems. These findings were of small effect size (0.20–0.35). Additionally, PG in females was associated with proportionately more non-strategic gambling with medium effect size (0.61). In contrast, PG in males was associated with significantly greater lifetime history of alcohol use disorder and any substance use disorder (small effect sizes 0.22–0.38); and slower motoric reaction times (medium effect size, 0.50). Response inhibition and cognitive flexibility were similar between the groups.

Conclusions—These data suggest that important differences exist in the features of pathological gambling in women and men. Findings are of considerable relevance to clinicians and in terms of targeted treatments.

Keywords

gender; impulse control disorders; gambling; cognition; addiction; phenomenology

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INTRODUCTION

Pathological gambling (PG), characterized by persistent and recurrent maladaptive patterns of gambling behavior, is associated with impaired functioning, reduced quality of life, and high rates of bankruptcy, divorce and incarceration (American Psychiatric Association, APA, 2010). Prevalence rates for PG in the USA are of the order of 0.4% to 1.1% (Blanco et al., 2006; Hodgins et al., 2011), and approximately 20–40% of individuals with PG are women (Potenza et al., 2001; Petry & Oncken, 2002; Fong et al., 2011).

Recent health initiatives have highlighted the importance of understanding gender differences (<http://www.womenshealth.gov/about-us/>). In terms of clinical presentation, there is considerable heterogeneity within PG (Hodgins et al., 2011) and several studies have found that significant clinical differences exist between male and female pathological gamblers. Men with PG are more likely to be single and live alone (Feigelman et al., 1998; Crisp et al., 2004). In addition, male pathological gamblers are more likely to have sought treatment for substance abuse (Ladd & Petry, 2002), have higher rates of antisocial personality traits (Ibáñez et al., 2003), and have greater negative marital consequences due to their gambling (Ibáñez et al., 2003). Whereas male gamblers report advertisements as eliciting urges to gamble, female gamblers more frequently report feelings of boredom or loneliness as triggers (Ladd & Petry, 2002; Grant & Kim, 2002). As compared to men, women with PG typically begin gambling later in life and the time interval between starting to gamble and developing a gambling problem is usually shorter for women, consistent with a “telescoping” progression (Potenza et al., 2001; Grant & Kim, 2002; National Opinion Research Center, 1999).

Pathological gambling has been associated with a variety of neurocognitive problems, across such domains as decision-making, working memory, cognitive flexibility, and inhibitory control (van Holst et al., 2010). Some of these findings may have been driven by inclusion of patients with Axis-I co-morbidities (such as depression, linked to cognitive problems itself). The issue of cognitive dysfunction in PG is important in order to understand the neurobiology of the disorder, its relationship with other psychiatric conditions, and treatment mechanisms (Clark, 2010). In particular, we have previously reported impaired cognitive flexibility and response inhibition using objective computerized tasks in people with PG who were free from Axis-I co-morbidities (Odlaug et al., 2011). These deficits were not evident in people at increased risk (subsyndromal gamblers), suggesting that they manifest only in the more extreme, pathological form. Despite some evidence, outlined above, that clinical and demographic characteristics differ between male and female pathological gamblers, little is known as to whether gender plays a differential role in the manifestation of cognitive deficits. Common cognitive problems in males and females may be suggestive of common neural dysfunction while distinct deficits may be suggestive of differential pathophysiology.

The goal of the present study was to examine clinical and cognitive commonalities and differences in males versus females with PG presenting as part of research trials. Based on multiple studies that suggest men with PG progress to addiction at a slow rate (e.g. Potenza et al., 2001) and have high levels of impulsiveness that extend across multiple domains (Slutske et al., 2001), our first hypothesis was that men with PG would report a longer progression from gambling onset to meeting PG diagnostic criteria, have greater impairment of impulse control as evidenced by more severe PG symptoms, and greater problems with substance use disorders. With respect to cognitive dysfunction, we hypothesized that because male gamblers tend to score higher on measures of risk-taking (Johansson et al., 2009), they would score higher on cognitive assessments of impulsivity.

METHODS

Subjects

Participants included 501 consecutive adults aged 18 years meeting current (past-year) DSM-IV criteria for pathological gambling. Participants were recruited by advertisements and referrals and had participated in treatment studies (cognitive behavioral therapy and pharmacotherapy). No significant differences in demographics were noted between those enrolled in drug vs therapy trials. Subjects were recruited over a 9-year period (2002–2011) at a public university medical center, and clinical characteristics of subgroups within this study have been previously reported (Grant & Potenza, 2005; Grant et al., 2008; Schreiber et al., 2009; Grant et al., 2009; Grant et al., 2010).

All subjects who met criteria for a treatment study were included in this database if they meet the inclusion criteria: 1) primary diagnosis of current DSM-IV pathological gambling; 2) age 18 or older; 3) Hamilton Depression Scale scores ≤ 17 ; and 3) able to be interviewed in person. Exclusion criteria for the clinical trials from which these data were derived included: 1) lifetime psychotic or bipolar disorder; 2) an inability to understand and consent to the study; and 3) substance abuse or dependence (past 12-months). Subjects were allowed to maintain current psychotropic medication use provided they had been on a stable dose of medication for at least six-weeks preceding study enrollment. The studies were carried out in accordance with the Declaration of Helsinki. The Institutional Review Board of the University of Minnesota approved the studies and the consent statements. Study participants provided voluntary written informed consent. Not all subjects underwent every assessment as a variety of measures were added during the past nine years (degrees of freedom reflect the total sample for a particular measure).

Assessments

Diagnosis—The diagnosis of pathological gambling was made using the Structured Clinical Interview for Pathological Gambling (SCI-PG). The SCI-PG, which is based on DSM-IV criteria and includes exclusion criteria for gambling secondary to hypomania, mania, or antisocial personality disorder (Grant et al., 2004).

Clinical Characteristics—All subjects underwent a semi-structured clinical interview with a board-certified psychiatrist to assess clinical characteristics of PG. Clinical questions assessed age at onset of gambling, frequency of gambling, triggers to the behavior, and consequence of gambling.

Severity of Pathological Gambling—The severity of gambling was assessed at initial evaluation using the Clinical Global Impression - Severity scale (CGI) (Guy, 1976). The CGI consists of a 7-item Likert scale used to assess severity in clinical symptoms. The CGI severity scale ranges from 1 = “not ill at all” to 7 = “among the most extremely ill.”

The clinician-administered Yale-Brown Obsessive-Compulsive Scale modified for Pathological Gambling (PG-YBOCS) was also obtained (Pallanti et al., 2005). This is a 10-question scale that rates gambling symptoms over the preceding seven days, on a severity scale from 0 to 4 for each item (total scores range from 0 to 40 with higher scores reflecting greater illness severity). There are two subscales, each with five questions: urges/thoughts to gamble and gambling behavior.

Subjects reported their symptom severity using the Gambling Symptom Assessment Scale (G-SAS), a 12-item self-report scale used in previous studies (Kim et al., 2009). The G-SAS assesses gambling urges, thoughts, and behaviors during the previous seven days.

Clinical Assessments—Sheehan Disability Scale (SDS) (Sheehan, 1983). The SDS is a three-item self-report scale that assesses functioning in three areas of life: work, social or leisure activities, and home and family life. Scores on the SDS range from 0 to 30.

Hamilton Anxiety Rating Scale (HAM-A) (Hamilton, 1959). The HAM-A is a clinician-administered, 14-item scale that provides an overall measure of global anxiety.

Hamilton Depression Rating Scale (HAM-D) (Hamilton, 1960). The HAM-D is a 17-item, clinician-administered rating scale assessing severity of depressive symptoms.

The Quality of Life Inventory (QoLI) (Frisch et al., 1993), a 16-item self-administered rating scale, assesses the domains of health, work, recreation, friendships, love relationships, home, self-esteem and standard of living.

Family history of psychiatric conditions, including substance use/abuse, was obtained from subjects. Family history data were collected from interviews with the probands and no first-degree relatives were interviewed directly.

Comorbid Psychiatric Disorders—Each subject was evaluated with the Structured Clinical Interview for DSM-IV Axis-I and Axis-II (SCID-P and SCID-II) (First et al., 1995; First et al., 1997) to assess current and lifetime comorbid disorders. SCID-compatible modules were used to examine current and lifetime rates of other impulse control disorders.

Cognitive Assessments

A sub-sample (n=77) of subjects underwent neurocognitive testing assessing cognitive flexibility (Intradimensional/Extradimensional Set-Shifting Task) and motor inhibition (Stop-Signal Task) using the Cambridge Neuropsychological Test Automated Battery (CANTAB) (Cambridge Cognition, 2006).

Intra-dimensional/Extra-dimensional Set Shift task (IDED)—The IDED task was derived from the Wisconsin Card Sort Test (Lezak et al., 2004), and examines aspects of rule learning and flexible behavior. There are nine stages to the task, requiring different components of set acquisition, reversal, and flexibility. Intradimensional (ID) shifting involves keeping one's attention on the same previously relevant stimulus dimension while extradimensional (ED) shifting involves cognitive flexibility, or shifting attention away from a previously relevant dimension towards a dimension that was previously irrelevant. The primary outcome measure on the task is the total numbers of errors made, adjusted for stages not completed. Secondary outcome measures are total errors on the intra-dimensional (ID) and extra-dimensional (ED) stages.

Stop-signal test (SST)—The Stop-signal test is a well-validated task quantifying the ability to suppress impulsive premature responses (Logan et al., 1984; Aron et al., 2004). This task provides a sensitive estimate of the time taken by the subject's brain to stop a prepotent response, referred to as the 'Stop-Signal Reaction Time' (SSRT, the primary outcome measure). Median reaction times for go trials are also recorded (secondary outcome measure).

Statistical Analysis

274 female subjects were compared with the 227 male subjects with regard to demographic features, clinical characteristics of PG, symptom severity, psychosocial functioning, treatment history, comorbidity, and cognitive functioning (for the latter, data from a subset of the sample only were available). Between-group differences were tested using t-test, or

chi-square as appropriate. The tests were 2 tailed with an α level of .05. This being an exploratory study with a priori hypotheses where the interest lay equally in determining no difference in features between groups versus there being differences in features between groups, p values were uncorrected. Where effects of group were significant, we also report effect size (ES) estimates, which were determined for t tests with Cohen's d ($d = 0.2$ is a small effect size, 0.5 is medium, and 0.8 is large) and for χ^2 with ϕ coefficient (Cramer's V) ($V = 0.1$ is considered a small effect size, 0.3 is medium, and 0.5 is large).

RESULTS

501 adults ($n=274$ [54.7%] females) with PG were included in this study. The comparison of males and females on demographic characteristics is provided in Table 1, where it can be seen that male gamblers were significantly younger.

Clinical characteristics relating to gambling symptoms are summarized in Table 2. In comparison to males, females showed worse disease severity (PG-YBOCS total and urge/thought subscale scores; Gambling Symptom Assessment Scale scores; CGI). Males reported a significantly earlier age of first gambling and earlier age of disease onset; they also showed a significantly greater proportion of strategic-type gambling (i.e., games in which the gambler attempts to use knowledge of the game to influence or predict the outcome such as poker, blackjack, dog and horse racing, sports betting, and craps/dice games) than females. Females were significantly more likely to report that a mood state (i.e., depressive symptoms, loneliness, boredom, anxiety) was a primary trigger to their gambling episodes. There were no gender differences in terms of the proportionate expression of different problems due to gambling. Males and females did not differ significantly in illegal activity used to fund gambling, nor in terms of history of different gambling treatments.

Clinical characteristics relating to co-morbidities, mood, and psychosocial functioning are shown in Table 3. Compared to males, females showed significantly higher current depression scores and higher anxiety scores. Females also showed significantly higher lifetime occurrence of affective disorders, but significantly lower lifetime occurrence of any alcohol use disorder and any substance use disorder, than men. The likelihoods of first-degree relatives having a gambling problem or any alcohol problem were higher in females.

On the neuropsychological measures (Table 4), male and female gamblers did not differ significantly on the primary outcome measures. However, males showed significantly lengthened reaction times for go trials on the stop-signal task. There was a significant correlation between personal history of substance use disorders and the magnitude of median go reaction times across all recruits (Spearman's $r=0.233$, $p=0.041$). This relationship was only significant in the males ($r=0.322$, $p=0.038$), not in the females ($r=-0.085$, $p=0.628$).

The effect of gender on go reaction times appeared not to be influenced by possible differences in medications between males and females since (i) the proportion of males versus females taking psychotropic medication(s) in the cognitive sample did not differ significantly (chi-square 2.311, $df=1$, $p=0.129$); and (ii) exclusion of those taking psychotropic medication(s) yielded average reaction times that were akin to those in the full cognitive sample (means of 546.5msec and 479.3msec for males and females respectively).

DISCUSSION

The present study is one of the largest to date to examine gender-related differences in a large sample of treatment-seeking individuals with PG. Consistent with previous research (Potenza et al., 2001; Ladd & Petry, 2002; Grant & Kim, 2002; Desai & Potenza, 2008),

men were more likely to start gambling at a younger age (small effect size, 0.36) and to prefer strategic forms of gambling (medium effect size, 0.61) (Table 2). In contrast, women were more likely to report depressive and anxiety symptoms, and to have a history of an affective disorder (small effect sizes, 0.20–0.35, Table 3). Interestingly, although men were younger overall (small effect size, 0.24), the lag time between initial gambling and the onset of PG did not differ between men and women.

Our first hypothesis - that men would report longer time of progression of illness and display more problems in areas characterized by impaired impulse control, including more severe PG symptoms and greater rates of substance use disorders - was only partially supported. In fact, this study found that men and women progressed to PG within the same general time frame, and that females reported more severe gambling symptomatology (with even a statistical trend toward more illegal behaviors secondary to gambling). Although previous research in PG has shown that women progress more rapidly to addiction after starting a behavior (i.e., the “telescoping phenomenon”) (Potenza et al., 2001), there was no statistically significant difference found in this study between the groups with respect to progression from onset of gambling to developing PG. Women compared with men reported a numerically shorter timeframe (~9 years from onset of gambling in women compared with ~10 years in men) but this was not statistically significant. One explanation could be that both current strategic and non-strategic forms of gambling lead rapidly to problematic use. Like other risk-taking behaviors (substance use), gambling may require early intervention for both men and women with PG.

Women with PG reported more severe PG symptoms on several instruments versus men (small effect sizes, 0.26–0.30) and this finding is somewhat inconsistent with previous research which has found that men and women generally have not differed based on gambling expenditures and days gambled (Ladd & Petry, 2002). Interpretation of this finding, however, is complicated by how one defines severity of gambling behavior. Previous research has found that women report worse gambling behavior based on certain measures (e.g., Addiction Severity Index gambling composite scores; Ladd & Petry, 2002), but that women and men with PG appear to have equivalent but perhaps unique dysfunctional consequences due to gambling (e.g., females report more debt related to gambling while men report more gambling-related arrests; Potenza et al., 2001). Although a consensus on how one measures gambling severity is perhaps still unsettled, these findings emphasize the need to examine multiple domains of dysfunction related to gambling and how gender impacts areas differently.

Lifetime substance use disorders were more frequently reported by men with PG compared with affected women (small effect size 0.22), and this finding was expected. High rates of alcohol use among pathological gamblers have been previously identified (Ladd & Petry, 2002; Blanco et al., 2006), and previous research suggest that men with PG are more likely to report a history of substance abuse. Although this finding might be in part attributable to general gender differences, it might also represent “switching” of addictive behaviors in men. The findings suggest a complex relationship among multiple addictive disorders in men, and a more complete understanding of the temporal progressions and inter-relationships would help clinicians target interventions.

From a cognitive perspective, no significant differences in response inhibition and cognitive flexibility were identified between men and women with PG. These two domains have previously been shown to be impaired in PG versus healthy controls, even in the absence of co-morbidity (Odlaug et al., 2011). Viewed collectively, findings suggest common overlap in terms of underlying dysfunction of neural circuitry mediating these two functions. However, we found that males showed significantly lengthened reaction times on the stop-

signal task for correct 'go' trials versus women – this measure relates not to inhibition but to more general aspects of psychomotor speed. The difference in group means of around 70ms would be of some impact in day-to-day life, and the reasons for it are unclear. It may be that this slowing of reaction times was due, at least in part, to the elevated rates of chronic substance use likely to have been evident in the male patients; in support of this view, we found significant correlation in men only between this measure and personal history of substance use disorders. Numerically, males with a history of substance misuse showed longer median reaction times than those who did not (586ms versus 509ms).

Though this is one of the largest studies of gender influences in treatment-seeking PG to date, several potential limitations should be considered. Significance was defined a priori as $p < 0.05$ uncorrected, therefore significant findings merit replication in future work. The majority of gender differences identified were of small effect size, and therefore their clinical importance may be limited. Exceptions were the finding of proportionately more non-strategic gambling in women and slower motoric reaction times in males, which were of medium effect size and may therefore be clinically more important. The study was based on pooling of data from subjects across research trials conducted over a number of years. In general, these trials recruited participants with PG from the community using advertisements and word of mouth and similar enrollment criteria; it is not clear whether the findings would generalize to individuals presenting to their general practitioners or to routine clinics *per se*, and to non treatment-seeking individuals. By virtue of such a design, data for all variables were not available for all participants. Nonetheless, sample sizes were large even where there were attrition of data. For pragmatic (time) reasons, only two cognitive tasks were included and only in a subset of participants: however, these tasks were selected based on their extensive validation across clinical populations. Evaluation of other cognitive tasks in future studies would be valuable, such as decision-making/gambling paradigms dependent on orbitofrontal integrity. It is not clear whether clinical/cognitive features associated with men versus women stem from PG expression itself or other potential confounds – for example, males studied here were significantly younger. We did not attempt to co-vary for such potential confounds since we wished to study – from a pragmatic perspective – whether men and women differed in their actual presentation when attending for research trials. The possibility also exists that some of the findings were mediated by confounds that were not recorded – though this is a problem afflicting all such studies. Finally, not all subjects for whom cognitive data were available were drug free. The difference in go reaction times between men and women appeared to be unaffected by drug status, since average performances were similar after excluding people taking one or more medication(s). We acknowledge though that the issue of medication influences over cognition is an important one, but not one that this study was designed to address.

In summary, this study identified common and distinct characteristics of PG in men compared to in women, with small to medium effect sizes. The findings from this study, while confirming some previous research results, have also called into question other commonly held beliefs about gender differences in PG (e.g., telescoping phenomenon). In addition, questions remain as to whether the nature of these gender differences is due to the phenomenon of PG itself, societal and developmental influences, or a dynamic combination of each. A more detailed understanding of gender-specific variables in PG may assist in developing strategies for prevention and treatment.

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Table 1

Demographics of Individuals with Pathological Gambling

Demographic	Female	Male	Statistic	df	p-value [Effect Size]
Age Mean ($\pm$ SD), years	48.7 (11.2)	45.9 (11.7)	2.657 ^t	499	0.008 [0.24]
Ethnicity, n (%) Caucasian African-American Other	239 (92.6) 10 (3.9) 9 (3.5)	195 (89.9) 8 (3.7) 14 (6.5)	2.25 ^c	2	0.325
Education, n (%) High school or less Some college College degree or more	103 (39.0) 86 (32.6) 75 (28.4)	87 (41.0) 65 (30.7) 60 (28.3)	0.26 ^c	2	0.878
Marital Status, n (%) Single Married/living together Divorced/Separated/Widowed	77 (28.5) 119 (44.1) 74 (27.4)	81 (36.3) 97 (43.5) 45 (20.2)	4.97 ^c	2	0.083

All items are n (%) unless otherwise noted

Statistic: c=Chi-Square; t=t-test

Table 2
Clinical Comparison of Gambling Symptoms of Women and Men with Pathological Gambling

Clinical Variable	Female	Male	Statistic	df	p-value [Effect Size]
PG-YBOCS					
Urge/Thought Subscale	10.71 (3.18)	9.81 (2.89)	2.525t	296	0.012 [0.30]
Behavior Subscale	10.48 (3.82)	10.03 (3.61)	1.044t		0.297
Total Score	21.19 (5.7)	19.79 (5.19)	2.208t		0.028 [0.26]
Gambling Symptom Assessment Scale	36.58 (13.28)	32.84 (11.37)	2.904t	371	0.004 [0.30]
Clinical Global Impressions-Severity	4.88 (0.85)	4.62 (0.86)	3.080t	427	0.002 [0.30]
Age of 1st Gambling	31.61 (12.86)	26.55 (14.91)	3.765t	426	<0.001 [0.36]
Age of Disease Onset	40.19 (11.56)	36.21 (14.05)	3.081t	391	0.002 [0.31]
Lag Time Between 1st Gambling and PG Onset	8.68 (9.67)	9.71 (10.64)	0.981t	390	0.326
Primary Form of Gambling, n (%) *	18 (8.3)	47 (31.3)	42.37c	2	<0.001 [0.61]
Strategic	179 (82.5)	78 (52.0)			
Non-Strategic	20 (9.2)	25 (16.7)			
Both					
Triggers for gambling, n (%) **	91 (33.2)	66 (29.1)	0.99c	1	0.32
Having money	209 (76.3)	116 (51.1)	34.53c	1	<0.001 [0.54]
Mood state	69 (25.2)	48 (21.1)	1.13c	1	0.288
Advertisements					
Problems secondary to gambling, n (%) **	173 (63.1)	145 (63.9)	0.03c	1	0.862
Credit Card Debt	156 (57.0)	133 (58.6)	0.14c	1	0.708
Borrowing Money	38 (13.9)	25 (11.0)	0.92c	1	0.337
Pawning Possessions	96 (35.0)	86 (37.9)	0.44c	1	0.507
Payday Loans	91 (33.2)	72 (31.7)	0.13c	1	0.718
Loss of Savings	36 (13.1)	43 (19.0)	3.15c	1	0.076
Marital Problems	56 (20.4)	36 (15.9)	1.74c	1	0.187
Work Problems	62 (22.6)	41 (18.1)	1.58c	1	0.209
Bankruptcy					
Illegal Activity to fund gambling, n (%)	77 (28.1)	58 (25.6)	0.41c	1	0.522
Bad checks / Forging	18 (6.6)	6 (2.6)	4.2c	1	0.040 [0.18]
Theft/Embezzlement					
History of gambling treatment, n (%)	91 (63.4)	72 (73.5)	2.69c	2	0.260
Gamblers Anonymous	38 (26.6)	20 (20.4)			
	14 (10.0)	6 (6.1)			

Clinical Variable	Female	Male	Statistic	df	p-value [Effect Size]
Outpatient therapy					
Inpatient treatment					

All results are mean ± SD unless otherwise noted

Statistic: c=Chi-Square; |t=t-test

Abbreviations: PG- YBOCS= Yale-Brown Obsessive Compulsive Scale modified for Pathological Gambling

* Strategic gambling = poker, sports, cards, dice, blackjack, track (horses, dogs)

Non-strategic gambling = slots, lottery, pull tabs, bingo, keno, video poker

** categories not mutually exclusive – percentages refer to percentage of sample endorsing problem – hence percentages may tally to >100% across categories

Table 3

Clinical Comparison of Women and Men with Pathological Gambling

Clinical Variable	Female n=274	Male n=227	Statistic	df	p-value [Effect Size]
Sheehan Disability Scale, total score	14.51 (7.48)	14.92 (6.39)	0.284t	296	0.777
Hamilton Depression Scale, total score	8.21 (4.37)	7.05 (4.25)	2.698t	406	0.007 [0.30]
Hamilton Anxiety Scale, total score	8.37 (4.72)	6.79 (4.38)	3.449t	406	<0.001 [0.35]
Psychiatric Hospitalizations, n (%)	10 (3.6)	11 (4.8)	0.44c	1	0.655
Current Tobacco Use, n (%)	99 (36.1)	78 (34.4)	0.17c	1	0.708
Lifetime Psychiatric Disorders, n (%)					
Any affective disorder	103 (37.6)	64 (28.2)	4.93c	1	0.028 [0.20]
Any anxiety disorder	38 (13.9)	32 (14.1)	0.02c	1	1
Any alcohol use disorder	36 (13.1)	64 (28.2)	17.61c	1	<0.001 [0.38]
Any substance use disorder	30 (10.9)	42 (18.5)	5.76c	1	0.021 [0.22]
Any impulse control disorder	26 (9.5)	20 (8.8)	0.07c	1	0.877
Any personality disorder	54 (19.7)	43 (18.9)	0.05c	1	0.91
First Degree Relatives, n (%)					
Gambling problem	135 (49.3)	87 (26.1)	6.03c	1	0.015 [0.22]
Any alcohol problem	153 (55.8)	97 (42.7)	8.53c	1	0.004 [0.26]

All results are mean $\pm$ SD unless otherwise noted

Statistic: c=Chi-Square; t=t-test.

Table 4

Performance on Cognitive Tasks by Gender

	Performance (mean $\pm$ SD)		
	Female <i>n</i> =35	Male <i>n</i> =42	p-value
IDED total errors (adjusted)	25.89 (20.05)	31.59 (22.14)	0.243
ID Shift	0.51 (0.61)	0.74 (1.17)	0.311
ED Shift	10.17 (9.66)	13.29 (10.08)	0.173
SST SSRT	197.3 (80.6)	187.3 (64.4)	0.546
SST median go reaction time	473.3 (104.6)	542.1 (162.1)	0.034 [0.50]

All data are in milliseconds: Mean (SD)

T = t-test: two sample assuming equal variances

P = two-tailed t-test

Abbreviations: IDED = Intradimensional Extradimensional; SST = Stop Signal Task; SSRT = Stop-Signal Reaction Time