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Racial-ethnic Related Clinical and Neurocognitive Differences in Adults with Gambling Disorder

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Abstract

Recent epidemiological data suggest that the lifetime prevalence of gambling problems differs depending on race-ethnicity. Understanding variations in disease presentation in blacks and whites, and relationships with biological and sociocultural factors, may have implications for selecting appropriate prevention strategies. 62 non-treatment seeking volunteers (18-29 years, n=18 [29.0%] female) with gambling disorder were recruited from the general community. Black (n=36) and White (n=26) participants were compared on demographic, clinical and cognitive measures. Young black adults with gambling disorder reported more symptoms of gambling disorder and greater scores on a measure of compulsivity. In addition they exhibited significantly higher total errors on a set-shifting task, less risk adjustment on a gambling task, greater delay aversion on a gambling task, and more total errors on a working memory task. These findings suggest that the clinical and neurocognitive presentation of gambling disorder different between racial-ethnic groups.

Keywords

addiction; cognition; gambling; impulsivity; phenomenology; race

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Contributors

Samuel Chamberlain: Dr. Chamberlain undertook statistical analyses and drafted the manuscript.

Sarah Redden: Ms. Redden collected the data and assisted in drafting the manuscript.

Eric Leppink: Mr. Leppink collected the data and assisted in drafting the manuscript.

Brian Odlaug: Dr. Odlaug collected the data and assisted in drafting the manuscript.

Jon Grant: Dr. Grant designed the study, collected the data and drafted the manuscript.

1.0 Introduction

Gambling is a commonplace activity across cultures, and in extreme forms, can evolve into Gambling Disorder, a behavioral problem characterized by persistent, recurrent maladaptive patterns of gambling behavior and functional impairment (APA, 2013). Recent research that examined racial-ethnic differences in lifetime prevalence of problem gambling found higher rates among black (2.2%) compared to white (1.2%) respondents (Alegria et al., 2009). Health initiatives have highlighted the importance of understanding racial-ethnic differences (<http://minorityhealth.hhs.gov>). In terms of clinical presentation, there is considerable heterogeneity across individuals with gambling problems (Hodgins et al., 2011), and a small but growing number of studies have found that significant clinical differences exist between African-American and White gamblers (Barry et al., 2008, 2011; Sacco et al., 2011). For example, African-American low- or at-risk gamblers appear more likely to report a co-occurring mood disorder, hypomania, or any substance use disorder (Barry et al., 2011). Other research has shown that African-American gamblers appear more likely to endorse trying to cut back on their gambling and more likely to endorse the suffering of losses (Sacco et al., 2011). Telephone callers to a gambling helpline who were African-American were more likely than white callers to report longer durations of gambling problems but less likely to report daily tobacco use or mental health treatment (Barry et al., 2008).

Several factors may influence race or ethnic differences in the development or presentation of gambling disorder, including environmental and clinical factors. Previous research on race-ethnicity and gambling has shown that environmental factors such as low-income and living in a disadvantaged neighborhood influence the development of psychiatric problems, including problematic gambling behavior, in children and adolescents (Martins et al., 2013). In addition to exploring these sociodemographic variables or overt clinical presentations with respect to race or ethnicity, however, it is potentially important to also consider the underpinning neurobiological factors such as cognition. This accords with the recent shift towards Research Domain Criteria (RDoC), proposed by the National Institute for Mental Health (NIMH), which seeks to improve classification and treatment of mental disorders by considering dimensions including cognition (Insel et al., 2010). Previous research demonstrated that individuals with gambling disorder demonstrate cognitive impairments relating to several aspects of decision-making (van Holst et al., 2010a). The finding of decision-making deficits in people with gambling disorder fits with neurobiological models of gambling disorder and findings from the broader cognitive literature (Clark, 2010; van Holst et al., 2010a; van Holst et al., 2010b). Unfortunately, racial-ethnic influences over cognitive function in relation to gambling disorder have yet to be explored. Common cognitive problems between races-ethnicities may be suggestive of common neural dysfunction while distinct deficits may be suggestive of differential pathophysiology.

Given the unknown influence of cognition on gambling pathophysiology, we sought to examine potential cognitive differences between young black and white adults with gambling disorder. Based on previous research suggesting higher rates of gambling problems among African-Americans, as well as greater comorbidity with mood and substance use disorder (Barry et al., 2011), our hypothesis was that black gamblers would

report more severe gambling symptoms and relative impairment with respect to inhibitory control, working memory, and decision-making.

2.0 Methods

2.1 Subjects

Participants comprised non-treatment-seeking young adults aged 18-29 years, recruited as part of a longitudinal study of impulsive behaviors. Subjects were self-selected in response to media announcements in a metropolitan area (“have you ever gambled?”). Inclusion criteria were: gambling in any form at least five times during the preceding 12-months, and presence of subsyndromal gambling disorder (for definition see below). Exclusion criteria included an inability to understand/undertake the procedures and to provide written informed consent, and presence of formal gambling disorder (as opposed to at-risk gambling disorder – see below). Since we sought to examine a naturalistic sample, subjects with psychiatric and substance use comorbidity, as well as those subjects currently taking psychotropic medications, were all allowed to participate.

The study procedures were carried out in accordance with the Declaration of Helsinki. The Institutional Review Board of the University of Chicago approved the study and the consent statement. After all study procedures were explained, subjects provided voluntary written informed consent. Participants were compensated with a \$50 gift card for a local department store.

2.2 Clinical Assessments

Raters assessed each participant using the Mini-International Neuropsychiatric Interview (Sheehan et al., 1998) to examine psychiatric morbidity; and the modified Structured Clinical Interview for Gambling Disorder (SCI-GD) (Grant et al., 2004), which is a nine-item instrument assessing symptoms of gambling disorder (consistent with DSM-5): a score of 4 indicates current gambling disorder (Grant et al., 2011). Participants were assessed for the frequency of gambling behavior as well as money lost gambling using a timeline follow-back method for gambling (Weinstock et al., 2004). In addition, participants completed the Padua Inventory, a 60-item questionnaire originally developed to study obsessive-compulsive thoughts and behaviors in the general population (Sanavio, 1988). It yields a total score, derived on the basis of factor analysis.

2.3 Cognitive Assessments

Cognitive testing was undertaken in quiet room using a touch-screen computer, with a trained assessor present. We utilized tests selected from the Cambridge Neuropsychological Test Automated Battery (CANTAB). The cognitive domains of interest were response inhibition, working memory, and decision-making. We focused on these areas after considering the existing literature, these being domains commonly implicated in the pathophysiology of gambling symptomatology (e.g. Clark, 2010; van Holst et al., 2010a; van Holst et al., 2010b; Grant et al., 2011; Odlaug et al., 2011).

We assessed response inhibition using the Stop-Signal Task (Aron et al., 2004), a paradigm in which the subject viewed a series of directional arrows appearing one per time on-screen, and made speeded motor responses depending on the direction of each arrow (left button for a left-facing arrow, and vice versa). On a subset of trials, an auditory stop-signal occurred ('beep') to indicate to volunteers that response suppression was needed for the given trial. This task uses a dynamic tracking algorithm to calculate the 'stop-signal reaction time', which is an estimate of the time taken by the given volunteer's brain to suppress a response that would normally be undertaken. The task also recorded median 'go' reaction times, which is the average response latency for trials not involving a stop-signal; this is a measure of general 'response speed' rather than inhibitory control.

All subjects completed the Spatial Working Memory task (SWM) (Owen et al., 1990). On the SWM test (8-box version), participants attempt to locate tokens hidden underneath boxes on-screen and try to avoid returning to boxes that previously yielded such tokens. The key outcome measures include the "total number of errors" (inappropriately returning to boxes that previously yielded tokens) and "strategy score" (lower score equates to more optimal strategy use).

Different aspects of decision-making were quantified using the Cambridge Gambling Task (CGT) (Rogers et al., 1999). There were four practice trials followed by eight blocks of nine trials. At the start of each block, the 'cumulative points' setting on the task was reset to 100. On each trial, subjects were shown a set of red and blue boxes, totaling ten. The ratio of red:blue boxes were varied over the course of the task pseudo-randomly (box-ratios: 9_1, 8_2, 7_3, 6_4). Subjects were informed that for each trial, the computer had hidden a 'token' inside one of the boxes, and that they had to indicate whether they felt the token would be hidden behind a red or a blue box. This choice was made by selecting 'red' or 'blue' using the touch-screen interface. After making this judgment, subjects were required to gamble a proportion of their points on whether their color choice was correct. The key outcome measures were (i) mean proportion of points gambled; (ii) quality of decision-making (the proportion of trials where the volunteer chose red when red boxes were in the majority and vice versa – i.e. made the logical color choice); and (iii) risk adjustment (tendency to adjust how many points are gambled depending on the degree of risk).

2.4 Data Analysis

Subjects meeting criteria for gambling disorder were grouped into two categories based on race-ethnicity: blacks and whites. Potential differences in demographic, clinical and cognitive variables between the groups were explored using three separate multivariate analyses of variance (MANOVAs); this approach provided protection against false positive errors, in that individual variables were only explored when the broader category of interest (demographic, clinical, or cognitive) showed a main effect of group. Where a composite MANOVA test obtained statistical significance, differences between groups were explored for individual variables within the given analysis. Demographic variables found to differ significantly between groups were entered as covariates into subsequent MANOVAs. Statistical significance was defined as $p < 0.05$ uncorrected. IBM SPSS Software, Version 19 was used for the analyses. For MANOVA, data were checked for normality and other

assumptions using Box's M, tests for linearity (inter-variable correlations), Levine's tests, and Mahalanobis distances. At the level of individual test measures, data were inspected for violation of assumptions including normality using Shapiro Wilk tests and visual displays; any significant findings at the level of individual variables, where there was any evidence of violation of model assumptions, were confirmed or refuted using non-parametric tests as appropriate.

We also undertook secondary analysis using correlation (Spearman's) tests, to explore possible relationships between clinical measures and cognitive measures; because of the number of tests entailed, and the exploratory nature of these tests, we corrected for multiplicity by using a significance threshold of $0.05/42 = 0.0012$ (42 correlational tests).

3.0 Results

The sample included 62 (18 [29.0%] female) young adults with gambling disorder. A total of 36 black and 26 white gamblers were included. MANOVA for demographic variables indicated that the two groups differed overall ($F=2.725$, $df\ 1,55$, $p=0.022$). As can be seen in Table 1 (top section), this was due to the black gamblers earning significantly more dollars in the preceding year than the white gamblers. This variable was taken forward as a covariate into subsequent analyses.

The MANOVA for the clinical variables demonstrated that the groups differed on these measures overall ($F=3.270$, $df\ 1,52$, $p=0.006$). As shown in Table 1 (bottom section), that was due to the black gamblers showing significantly higher gambling frequency per week, higher SCI-PG scores, and higher Padua total scores, as compared to the white gamblers.

In terms of cognitive measures, the two study groups differed overall on the MANOVA ($F=2.881$, $df\ 1,49$, $p=0.010$). This was due to the black gamblers, as compared to white gamblers, exhibiting significantly higher IDED total errors (adjusted), less CGT risk adjustment, higher CGT delay aversion, and more SWM Total errors (Table 2).

The two study groups did not differ in terms of occurrence of mainstream mental disorders, as shown in Table 3. In the exploratory correlational analysis, when the whole sample was considered, there was a significant negative correlation between CGT risk adjustment and SCIPG total scores ($r=-0.452$, $p<0.0001$ uncorrected). This relationship did not remain statistically significant at the corrected threshold when each group was considered separately.

4.0 Discussion

To our knowledge, this is the first study to assess clinical and cognitive differences between black and white young adults with gambling disorder. We found important clinical and cognitive differences between groups which merit further exploration.

We found some evidence that black gamblers exhibited higher levels of gambling symptomatology compared to whites: they met more diagnostic criteria for gambling disorder, and had higher gambling frequency per week. However, groups did not differ

significantly in terms of PG-YBOCS total scores, or the amount of money lost to gambling per year, suggesting that these clinical differences did not map onto worse longer-term symptom severity or outcomes. Contrary to other research which has found that black people report longer duration of gambling problems prior to seeking help (Barry et al., 2008), measures of current gambling severity in our study showed no differences. Whether race-ethnicity may play a larger role in symptom severity in those with lower levels of gambling behavior, however, awaits further research.

We found that black gamblers in our sample exhibited higher scores on the measure of obsessive-compulsiveness (Padua) compared to white gamblers; these findings were contrary to our *a priori* predictions.

From a cognitive point of view, people with gambling disorder have been found to experience impairments across a range of neuropsychological domains. In recent reviews of the literature, cognitive domains often implicated in gambling disorder have included measures of impulsivity (e.g. stop-signal, go/no-go, and temporal discounting), decision-making, and cognitive flexibility (e.g. set-shifting) (Goudriaan et al., 2004; Clark 2010; Goudriaan et al., 2014; Chamberlain et al., 2015). It is interesting to consider, therefore, whether these types of cognitive functions differ between racial-ethnic groups in people with gambling disorder.

In terms of impulsivity, our two racial-ethnic groups did not differ on the SST, a task of motor impulsivity. Previous research, using different methodologies to assess impulsivity, reported higher ratings of impulsivity in African-American youth gamblers (Martins et al., 2008). The Martins et al. (2008) study, however, used parental and teacher report of childhood impulsivity in a sample of adolescence. The impulsive behaviors they witnessed may have little if any relationship to the types of impulsivity we measured in this study. We did not measure temporal discounting in our study as such.

On other cognitive tests, we found an intriguing dissociation: the black gamblers showed relative impairment, versus white gamblers, in terms of cognitive flexibility (set-shifting task), aspects of decision-making (gambling task), and spatial working memory. In addition, lower risk adjustment correlated with higher SCI-PG scores across the whole sample, suggesting that this parameter may be particularly sensitive to the state versus trait aspects of the disorder. The prognostic value of such neurocognitive functions in gambling disorder has received limited research attention to date. In one study (n=22 cases), people with gambling disorder who relapsed over the course of one year did not differ from non-relapsers in terms of baseline neurocognitive performance (Iowa Gambling, Stroop, and Delay Discounting paradigms) (De Wilde et al., 2013). In another 1-year study, in 157 participants with subsyndromal gambling problems, baseline cognitive function (measured using the same tests used in the current study) did not differ significantly between those individuals whose symptoms remitted versus persisted (Grant et al., 2014). In a study involving 113 patients with gambling disorder assigned to psychological or psychoeducational treatment, reassessed 6-months later, improvements in decision-making (Iowa Gambling task) performance appeared related to higher chance of symptomatic recovery (Rossini-Dib et al., 2015). Lastly, in a study of 46 pathological gamblers, relapse by approximately one-year

was significantly predicted by higher baseline disinhibition (Stop-Signal task), worse decision-making (Card Playing task), and longer duration of disorder; the combination of these variables explained 53% of variance in relapse (Goudriaan et al., 2008). Collectively, these studies suggest that decision-making problems, and potentially other types of cognitive impairment, may have predictive value in terms of outcomes in gambling disorder. As such, our finding that decision-making differs as a function of racial-ethnic differences in gambling disorder may well have differential prognostic implications. This needs to be studied in future work.

While the extant research has not examined race as it relates to potential cognitive markers in young adults with gambling disorder, previous research has looked at risk factors associated with cognitive dysfunction in children. In a sample of 109 children employing cognitive testing, Martell (2013) found that race-ethnicity and low income were risk factors for ADHD and executive function deficits (Martell, 2013). Research into racial-ethnic differences in older adults, however, is conflicting with one study noting ethnic differences in terms of risk-taking (or 'delay') tasks (Sloan & Wang, 2005) while another study noted no significant neurocognitive differences between African-American and European-American participants (Baird et al., 2007). Coupled with the current study, these studies all had significantly different age cohorts and methodologies, limiting comparisons.

Thus, we emphasize the need to consider race research relating to gambling disorder, including treatment studies. If one regards these neurocognitive markers as candidate 'treatment targets' for people with gambling disorder, it is likely that they could be ameliorated via different psychological and pharmacological interventions.

There are several limitations to the current study. The sample size was relatively small – however, it was sufficiently powered to detect important clinical and cognitive differences; we feel that the study also has merit in view of the lack of data in this field, and we hope it will provide a springboard for more substantive work. Because of the sample size, and since post hoc tests were protected at the level of composite multivariate analysis of variance tests, we did not correct for multiple comparisons for our primary analyses. As such, some significant findings may reflect false positives. We did not assess all socio-economic variables in the current study, although education, employment and income were examined. The white gamblers had lesser income than black gamblers, but the groups did not differ on other socio-economic measures, and income per year was controlled for in the statistical analyses for clinical and cognitive measures.

Environmental factors such living in a disadvantaged neighborhood may have influenced gambling behavior (Martins et al., 2013). The longitudinal study conducted by Martins and colleagues (2013), however, reported on a slightly younger population, including children and adolescents, and may not be directly comparable to our sample of young adult gamblers. The current study did not include a non-gambling control group; as such, it is possible (although in our view unlikely) that group differences could be due to general race-ethnic related effects, rather than different presentation of clinical features as a function of race-ethnicity in people with gambling disorder. Information on medication status was not collected, nor was intelligence quotient. Future studies, using a longitudinal design and

variety of socio-demographic, clinical, and cognitive paradigms, may further our understanding of the influence of environmental and neurobiological factors in the development of gambling addiction.

These findings indicate that clinical and cognitive factors associated with gambling disorder may differ depending on race-ethnicity. Future work should address whether group differences are pre-disposing or rather a consequence of gambling disorder, an issue that cannot be addressed within the confines of the current study design. Understanding the potential differences in decision-making may aid in the development of early intervention for individuals with gambling disorder pathology. These initial data emphasize the need to further explore race-ethnicity in relation to neurobiological models for gambling.

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Table 1
Demographic and Clinical Characteristics of Blacks and Whites with Gambling Disorder,
Results from MANOVAs

	Blacks <i>n</i> =36	Whites <i>n</i> =26	Statistic	df	p-value
Demographic variables					
Age, years	24.9 (2.9)	23.9 (3.7)	1.409	1,58	.240
Female, <i>n</i> (%)	13 (36.1)	5 (19.2)	2.088c	1	.148
Education, <i>n</i> (%)			5.057c	4	.281
Less than high school	1 (2.8)	2 (7.7)			
High school degree or GED	10 (27.8)	1 (3.8)			
Some college	19 (52.8)	15 (57.7)			
College Graduate	5 (13.8)	8 (30.8)			
Post-Graduate	1 (2.8)	0 (0)			
Marital Status, <i>n</i> (%)			4.285c	3	0.232
Single	31 (86.1)	21 (80.8)			
Married	3 (8.3)	0 (0)			
Divorced/Separated	0 (0)	4 (15.4)			
Living together or engaged	2 (5.6)	1 (3.8)			
Employment, <i>n</i> (%)			6.029c	4	.197
Works full-time	11 (30.6)	6 (23.1)			
Works part-time	10 (27.8)	3 (11.5)			
Student	4 (11.1)	6 (23.1)			
Student and employed	1 (2.8)	6 (23.1)			
Unemployed	10 (27.8)	5 (19.2)			
Income for the past year, dollars	27779.08 (18866.58)	18096.15 (14271.66)	4.839&	1,58	.032
Clinical Variables					
Age at onset of first gambling, years	13.9 (4.0)	14.8 (4.7)	1.181	1, 58	.282
Gambling frequency per week, times per week	5.2 (3.7)	3.3 (2.3)	5.884&	1, 58	.018
Amount lost to gambling for the past year, dollars	5777.08 (6694.99)	5130.77 (8705.33)	0.361	1, 58	.550
SCI-PG scores#	6.4 (1.7)	5.0 (1.2)	11.250@	1,58	.001
PG-YBOCS total scores	18.7 (8.1)	15.7 (7.7)	1.312	1,58	.257
QOLI t-score	40.7 (15.6)	39.9 (17.8)	0.258	1,58	.613
Padua total score##	40.3 (32.6)	18.3 (11.0)	9.928@	1,58	0.003

SCI-PG=Structured Clinical Interview for Pathological Gambling (modified for gambling disorder and DSM-5); PG-YBOCS=Yale Brown Obsessive Compulsive Scale modified for pathological gambling; QOLI=Quality of Life Inventory

All values are Mean ($\pm$ SD) unless otherwise stated

Statistical tests reported in table are F tests, except where indicated: c=chi-square, with Yates' correction where appropriate.

@ homogeneity of variance violated; group differences remain significant with Welch's test for SCI-PG scores (Welch=14.269, df 1,59.988, $p<0.001$) and Padua total scores (Welch=14.163, df 1,45.355, $p<0.001$); & normality assumptions violated, remains significant with Mann-Whitney for past year income ($p=0.034$); gambling frequency per week ($p=0.015$).

Table 2
Cognitive Characteristics of Blacks and Whites with Gambling Disorder, Results from MANOVA

	Blacks n=36	Whites n=26	Statistic	df	p-value
IED Total errors	43.25 (30.22)	26.0 (21.03)	6.210&	1,59	.016
SST SSRT	176.30 (65.57)	192.14 (61.40)	0.163	1,59	.688
SST median go reaction times	576.14 (210.3)	484.10 (207.6)	1.770	1,59	0.189
CGT Quality of decision making	.905 (.091)	.907 (.106)	0.009	1, 59	.923
CGT Risk adjustment	.577 (.806)	1.358 (1.149)	7.495&	1, 59	.008
CGT Delay aversion	.454 (.246)	.236 (.340)	8.341&	1, 59	.006
SWM Total errors	34.36 (21.39)	22.73 (22.82)	6.621&	1, 59	.013

IED=Intradimensional/Extradimensional Set Shift task; CGT=Cambridge Gambling Task; SWM=Spatial Working Memory task

Statistical tests are F tests.& normality assumptions violated, group difference remains significant with Mann-Whitney for IED Total errors (p=0.013), risk adjustment (p=0.003), delay aversion (p=0.002), and SWM Total errors (p=0.020).

Table 3
Psychiatric Comorbidity in Blacks and Whites with Gambling Disorder

	Blacks n=36	Whites n=25	Statistic	p-value
Comorbid Current Disorders, n (%)				
Any mood disorder	7 (19.4)	2 (8.0)	1.536	.215
Any anxiety disorder	9 (25.0)	7 (28.0)	.069	.793
Post-traumatic stress disorder	2 (5.6)	0 (0)	1.436	.231
Any eating disorder	0 (0)	2 (8.0)	2.978	.084
Any psychotic disorder	2 (5.6)	0 (0)	1.436	.231
Any alcohol use disorder	15 (41.7)	10 (40.0)	.017	.896
Any substance use disorder	15 (41.7)	6 (24.0)	2.040	.153
Any current disorder	24 (66.7)	14 (56.0)	.715	.395

Statistic is chi-square; df=1