Neurocognitive dysfunction in problem gamblers with co-occurring antisocial personality disorder

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Abstract

Objectives: Problem gamblers with symptoms of antisocial personality disorder (ASPD) may represent a distinct problem gambling subtype, but the neurocognitive profile of individuals affected by both disorders is poorly characterized.

Method: Non-treatment-seeking young adults (18–29 years) who gambled ≥5 times in the preceding year were recruited from the general community. Problem gamblers (defined as those meeting ≥1 DSM-5 diagnostic criteria for gambling disorder) with a lifetime history of ASPD (N = 26) were identified using the Mini International Neuropsychiatric Interview (MINI) and compared with controls (N = 266) using questionnaire-based impulsivity scales and objective computerized neuropsychological tasks. Findings were uncorrected for multiple comparisons. Effect sizes were calculated using Cohen’s d.

Results: Problem gambling with ASPD was associated with significantly elevated gambling disorder symptoms, lower quality of life, greater psychiatric comorbidity, higher impulsivity questionnaire scores on the Barratt Impulsiveness Scale (d = 0.4) and Eysenck Impulsivity Questionnaire (d = 0.5), and impaired cognitive flexibility (d = 0.4), executive planning (d = 0.4), and an aspect of decision-making (d = 0.6). Performance on measures of response inhibition, risk adjustment, and quality of decision making did not differ significantly between groups.

Conclusions: These preliminary findings, though in need of replication, support the characterization of problem gambling with ASPD as a subtype of problem gambling associated with higher rates of impulsivity and executive function deficits. Taken together, these results may have treatment implications.

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1. Introduction

Gambling disorder (GD) has been associated with illegal behavior and antisocial personality disorder (ASPD) in multiple country contexts [1–4]. Among people with GD, estimates of criminal activity range from 25% to 43% [5] and 13% to 15% have co-occurring ASPD [6,7]. Demographically, disordered gamblers with ASPD are younger and more likely to be male, divorced, and to have fewer years of education than those without ASPD [8]. They are also more likely to report earlier onset of GD symptoms, greater severity of gambling and substance use problems, and to score higher on measures of paranoid ideation, somatization, and phobic anxiety than problem gamblers without ASPD [8].

Mounting evidence indicates that problem gamblers with antisocial personality traits and behaviors may represent a distinct subtype of GD. Following the development of the Pathways Model by Blaszczynski and Nower [9], recent
conceptual models have subtyped problem gamblers with ASPD symptomology based on their level of psychopathology and an array of personality dimensions, such as novelty seeking and self-directedness [10–12]. Results from these models suggest that problem gambling and antisocial behavior may be motivated by the same personality traits, especially risk acceptance [11,12]. A large twin study has also found evidence of a potential shared genetic vulnerability between GD and ASPD [7]. Other data support that GD and ASPD share a common familial etiology and possibly shared neurocircuitry and environmental risk factors (e.g., childhood adversity) [13].

A complementary way to approach the link between GD and ASPD is by identifying cognitive deficits associated with the disorders. Problem gamblers show cognitive dysfunction across a variety of domains, including inhibitory control, working memory, and decision-making [14,15]. By comparison, participants with ASPD drawn from a community sample have shown impaired inhibitory control and decision-making (but intact working memory) compared with controls [16]. No study to our knowledge, however, has examined neurocognitive functioning in the putative subtype of problem gamblers with ASPD (“PG + ASPD”). We therefore recruited young adult problem gamblers with and without ASPD from the general community and used a previously validated computerized battery [17] to compare dissociable cognitive functions between groups. Based on previous research, we hypothesized that PG + ASPD participants would show greater impulsivity and impaired executive functioning, consistent with underlying dysregulation of frontostriatal circuitry.

2. Methods

2.1. Participants

Non-treatment-seeking young adults (aged 18−29 years) were recruited from two large metropolitan areas by media advertisements as part of an ongoing longitudinal study of impulsive behaviors. Participants had gambled at least five times during the preceding 12 months. The only exclusion criteria for the study were an inability to understand and complete required study procedures or an inability/unwillingness to provide written informed consent. Respondents with psychiatric diagnoses and substance use comorbidities were allowed to participate, as the analysis sought to examine a naturalistic sample of young adult gamblers. Problem gamblers were grouped into two categories based on whether they had a lifetime history of ASPD (hereafter referred to as PG + ASPD) or had no such history (hereafter referred to as controls).

The Institutional Review Boards of the University of Chicago and University of Minnesota approved the study and consent process, and all procedures followed the principles of the Declaration of Helsinki. After a complete description of the study, participants provided voluntary informed consent. Participants were compensated for their time with a $50 gift card to an internet-based retailer or local department store.

2.2. Assessments

2.2.1. Psychiatric evaluation

All participants underwent a semi-structured clinical interview to assess clinical characteristics of gambling disorder. Clinical questions assessed age at onset of gambling, frequency of gambling, and consequences of gambling behavior, including amount of money lost to gambling in the past year.

Gambling disorder symptoms were assessed using the Structured Clinical Interview for Pathological Gambling (SCI-PG) modified for DSM-5, where a score of ≥4 is consistent with a current gambling disorder [18]. Gamblers who endorsed 1 to 3 criteria were characterized as having problem gambling, and a score of 0 indicates no risk.

Gambling severity was assessed using the clinician-administered Yale–Brown Obsessive–Compulsive Scale Adapted for Pathological Gambling (PG-YBOCS). The PG-YBOCS is a 10-question scale that rates gambling symptoms over the preceding 7 days on a scale from 0 to 4, with higher scores reflecting greater symptom severity [19]. The final score is the sum of two subscales – gambling thoughts/urges and gambling behavior – each of which includes 5 questions.

Participants also were evaluated using the Hamilton Anxiety Rating Scale (HAM-A) [20], a clinician-administered, 14-item scale measuring global anxiety; the Hamilton Depression Rating Scale (HAM-D) [21], a 17-item, clinician-administered scale assessing depressive symptoms; and the Quality of Life Inventory (QoLI) [22], a 16-item self-report measure of life satisfaction.

Psychiatric comorbidity was evaluated using the Mini International Neuropsychiatric Interview (MINI) [23] and the Minnesota Impulse Disorders Interview (MIDI) [24]. The MINI assesses common DSM-IV-TR psychiatric disorders, including antisocial personality disorder (ASPD). To receive a diagnosis of ASPD, respondents must respond “yes” to ≥2 (of 6) problematic childhood behaviors and ≥3 (of 6) antisocial behaviors since age 15. The MIDI is a self-report screening instrument for impulse control disorders. Quantitative details regarding substance use were also collected.

2.2.2. Impulsivity questionnaires

2.2.2.1. Barratt Impulsiveness Scale, Version 11 (BIS-11).

The BIS-11 is a 30-item, self-report measure of impulsivity across attentional (inability to concentrate), motor (acting without thinking), and non-planning (lack of future orientation) dimensions [25]. Each of the 30 items is rated on a 4-point scale from 1 (“rarely/never”) to 4 (“almost always”), where 4 indicates greater impulsiveness.
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