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Selective decision-making deficits in at-risk gamblers ☆

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ABSTRACT

Despite reasonable knowledge of pathological gambling (PG), little is known of its cognitive antecedents. We evaluated decision-making and impulsivity characteristics in people at risk of developing PG using neuropsychological tests. Non-treatment seeking volunteers (18–29 years) who gamble ≥5 times/year were recruited from the general community, and split into two groups: those "at risk" of developing PG ($n=74$) and those social, non-problem gamblers ($n=112$). Participants undertook the Cambridge Gamble and Stop-signal tasks and were assessed with the Mini-International Neuropsychiatric Interview and the Yale Brown Obsessive Compulsive Scale Modified for Pathological Gambling. On the Cambridge Gamble task, the at-risk subjects gambled more points overall, were more likely to go bankrupt, and made more irrational decisions under situations of relative risk ambiguity. On the Stop-signal task, at-risk gamblers did not differ from the social, non-problem gamblers in terms of motor impulse control (stop-signal reaction times). Findings suggest that selective cognitive dysfunction may already be present in terms of decision-making in at-risk gamblers, even before psychopathology arises. These findings implicate selective decision-making deficits and dysfunction of orbitofronto-limbic circuitry in the chain of pathogenesis between social, non-problematic and pathological gambling.

1. Introduction

Gambling is a commonplace phenomenon across cultures, and in extreme forms, can evolve into pathological gambling (PG), a disorder characterized by persistent, recurrent maladaptive patterns of gambling behavior and functional impairment. Despite reasonable knowledge of PG, little is known of its cognitive and neurobiological antecedents. Cognitive tests sensitive to cortico-subcortical dysfunction are well placed as candidate vulnerability markers in psychiatry, since they are situated along the chain of pathogenesis between underlying genetic-environmental contributions and the top-level manifestation of symptoms (Gottesman and Gould, 2003; Chamberlain and Menzies, 2009). Dysfunction of neural circuitry thought to underpin aspects of decision-making is central to neurobiological models of PG (Grant et al., 2006; Wilber and Potenza, 2006; Potenza, 2008) and patients with the disorder often manifest impaired decision-making on objective tests (van Holst et al., 2010a).

Several cognitive tasks have been used to explore decision-making in people with PG. The most frequently used paradigm has been the Iowa Gambling task (Bechara et al., 1994), in which participants try to win points by choosing cards from one of several card decks. Most cards result in a reward while some result in a penalty; some decks contain more rewarding cards than others, and healthy participants learn through experience to choose the more rewarding decks. Multiple studies have found that patients with damage to ventromedial/orbitofrontal cortices, but not the dorsolateral prefrontal cortices, draw cards from high payout/high risk decks to the detriment of long term performance (Damasio, 1996; Rogers et al., 1999). Petry (2001) found that substance abuse and PG had an additive effect on preference for decks containing greater immediate short-term gains, resulting in overall net losses i.e. decision-making impairment. Cavedini et al. (2002) also reported significant differences between PG and healthy volunteers, with PG preferring more disadvantageous decks and controls preferring more advantageous decks. Goudriaan et al. (2006) reported not only that pathological gamblers were worse than controls on the Iowa Gambling task, but that they also showed lower anticipatory skin conductance responses and heart rate decreases than controls when pondering choices of disadvantageous decks. Several other subsequent studies have also reported decision-making deficits in PG versus controls on the Iowa Gambling task, particularly in relation to choosing disadvantageous decks (Forbush et al., 2008; Roca et al., 2008).

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Elsewhere, authors have deployed the Game of Dice task in PG. On each trial, subjects guess the number that will appear in the next dice throw. They can choose one number or several; each choice is linked with different number of points that will be won or lost: 1000 units for the choice of a single number, 500 for two numbers, 200 for three numbers, and 100 for four numbers. The game distinguishes 'disadvantageous decisions' (choosing one or two numbers with winning probability <50% and high gains and penalties) from 'not disadvantageous decisions' (choosing three or four numbers with winning probability >50% with low gains and penalties) (Brand et al., 2005). Two studies have reported that patients with PG show inappropriate preference for disadvantageous choices compared to controls on this task (Brand et al., 2005; Labudda et al., 2007).

Another paradigm that has been developed to explore aspects of decision-making, which formed the focus of the present study, is the computerized Cambridge Gamble task. This offers several potential advantages — specifically, it allows for the fractionation of different components of decision-making across a range of well-defined and clearly indicated contingencies (Rogers et al., 1999). In contrast to the Iowa Gambling task, it measures decision-making under risk (i.e. with explicit probabilities) rather than under ambiguity. It also minimizes demands for stimulus-reinforcement learning, reversal learning, and working memory (Clark et al., 2008). Increased betting behavior on the Cambridge Gamble task has been reported in frontotemporal dementia (Rahman et al., 1999), subarachnoid hemorrhage of the anterior communicating artery (Mavaddat et al., 2000), and damage to orbitofrontal/ventrolateral and insular but not dorsolateral prefrontal cortices (Manes et al., 2002; Clark et al., 2008). Lawrence et al. (2009) recently reported that non-treatment seeking subjects with PG were intact in terms of deliberation times versus controls, but were more likely to go bankrupt, and gambled more points regardless of box ratio. The PG group showed numerically lower quality of decision-making overall than controls (mean 90% versus 96%) albeit this was not statistically significant in the model used.

One vital means of exploring candidate vulnerability markers in neuropsychiatry is to evaluate cognitive function in young adults who may be at risk of later developing the condition under study. We therefore recruited young adults who gamble five or more times per year, and investigated cognitive dysfunction in those at risk of gambling compared to those who were not. We hypothesized that those at risk of developing PG would exhibit impaired decision-making, implicating dysfunction of orbitofronto-limbic circuitry in the pathogenesis of the disorder itself (Clark, 2010), suggesting a vulnerability marker.

2. Methods

2.1. Subjects

Participants comprised non-treatment-seeking young adults aged 18–29 years, recruited as part of a longitudinal study seeking ultimately to characterize predictive factors in the later development of PG. Subjects were self-selected in response to media announcements in a metropolitan area, and were compensated with a $50 gift card to a local department store. The only inclusion criterion was that the subject had gambled in any form at least five times during the past 12-months. The only exclusion criterion was an inability to understand/undertake the procedures and to provide written informed consent. Since we sought to examine a naturalistic sample of people reflective of the broader population, 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 Minnesota approved the study and the consent statement. After all study procedures were explained to the subjects, voluntary written informed consent was obtained.

2.2. Assessments

Raters assessed each subject using the Mini-International Neuropsychiatric Interview (Sheehan et al., 1998) to examine psychiatric comorbidity; and the Structured Clinical Interview for Pathological Gambling (SCI-PG) (Grant et al., 2004), a 10-item instrument assessing symptoms of PG: a score of 0 indicates negligible/low risk, 1–2 "at risk", 3–4 actual problem gambling, and 5+ current PG.

Subjects reported frequency of gambling behavior as well as money lost gambling. In addition, subjects were asked questions about any legal, social, occupational or academic consequences from gambling in order to assess the overall functional impact of gambling and other health issues. All subjects were asked about addiction and psychiatric disorders in first-degree family members.

Dissociable aspects of decision-making were assessed using the Cambridge Gamble task, which has been validated previously in various clinical contexts including brain lesions (Manes et al., 2002; Clark et al., 2008) and PG (Lawrence et al., 2009). There were four practice trials followed by eight blocks of nine trials. At the start of each block, the 'cumulative points' were reset to 100. On each trial, participants were presented with an array of red and blue boxes, totaling 10 (see screenshot, Fig. 1). 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). Volunteers were informed that for each trial, the computer had hidden a 'token' inside one of the boxes, and that they had to decide 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 as to whether this choice was correct or incorrect. Choices of bets were offered on each trial, equating to 5%, 25%, 50%, 75%, or 95% of accumulated points. In the ascend condition (half of the blocks), the gamble option was presented from 5% upwards; and vice versa for the descend condition (half of the blocks). Subjects touched the screen when the desired choice of bet was displayed. Key outcome measures were (i) the mean proportion of points gambled at each box-ratio; (ii) the mean proportion of rational decisions made at each box-ratio, i.e. the proportion of trials where the volunteer chose red when red boxes were in the majority, and chose blue when blue boxes were in the majority; (iii) mean deliberation time at each box-ratio; and (iv) overall number of blocks where the participant went bankrupt.

We assessed response inhibition using the Stop-signal task (Logan et al., 1984), previously validated in neurosurgical patients (Aron et al., 2003) and in the context of impulsivity associated with attention deficit hyperactivity disorder (Chamberlain et al., 2011). On this task, volunteers 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') signaling that the participant should suppress the response for that one trial. This task estimated the time taken by each volunteer's brain to suppress an already triggered command, (the 'stop-signal reaction time'). The other outcome measure was the median response times for go trials.

2.3. Data analysis

Subjects were grouped a priori into two categories based on responses to DSM-IV-TR PG criteria (using the SCI-PG): those who met no criteria were classified as 'social/non-problem' gamblers; those who met 1–2 criteria were classified as 'at risk' gamblers. Those scoring 3 or more were excluded ($n = 3$). IBM SPSS Software, Version 19 was used for the analyses. Group demographic and clinical characteristics where compared using *t*-tests or chi-squared tests (with Yates correction where expected cell count <5) as appropriate. Cambridge Gamble task results were analyzed using repeated-measures analysis of variance (rmANOVA) with group (low risk/at risk) as the between-subject factor, and within-subject factors of box ratio (9:1/8:2/7:3/6:4) and condition (ascend/descend). Stop-signal task results were analyzed using *t*-tests. Where at-risk and non-problem gamblers differed significantly in terms of a cognitive performance measure, subgroup analyses were conducted in the at-risk gamblers to compare those with and

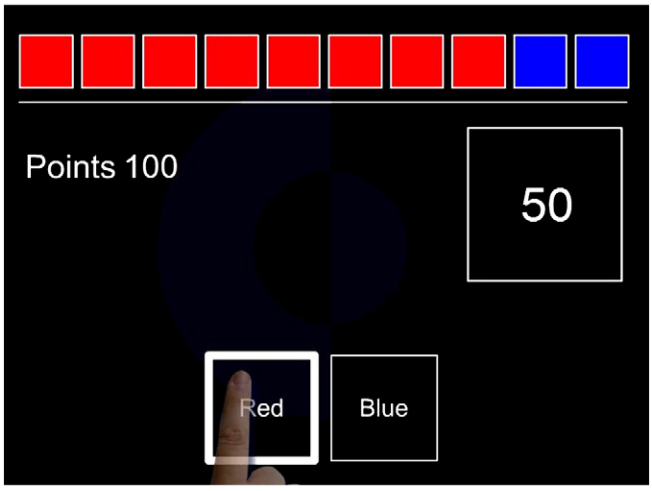

Fig. 1. Example of screen display from the Cambridge Gamble task. Reproduced with permission from Cambridge Cognition.