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Pathological Choice: The Neuroscience of Gambling and Gambling Addiction

Luke Clark,1 Bruno Averbeck,2 Doris Payer,3 Guillaume Sescousse,4 Catharine A. Winstanley,5 and Gui Xue6
1Department of Psychology, University of Cambridge, Cambridge CB2 3EB, United Kingdom, 2Laboratory for Neuropsychology, National Institute of Mental Health, Bethesda, Maryland 20892, 3Research Imaging Centre, Centre for Addiction and Mental Health, Toronto, Ontario M5T 1R8, Canada, 4Donders Institute for Brain, Cognition and Behaviour, Radboud University Nijmegen, 6525 HP Nijmegen, The Netherlands, 5Department of Psychology, University of British Columbia, Vancouver, British Columbia V6T 1Z4, Canada, and 6State Key Laboratory of Cognitive Neuroscience and Learning, and IDG/McGovern Institute for Brain Research, Beijing Normal University, Beijing, China 100875

Gambling is pertinent to neuroscience research for at least two reasons. First, gambling is a naturalistic and pervasive example of risky decision making, and thus gambling games can provide a paradigm for the investigation of human choice behavior and “irrationality.” Second, excessive gambling involvement (i.e., pathological gambling) is currently conceptualized as a behavioral addiction, and research on this condition may provide insights into addictive mechanisms in the absence of exogenous drug effects. This article is a summary of topics covered in a Society for Neuroscience minisymposium, focusing on recent advances in understanding the neural basis of gambling behavior, including translational findings in rodents and nonhuman primates, which have begun to delineate neural circuitry and neurochemistry involved.

Introduction
Gambling is a branch of the entertainment industry where patrons stake an object of value (typically money) on the uncertain prospect of a larger reward (the “jackpot”). Gambling dates back several millennia and remains ubiquitous across human societies, with lifetime gambling participation reported as 78% in the United States (Kessler et al., 2008). As such, gambling games serve as a useful model of risky choice, to the extent that laboratory tasks modeling the choice between two lotteries are regarded as “the fruitfly of behavioral economics” (Kahneman, 2011). In light of the widespread recognition that the expected value of gambling is negative (“the house always wins”), gambling games may shed further light on some of the errors and biases that characterize human decision making. Examining their underlying neural mechanisms is naturally relevant to the emergent discipline of neuroeconomics.

Gambling also has a more insidious side. Pathological gambling was first recognized as a psychiatric disorder in 1980 and was grouped initially in the Impulse Control Disorders. An international program of research over the past decade has revealed multiple similarities between pathological gambling and the substance use disorders, including neurobiological overlap (Petry, 2006; Leeman and Potenza, 2012). Whereas the comparability with obsessive compulsive disorders was also evaluated, the support for placement on a “compulsive spectrum” was mixed (Holander and Wong, 1995). This process culminated in the recent reclassification of pathological gambling (now to be called “Gambling Disorder”) into the addictions category of the DSM5 (Petry et al., 2013). This ratification of the so-called “behavioral addictions” is a pivotal step for not only the gambling field, but for addictions research in general.

The current article aims to provide a concise overview of recent developments in our understanding of decision making during gambling and the relevance of these processes to problem gambling (for comprehensive overviews, see van Holst et al., 2010; Hodgins et al., 2011; Leeman and Potenza, 2012). We begin by describing some emerging methods for probing gambling decisions, highlighting translational models, behavioral economic tasks, and cognitive distortions associated with gambling (Fig. 1). We then consider the underlying neural mechanisms, distinguishing neurochemical substrates and neuroanatomy.

Models of gambling decisions: translational probes
Given that the calculation of risk versus reward trade-offs is inherent in numerous aspects of real-world choice and foraging behavior, it should be unsurprising that laboratory animals are capable of performing decision-making tasks that resemble gambling. Recent work has aimed to model gambling decisions in rats using operant behavioral tasks derived from the established probes of choice behavior in human neuropsychology and cognitive psychology. One widely used human test is the Iowa Gambling Task (Bechara et al., 1994), which quantifies the deficits in
affective decision making seen after injury to the ventromedial prefrontal cortex. In humans, this task involves a series of choices between four decks of cards that offer gains and losses of varying amounts of money. A key challenge in translating the procedure into animals concerns the representation of “loss”; standard reinforcers, such as sugar pellets, are instantly consumed and thus cannot be deducted in the same way as money or points. In the rat Gambling Task (Zeeb et al., 2009), rats choose between four apertures that vary in the probability of delivering a smaller or larger number of sugar pellets, as well as the probability of receiving time-out penalties of varying durations. Like the human version, the two apertures that offer larger rewards are also associated with longer and more frequent time-outs, and most rats learn to avoid these tempting options to maximize their sugar pellet profits over the duration of the task. (The key decision here is probabilistic and the task should not be confused with temporal discounting).

Postacquisition lesions to BLA skewed rats’ preference toward the high-risk high-reward options, matching the observation that amygdala damage leads to disadvantageous choice in the Iowa Gambling Task (Bechara et al., 1999; Zeeb and Winstanley, 2011). If BLA lesions were made before task acquisition, animals struggled to develop the optimal strategy and correctly discriminate between the options. Lesions to the orbitofrontal cortex (OFC) impaired acquisition of the rodent task in an identical manner but did not affect performance if the lesions were implemented after animals had learned the correct strategy. Such data support the suggestion that the classic disruption of everyday decision making associated with ventromedial prefrontal cortex lesions may stem from a difficulty in learning the optimal strategy, rather than an increase in preference for risky outcomes per se (Bechara et al., 2000; Fellows and Farah, 2005). Moreover, the similarity between the effects of BLA and OFC lesions on task acquisition suggested that these two areas work together to promote development of the optimal strategy, a hypothesis recently confirmed using a functional disconnection procedure (Zeeb and Winstanley, 2013). Hence, similar brain regions appear to be involved in guiding decision making under uncertainty in both rats and humans.

Prefrontal connectivity with the striatum is also implicated in choice behavior. Contemporary hypotheses of frontostriatal function emphasize a primary role in either action selection or reinforcement learning, both of which are likely important in substance addiction and behavioral addictions. To differentiate these elements, Seo et al. (2012) trained monkeys on a task in which they had to select rewarding actions using either reinforcement learning or perceptual inference. While the animals performed this task, neural activity was monitored simultaneously in anatomically connected regions of lateral prefrontal cortex (LPFC; caudal area 46) and the dorsal striatum (DS, primarily the anterior caudate nucleus). A larger fraction of LPFC neurons represented selected actions, independent of how they were selected. In the perceptual inference condition, the LPFC representation of the selected action preceded the DS representation of the selected action, whereas in the reinforcement learning condition, both structures represented the actions up to 500 ms before they were executed, with no clear temporal ordering. Additionally, DS more often represented the value of the selected action when it was selected using both perceptual inference and reinforcement learning. Thus, a hypothesis that the DS was important for action selection was not supported, but DS did often represent action values, when driven by either reinforcement learning or perceptual inference. LPFC, by contrast, appears to play a dominant role in representing and selecting actions, particularly when the selection is based on perceptual inference.

**Insights from behavioral economics**

Behavioral economic aims to decompose the processes of option valuation into simple components that can be quantified with discrete parameters (Schonberg et al., 2011). Prospect theory (PT) remains the most influential of these accounts because of its ability to describe a range of common behaviors and deviations from normative expected value theory (Kahneman and Tversky, 1979). A central feature of PT is “loss aversion,” referring to the empirical observation that humans (and other species) are more sensitive to losses than to gains. For example, subjects typically reject mixed gambles that offer a 50–50 chance of winning or losing a given amount of money. Loss aversion may be underpinned by value computations in the ventral striatum and amygdala (Tom et al., 2007; De Martino et al., 2010) and has been shown to be modulated by thalamic norepinephrine (Takahashi et al., 2013). In addition to this asymmetry between gains and losses, PT describes a value function for gains that is concave,
contrasting with a value function for losses that is convex. This disparity accounts for subjects’ tendency to be risk averse in the gain domain and risk seeking in the loss domain, which may account for the “loss chasing” behavior that is characteristic of problem gamblers (Campbell-Meiklejohn et al., 2008, 2011). Although recent work has demonstrated impaired processing of loss information (Brevers et al., 2012) and aversive signals (Brunborg et al., 2012) in pathological gamblers, loss aversion is yet to be formally quantified in pathological gamblers.

PT also posits nonlinearity in probability calibration, whereby small probabilities are overestimated and medium to high probabilities are underestimated, in an inverse S-shaped “probability weighting function.” These subjective distortions are reflected in brain activity profiles observed in the ventral striatum and dorsolateral prefrontal cortex (Tobler et al., 2008; Hsu et al., 2009) and also correlate with striatal dopamine D₃ receptor density (Takahashi et al., 2010). Behaviorally, the overestimation of small probabilities may contribute to the attractiveness of gambles, such as a lottery (Trepel et al., 2005). Ligneul et al. (2013) tested this hypothesis in pathological gamblers, calculating “certainty equivalents” across varying levels of objective probability from 0 to 1. As expected, the results revealed elevated risk taking in gamblers compared with nongambling controls; however, this behavior was not linked to a specific distortion of small probabilities but rather to a general overweighting across the entire probability range. Similar approaches using the discounting framework have demonstrated fine alterations of value representations in the ventral striatum in pathological gamblers (Miedl et al., 2012; Peters et al., 2012).

Gambling-related cognitive distortions

In addition to the computational characterization of gambling offered by behavioral economics, psychological models of gambling have additionally highlighted the central role of cognitive distortions during gambling. These distortions refer to how the gambler thinks about randomness, chance, and skill (Ladouceur and Walker, 1996; Clark, 2010) and foster an inappropriately high expectation of winning during the game. A number of specific biases have been described, and these cognitions can be effectively targeted as one element of psychotherapy for pathological gambling (Fortune and Goodie, 2012).

Arguably, the most classic distortion is the gambler’s fallacy, which is a bias in the processing of random sequences. In this compelling example, the expectancy of a certain event (e.g., heads in a coin flip) becomes less likely after a long series of the same event (e.g., three successive tails). The phenomenon occurs in a coin flip) becomes less likely after a long series of the same which is a bias in the processing of random sequences. In this hypothesis in pathological gamblers, calculating “certainty equivalents” across varying levels of objective probability from 0 to 1. As expected, the results revealed elevated risk taking in gamblers compared with nongambling controls; however, this behavior was not linked to a specific distortion of small probabilities but rather to a general overweighting across the entire probability range. Similar approaches using the discounting framework have demonstrated fine alterations of value representations in the ventral striatum in pathological gamblers (Miedl et al., 2012; Peters et al., 2012).

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Neurochemical substrates: dopamine dysregulation

Dopamine has been a prime candidate for investigation of neurochemical abnormalities in pathological gamblers, given its established roles in both drug addiction and rewarded behavior. In patients with Parkinson’s disease, sudden onset gambling can be observed, alongside other reward-driven behaviors, including compulsive shopping and hypersexuality, as a side effect of dopamine agonist medications (Ambergam, 2011; Voorn et al., 2011). The most direct approach for quantifying dopamine transmission in human brain is PET imaging of dopamine ligands, of which the most widely studied is [¹¹C]-raclopride, a D₂/D₃ receptor antagonist that binds predominantly in the striatum. Building upon evidence for robust reductions in striatal dopamine D₂/D₃ receptor availability in substance users dependent on a number of distinct drugs (Martinez et al., 2004; Volkow et al., 2007; Fehr et al., 2008), recent studies have used this ligand in pathological gamblers (Limnet et al., 2011; Clark et al., 2012; Joutsa et al., 2012; Boileau et al., 2013). Notably, none of these studies has detected a significant group difference in dopamine D₂/D₃ binding, although some individual differences have been observed, for example, against trait impulsivity (Clark et al., 2012).

Ongoing work is using alternative PET tracers that offer advantages over [¹¹C]-raclopride. [¹¹C]-([+]-Propyl-hexahydro-naphthoxazin (PHNO) is a D₂ receptor antagonist that binds preferentially to D₃ over D₂ receptors in vivo (Narendran et al., 2006). The D₃ receptor subtype is localized to limbic circuitry and implicated in drug self-administration and relapse behavior in preclinical models (Heidbreder et al., 2005). As a dopamine receptor agonist, [¹¹C]-([+]-PHNO is also more sensitive to displacement by endogenous dopamine than [¹¹C]-raclopride (an antagonist) (Willeit et al., 2008). A recent PET study using [¹¹C]-([+]-PHNO in methamphetamine abusers indicated higher baseline binding in the D₃-rich substantia nigra and pallidum, coupled with lower binding in the D₂-rich dorsal striatum (Boileau et al., 2012). Employing a multimodal PET design in pathological gamblers using both [¹¹C]-([+]-PHNO and [¹¹C]-raclopride (Boileau et al., 2013), no group differences were detected, but a positive correlation was observed between gambling severity scores and [¹¹C]-([+]-PHNO binding in substantia nigra, a region where signal is fully attributable to D₃ (Tziortzi et al., 2011). This association suggests that D₃ expression is relevant to symptom severity in problem gambling, and as an addiction phenotype, it may be a useful marker for risk.

Preliminary work has also begun to examine dopamine release in pathological gamblers, with some provocative early findings. The change in [¹¹C]-([+]-PHNO binding was examined after a challenge dose of oral amphetamine (0.4 mg/kg) in pathological gamblers: greater dopamine release was detected in the dorsal (associative)
striatum in the pathological gamblers (D. Payer, I. Boileau, D. Lobo, B. Chugani, A. Behzadi, A. Wilson, S. J. Kish, S. Houle, M. Zack, unpublished observations). This result effect is echoed in more two further experiments examining task-related changes in \([^{11}]C\)-raclopride binding in pathological gamblers, where higher levels of dopamine release were correlated with greater subjective excitement (Linnet et al., 2011) and gambling severity (Joutsa et al., 2012). Notably, the available data in drug addiction show blunted dopamine release in response to psychostimulant administration (Volkow et al., 1997; Martinez et al., 2007). The extent to which these discrepancies reflect etiological differences between substance and behavioral addictions, or the masking of incentive sensitization processes via drug-induced depletion of dopamine stores (Robinson and Berridge, 2003), is a key question in ongoing research.

Rodent models have also provided a means of examining the neurochemistry of gambling, implicating dopamine and seroton in influences. In light of the effects of dopamine agonist medications in Parkinson’s disease, it is notable that administration of selective D2 agonists did not affect choice behavior on the rat Gambling Task (Zeeb et al., 2009). However, whereas the D2 receptor antagonist eticlopride improved choice of the best option, amphetamine and the 5-HT1A agonist 8-OH-DPAT were found to impair performance (Zeeb et al., 2009). Selective dopamine agonists did not block the effects of amphetamine on choice, even though such agents did attenuate amphetamine-induced increases in motor impulsivity (Zeeb et al., 2013). Furthermore, amphetamine’s effects were not mimicked by dopamine, 5-HT, or noradrenaline reuptake inhibitors but were reproduced by different combinations of these drugs (Baarendse et al., 2013). Such results imply concurrent regulation of choice behavior on the rat Gambling Task by multiple monoaminergic systems, consistent with human data (Rogers, 2011).

A modified choice procedure in the rodent has been used to assess the sensitivity to stake size, implicating striatal D2/3 transmission specifically (Cocker et al., 2012). In this task, the rats choose between two options of equivalent value, one of which delivers a guaranteed reward, and the other offers either double that reward or nothing, with 50:50 odds. The reward size varies over the session from 1 to 3 pellets. Whereas some animals are largely indifferent to this “escalation of commitment,” other rats become markedly risk avoidant as the stake increases, increasing their preference for the guaranteed option (Campbell-Meiklejohn et al., 2012; Sescousse and den Ouden, 2013). Critically, such a behavioral shift confers no advantage in terms of reward earned and may be considered irrational in a similar vein to the framing effects observed in human choice under risk. PET imaging using \([^{11}]C\)-raclopride revealed that D2/3 receptor binding in the dorsal striatum correlated significantly with the degree of wager-sensitivity rats exhibited. Moreover, in a key advantage over human PET imaging, it was possible to use autoradiography to confirm that the differences in raclopride binding were the result of lower levels of D2/3 receptor expression, as opposed to variation in endogenous dopamine levels.

Functional neuroimaging of reward-related circuitry

Functional neuroimaging studies have also contributed much to our understanding of appetitive processing in pathological gamblers and provide data that complement the investigations of dopamine transmission (Schott et al., 2008). Several fMRI studies in pathological gamblers have reported blunted neural responses to monetary gains and appetitive cues, primarily in ventral striatum and orbital/lateral PFC (Reuter et al., 2005; de Ruiter et al., 2009; Balodis et al., 2012). This observation can be interpreted in terms of the reward deficiency hypothesis (Comings and Blum, 2000), consistent with the PET evidence reviewed above indicating reduced dopamine receptor levels in addiction. However, other recent studies have described increased, rather than decreased, responses to monetary rewards in the same population (Hewig et al., 2010 using EEG; van Holst et al., 2012 using fMRI), prompting ongoing debate about the impact of naturalistic cues and stage of processing (anticipation vs outcome) in these effects (Leyton and Vezina, 2013; Limbrick-Oldfield et al., 2013).

One means of resolving these discrepancies is to consider the sensitivity to nonmonetary (i.e., nonaddiction related) rewards in gamblers. Using an incentive delay protocol involving both monetary and visual erotic rewards, pathological gamblers showed a markedly decreased response to the erotic cues, compared with monetary cues, in the ventral striatum (Sescousse et al., 2013). This differential response was correlated with the severity of gambling symptoms and accompanied by a similarly reduced behavioral motivation for erotic rewards. Comparable designs indicate blunted brain responses to non–drug-related cues in drug-addicted groups (Goldstein et al., 2007; Wrase et al., 2007; Bühler et al., 2010). These findings suggest that the key variable of interest may be the differential response to monetary (or drug) rewards versus other (primary) appetitive cues, rather than the response to money or drugs per se.

Experiments on gambling-related cognitive distortions also implicate reward-related circuitry, as well as the interactions with regions responsible for top-down cognitive control. Specifically, the gambler’s fallacy appears to arise from an imbalance between cognitive and emotional decision making mechanisms in the brain (Shiv et al., 2005; Xue et al., 2011). Using a card guessing task to capture subjects’ tendency to predict the break of a streak as it continued (a signal of the gambler’s fallacy), enhanced neural responses in left LPFC were observed to outcomes that were followed by a gambler’s fallacy switch (Xue et al., 2012b). A follow-up experiment applied anodal transcranial direct current stimulation, a procedure known to enhance cortical excitability and cerebral perfusion (Stagg et al., 2013), over the same region. Corroborating the fMRI data, stimulation to left LPFC increased the use of the gambler’s fallacy (Xue et al., 2012b) and point to a causal role of this region in implementing this suboptimal decision strategy, guided by false world models.

Extending this interpretation in a larger sample of college students (N = 438), the use of the gambler's fallacy was positively correlated with general intelligence (Raven’s matrices) and executive function (2-back working memory and Stroop tasks) but was negatively correlated with affective decision making (Iowa Gambling Task) (Xue et al., 2012a). Thus, the gambler’s fallacy seems to be associated with (1) weak function in the affective decision making system and (2) strong function in the LPFC cognitive control system (Xue et al., 2011, 2012b).

In contrast to these cortical responses, the robust striatal activations seen in response to monetary wins are not evidently modulated by the psychological context that characterizes these gambling distortions. For example, the striatal responses to winning outcomes did not differ between the first win in a streak, compared with the fourth successive win (Akitsuki et al., 2003). In a study investigating the illusion of control, striatal activity did not differ between choice and no-choice conditions, even though perceived control did enhance subjective confidence (Kool et al., 2013). However, both distortions appear to be coded in higher cortical regions. Using a card guessing game to compare trials where either the subject or computer predicted the location of the winning card, agency affected not only the amount bet but also
subjects’ “world model” regarding the outcome dependency (Xue et al., 2013). Functional imaging results revealed that the decision-related activation in the lateral and medial PFC was significantly modulated by both agency and previous outcome and that these effects were further predicted by the trait-like disposition to attribute negative events externally. These results suggest that the prefrontal decision making system can be modulated by abstract beliefs and are thus vulnerable to factors, such as false agency and attribution.

Nevertheless, subcortical responses have been observed to near-miss outcomes during a simulated slot machine task. Specifically, these events recruited overlapping neural circuitry to the jackpot wins in the ventral striatum, amygdale, and anterior insula (Clark et al., 2009; Shao et al., 2013). Of these responses, the effect in the insula was seen to covary with trait levels of gambling distortions and individual differences in the motivational effect of the near misses (Clark et al., 2009). Administering a behavioral version of the slot machine game to patients with focal brain injury, a group with insula lesions was seen to be insensitive to near-misses (i.e., did not show the typical motivational response) and also failed to manifest the gambler’s fallacy (L. Clark, B. Studer, J. Bruss, D. Tranel, A. Bechara, unpublished observations). Thus, these results again highlight how gambling cognitions and persistent play are most likely to emerge from an imbalance between bottom-up emotional systems and prefrontal control systems, rather than a disruption in either component in isolation.

In conclusion, studies of cognitive processing during gambling have begun to uncover a wealth of phenomena that are relevant to problematic gambling, and useful as more general models of human decision making. The arsenal of tasks that probe gambling-related decision making can be implemented in other mental health problems associated with decision making disruption, including schizophrenia (Balzan et al., 2013), obsessive compulsive disorder (Reuven-Magril et al., 2008), and suicidal behavior (Dombrovski et al., 2011). The convergence with translational models of choice behavior in nonhuman species carries enormous potential for delineating the neural circuitry. Thus far, the translational work has progressed primarily in the area of value-based decision making, highlighting the roles of prefrontal cortex, striatum, and amygdala, innervating by ascending dopamine and serotonin inputs. Further progress may be achieved by modeling constructs from behavioral economics, and gambling-related cognitive distortions, in nonhuman species. For example, recent data from a rodent slot machine task indicate that rats are susceptible to near-miss outcomes and that erroneous attempts to collect reward on near-miss trials were dramatically increased by the D₂-receptor agonist quinpirole (Winstanley et al., 2011). Interestingly, these effects appear to be mediated by the D₂ receptor subtype specifically, leading to the exciting possibility that D₂ receptor antagonists may be a useful treatment for compulsive slot machine play (P. Cocker, B. Le Foll, R. Rogers, C. A. Winstanley, unpublished observations).

Studies of patients with pathological gambling are also beginning to provide clues about the mechanisms involved in addiction. One interpretation of the null results from investigations of (11C)-raclopride binding in pathological gamblers is that the robust reductions observed in drug addiction may represent a consequence of long-term drug exposure, rather than a preexisting vulnerability marker (Groman et al., 2012). Over the coming decade, we anticipate a similar program of research for other candidate behavioral addictions, such as excessive online video gaming (Kim et al., 2011; Kühn et al., 2011).

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J. Neurol., November 6, 2013 • 33(45):17617–17623 • 17621
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