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What can affective neuroscience teach us about gambling?

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For the past 25 years, Jaak Panksepp, professor of psychology at Bowling Green State University, has waged a sometimes lonely battle against the purveyors of what he calls “neurobehaviorism.” In his opinion, behavioral neuroscientists have simply replaced the environmental orientation of classic behaviorists, like Watson and Skinner, with a neurochemical orientation. In each case, the living being is essentially a “scarecrow” that responds to stimuli. The subjective experience of that living being is granted little if any importance because it cannot be empirically verified or tested. In the quest for objectivity, neuroscientists—like behaviorists before them—have eschewed the fundamental issue of consciousness. Panksepp believes that this has impeded progress in our understanding and treatment of many forms of psychopathology, and particularly those that involve disturbances in motivation, such as addiction.

A consequence of neurobehaviorism is illustrated by the ongoing debate on the role of dopamine in addiction. In the past 25 years, the subjective state associated with brain dopamine activation has been variously described as pleasure, reward, reinforcement, drive, wanting, salience, and expectancy. The most recent formulation describes brain dopamine activation as the neural response to a “reward prediction error” (Schultz, 2001). Despite a quarter century of debate, the true subjective state associated with dopamine activation (if one exists) remains unclear.

Much of the difficulty, according to Panksepp, stems from the contrived manner in which neuroscientists assess processes such as reward. For example, when an animal returns to a location where it previously received a drug (e.g., cocaine), this behavior is interpreted as an indication of cocaine-induced reward (or the memory of such a reward). Although this is a reasonable inference, the Conditioned Place

Preference model of drug reward has difficulty contending with critical anomalies. For example, alcohol is widely enjoyed and abused by humans yet consistently leads to avoidance in the Place Preference paradigm. This is not due to extreme intoxication, because avoidance is seen at a range of doses, nor to the aversive aftereffects of drinking (hangover), because the animals are returned to their home cages well before such effects emerge.

An alternative approach to behavioral neuroscience is what Panksepp terms “affective neuroscience.” This approach focuses to a greater extent on ecologically valid stimuli and spontaneous responses to assess the neural basis of a phenomenon. The primary question to be answered is, “What is the subjective emotional state of the organism in this particular situation?"

Panksepp has shown that animals (rodents) emit sounds of particular frequencies that correspond to particular naturally occurring states. High-frequency sounds accompany positive anticipatory or happy states like social play; low-frequency sounds accompany states of stress or dysphoria. To Panksepp, these spontaneous vocalizations correspond to self-reports of affective state in humans. This assertion is supported by numerous studies where drugs with known subjective effects in humans produce the expected pattern of vocalization in animals. He has even shown that rodents vocalize in the expected manner when tickled.

Panksepp favors these natural responses as dependent measures because they “reflect the operation of distinct emotional operating systems that are concentrated in sub-neocortical regions in the brain” (Panksepp, 2005a, p. 31). In other words, these responses reflect how the brain actually operates in response to events in the real world. As such, the neural activity that gives rise to these responses may be able to tell us more about real-world conditions such as addiction and depression.

Although both the affective neuroscience and the behavioral neuroscience approaches examine behavior, a critical difference is that, in the former case, conscious experience is presumed to play a causal role. By contrast, in the latter case, conscious experience is considered epiphenomenal—an incidental byproduct of neural activity with no causal impact. Indeed, among behavioral neuroscientists, consciousness has sometimes been likened to the whirr of the lawnmower: It's loud and impressive, but it doesn't cut the grass.

Another issue is that, compared to human self-reports, which are subtle, rich, and variable, animal vocalizations seem coarse and one-dimensional. This may partly explain why behavioral neuroscientists have adopted more contrived measures (e.g., time spent in a location where a drug was given). In psychological parlance, Panksepp appears to be arguing for the importance of ecological validity (the manipulation produces an effect that generalizes to the real world) over internal

validity (the manipulation accomplishes what it is intended to). The relative importance of external/ecological validity versus internal validity is, of course, an ongoing debate in all of science.

In the clinic, unlike in the laboratory, self-report is the primary currency. As such, inferring cause and predicting effective interventions based on self-report data are not extraordinary to the clinician. For example, self-reports can specify clients' perceived motivation for their excessive behavior: cravings, loss of control following exposure to addictive stimuli, or coping with negative affect. Although this information is often accurate, its utility can be enhanced by a cogent theoretical framework. Affective neuroscience provides one such framework. The value added by an affective neuroscience framework may be especially great in the case of problem gambling, a disorder that does not fit neatly into the existing diagnostic schema.

In a similar vein, an affective neuroscience approach may shed light on aspects of mental disorders that have thus far eluded understanding or effective treatment. An excellent example of this is the recent work on the biological basis of separation distress. Panksepp has shown that opiate drugs such as heroin and morphine quell separation distress effectively and at lower doses than they do anger or fear. On this basis, he has argued that the brain opioid system mediates social pain (shame, loss, grief, jealousy). Given the well-established role of the opioid system in analgesia, the findings imply that separation distress is neurochemically similar to physical pain. In line with this reasoning, neuroimaging research in human volunteers has shown that the same brain regions that “light up” during physical pain also light up in response to social exclusion (Eisenberger, Lieberman, & Williams, 2003). Based on such findings, Panksepp proposed that certain depressive syndromes (e.g., those induced by loss) that do not respond optimally to conventional antidepressants could benefit from medications such as buprenorphine that recalibrate brain opioid function. Not surprisingly, these medications have also proven very effective in the management of opiate addiction.

The brain opioid system is one of seven evolutionarily defined systems that Panksepp's model has identified in the mammalian brain. He refers to these systems as SEEKING, FEAR, RAGE, LUST, CARE, PANIC, and PLAY. Activation of the opioid system with low doses of opiate drugs enhances PLAY, whereas deactivation induces PANIC. High doses of opiates produce sublime contentment similar to that observed in babies suckling at their mother's breast.

The other system Panksepp has emphasized as critical for addiction is the SEEKING system. This is consistent with the intense craving and compulsive reward-seeking that are the hallmarks of addiction. Panksepp proposes that the SEEKING system is a survival-oriented system that gives rise to foraging behavior

when internal signals indicate a deviation from homeostasis (e.g., hunger). This system is predominantly mediated by dopamine. The dopamine system responds selectively to novel, attention-grabbing events and stimuli that predict reward. Activation of the SEEKING system leads to “an invigorated positive feeling of engagement with tasks that can border on euphoria. All psychostimulants [e.g., amphetamine, cocaine] promote such feelings, helping explain the addictiveness of certain drugs, and also indicating why goal-directed behaviors have such a persistent quality” (Panksepp, 2005a, p. 49).

Not only are the PLAY and SEEKING systems strongly implicated in chemical addictions, but growing evidence suggests they may be involved in problem gambling as well. For example, drugs that block brain opioid receptors (e.g., nalmefene, naltrexone) may reduce some of the pleasurable high of gambling. Accordingly, initial clinical trials suggest that these drugs may be beneficial for the treatment of problem gambling (Grant et al., 2006; Kim, Grant, Adson, & Shin, 2001). Neuroimaging studies have shown that anticipation and receipt of money—core aspects of gambling—activate brain regions rich in dopamine in healthy volunteers (Knutson, Westdorp, Kaiser, & Hommer, 2000). Participation in a gambling-like task that yields rewards also activates the brain dopamine system in problem gamblers, and the degree of activation is inversely related to the severity of gambling symptoms (Reuter et al., 2005). In other words, pathological gambling is associated with deficits in the ability of gambling to activate dopamine. This may explain tolerance to low-intensity gambling activity and the progressive escalation in risky, high-stakes betting that characterize pathological gambling. Other research has tested the hypothesis that pharmacological activation of the SEEKING system can prime the motivation to gamble. In one study, the psychostimulant drug amphetamine was found to increase self-reported desire to gamble and to decrease confidence to refrain from gambling in problem gamblers, effects that were not seen in healthy control subjects or problem drinkers with no history of gambling problems (Zack & Poulos, 2004).

The idea that the PLAY and SEEKING systems are involved in gambling makes intuitive sense. It also provides the basis for testing interventions to modify these behaviors. This is a critical issue because a viable animal model of gambling has thus far proven elusive. Conventional behavioral neuroscience approaches do not appear to capture some of the essential features of gambling. For example, although operant responding and delayed extinction under an intermittent reinforcement schedule characterize the persistent pattern of gambling that occurs in some gamblers, they fail to capture the inherent risk of loss entailed by each new trial in a gambling situation. In contrast, foraging (SEEKING) in unfamiliar, potentially dangerous environments appears to capture deliberate risk-taking with a view toward the prospect of eventual gain.

An important implication of an affective neuroscience formulation of gambling is that aversive feelings would be expected to accompany the absence of gambling in someone dependent upon it. Thus, if activation of the opiate system characterizes the experience of gambling, deactivation of this system would be predicted to characterize the experience of gambling withdrawal. Based on Panksepp's research on play and social attachment, gambling withdrawal would be expected to involve feelings of social distress, grief, and loss. If so, high rates of depression in problem gamblers may derive not only from the distress of economic and interpersonal hardship but also from neurochemical deficits occasioned by opiate-like withdrawal from gambling.

With respect to the SEEKING system, gambling withdrawal would be expected to involve feelings of boredom or restlessness: an uncomfortable state of disengagement with the world. Clearly, these aversive states could motivate gambling, particularly in those familiar with its palliative effect. In line with this, recent evidence has shown that partial deactivation of the dopamine system by a drug increases the pleasurable effects of an actual gambling episode along with post-game desire to gamble in problem gamblers (Zack, Poulos, & Desmond, 2004).

A related implication of Panksepp's model is that the incentive value of gambling should increase during periods of *non-gambling-related* deficits in dopamine and opioid function. Thus, just as eating is especially pleasurable when food is scarce, a suppressed SEEKING system would make gambling especially pleasurable. And just as freedom is especially valued when one has been constrained, a suppressed PLAY system would make gambling especially valuable. The recent devastating floods in New Orleans provide a real-world example of such effects. The pervasive destruction incurred by hurricane Katrina would make foraging a futile exercise; there is nothing to find. Similarly, the loss of home and possessions would have shaken one's sense of security and, as the days passed without respite, led to feelings of PANIC. For people in this situation, gambling could provide powerful relief: hope to the SEEKERS and comfort to the PANIC stricken. Within this framework, it is not surprising that “compared to the pre-Katrina world of November 2004, casino revenues in Lake Charles were up 41 percent, in New Orleans were up 63 percent and in Baton Rouge were up 69 percent last month [November 2005]. Overall, Louisiana saw a 32 percent increase even though three casinos still are closed as a result of the storms” (“Louisiana business shorts,” 2005). Increased gambling in Louisiana may partly reflect displaced demand due to the closure of riverboat casinos in Mississippi. Such “migratory” gambling would be consistent with the targeted SEEKING model and the presumed increase in the incentive value of gambling in the face of disaster. Clearly, an affective neuroscience explanation is only one of many possible ways to interpret these events. Nevertheless, this approach has the advantage of predicting the kinds of

interventions that should reduce disaster-related gambling, namely those that restore dopamine and opioid function. While drugs may accomplish this, genuine compassion from official parties and engagement of citizens in the rebuilding process would seem to be the best real-world antidotes.

The affective neuroscience model helps to explain some of the proximal causes of gambling. It also suggests which individuals, among those exposed to these causes, will escalate to problem gambling, namely those whose SEEKING and PLAY systems are inherently fragile. In line with this, the literature shows that individuals with genetic deficits in dopamine (D2) receptor function are significantly more prone to problem gambling than those without such deficits (Comings et al., 1996). Other research has found that “repeated periods of MS [maternal separation] early in life in male Wistar rats … induce long-lasting and possibly permanent alterations in the opioid peptide systems” (Ploj, Roman, & Nylander, 2003, p. 149). That such changes may be pathogenic is supported by the finding that pathological gamblers report significantly lower levels of parental bonding and parental care compared to healthy controls (Grant & Kim, 2002). Thus, both nature and nurture appear to sculpt the neural circuitry that promotes or protects against pathological gambling.

The brief overview of findings provided above highlights the importance of primary affective states as a basis for guiding research on gambling. The affective neuroscience model also has important implications for how we might approach gambling addiction at the clinical and social levels. In this regard, Panksepp notes, “if people's deepest feelings of social attachment are related to molecules that can also mediate drug addiction, then ‘wars on drugs’ may need to recognize certain painful psychobiological realities to become more effective. For instance, if people take opiates [or gamble] not just for superficial thrills but to achieve emotional homeostasis (Baker et al., 2004) [Baker, Piper, McCarthy, Majeskie, & Fiore, 2004], then addiction proneness will be related to how well prevailing social structures allow individuals to navigate the painful emotional passages of their lives” (Panksepp, 2005b, p. 228). By this reasoning, public health would be well served if agencies that profit from gambling reinvest their gains into socioeconomic opportunities, services, and supports that might obviate some people's need to gamble to induce artificially those states they cannot achieve naturally in their daily lives.

Statement of purpose

The *Journal of Gambling Issues* (*JGI*) offers an Internet-based forum for developments in gambling-related research, policy and treatment as well as personal accounts about gambling and gambling behaviour. Through publishing peer-reviewed articles about gambling as a social phenomenon and the prevention

and treatment of gambling problems, it is our aim is to help make sense of how gambling affects us all.

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