Gambling Disorder

Andreas Heinz
Nina Romanczuk-Seiferth
Marc N. Potenza
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Andreas Heinz
Department of Psychiatry
and Psychotherapy
Charité – University Medicine Berlin
Berlin
Germany

Nina Romanczuk-Seiferth
Department of Psychiatry
and Psychotherapy
Charité – University Medicine Berlin
Berlin
Germany

Marc N. Potenza
Department of Psychiatry
Yale University
New Haven, CT
USA

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Contents

What Is an Addiction?

1

Andreas Heinz and Anne Beck

1.1 Introduction

When should behavioural problems such as pathological gambling be considered to be an addiction?

If we want to define addictive disorders, we can look at current classification systems, neurobiological findings and the intuitions that structure both clinical and biological research. In this chapter, we will start with the classification systems and their underlying ideas, discuss the plausibility of neurobiological correlates and consistency of respective findings and finally compare some key theories about addiction that are currently guiding research.

In ICD-10 as well as DSM-IV, substance-related addictions are characterized by the development of tolerance to the effects of the drug of abuse, the manifestation of withdrawal symptoms upon detoxification, strong craving to consume the drug (this criterion was only recently introduced in DSM-5 and was not previously listed in DSM-IV) and reduced control of drug intake (in DSM-IV and DSM-5, this criterion is differentiated into the aspect of long-term high drug intake on the one hand and unsuccessful attempts to reduce it on the other hand). Further criteria describe harmful consequences of drug intake as well as a substantial increase in time spent to acquire and consume the drug of abuse at the expense of other activities [1–3]. In ICD-10 and DSM-IV, harmful drug use in the absence of further key aspects of substance dependence was classified as a separate category, while in DSM-5, harmful use, associated social problems and impairment of important obligations have been included together with the previously listed symptoms of

A. Heinz (✉) · A. Beck
Department of Psychiatry and Psychotherapy, Charité—Universitätsmedizin Berlin, Berlin, Germany
e-mail: andreas.heinz@charite.de; anne.beck@charite.de

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addiction into a dimensional approach that classifies substance use disorders [2]. A rationale behind this decision was that in epidemiological studies, there is a continuous increase in drug-related problems rather than sharp boundaries distinguishing harmful use from addiction. On the other hand, it has been criticized that if DSM-5 criteria are applied, legal restrictions (e.g. due to alcohol being illegal in many countries) can turn the desire to consume a glass of wine in the evening into a substance use disorder if acquiring and consuming the illegal drug causes social problems and, for example, due to incarceration, impairs performance in accordance with important social obligations [4]. With respect to non-substance-related disorders, DSM-5 was the first classification system to include certain behavioural syndromes in the wider category of addiction [2]. More specifically, pathological gambling, which in DSM-IV was classified as a disorder of impulse control, is now included in this wider addiction category. Transferring the concepts of substance-related addiction into the area of behavioural syndromes, tolerance development can be compared to the observation of increasing amounts of money required to satisfy the gambler's desire to participate in the game, withdrawal symptoms can be represented by restlessness and dysphoria when gambling is interrupted, craving and a rather large amount of time dedicated to the addiction can be indicated by a gambler being preoccupied with his or her game, and loss of control can be reflected in unsuccessful attempts to control or even stop gambling. Further rather specific syndromes associated with pathological gambling are chasing losses, i.e. the attempt to regain larger amounts of money previously lost in the gamble by increasing stakes, the use of gambling as a maladaptive tool to cope with negative emotions as well as the reliance on others to provide enough money to continue gambling. In accordance with the dimensional approach of DSM-5, social problems and impaired role performance, which previously represented criteria for harmful addictive behaviour, are now also included as criteria used to classify gambling disorder as an addiction.

Current classification systems claim that all symptoms are to be treated equal and that the presence or absence of two or more symptoms fulfilling the criteria listed above suffices to diagnose an addiction [2]. However, it is immediately clear that with respect to neurobiological research, some of the symptoms listed above have rather clear-cut biological correlates, while others are so deeply embedded in social interactions and legislation that the search for neurobiological correlates appears to be not only hopeless but also misguided. A famous example is the now abolished criterion "repetitive problems with the law", which was reflected in "illegal acts associated with gambling" in the former classification of pathological gambling in DSM-IV. But even beyond such descriptions of problematic behaviour that clearly depend upon legislation, social problems and impairments in role performance depend very strongly on cultural and social settings as well as demands on the individual [4]. The same is true with respect to the time required to get and consume a drug of abuse or to participate in gambling: This criterion is strongly influenced by the availability of the desired acts, which is of course reduced when the substance is illegal or gambling is prohibited. Accordingly, neurobiological research has largely focussed on the development of tolerance, withdrawal

symptoms associated with the sudden interruption of drug intake or gambling, craving for the addictive behaviour or drug of abuse as well as reduced control in dealing with drug intake or gambling [5–8].

1.2 Addiction Versus Dependence: Conceptual Changes

Traditionally, there has been a shift in focus when dealing with addictive disorders: Some decades ago, Edwards focussed on the "dependence" aspect of addictive behaviours, suggesting that tolerance development and withdrawal symptoms are at the core of drug-related problems [9]. Specifically, Edwards [9] suggested that all drugs of abuse cause biological alterations when chronically consumed, which result in withdrawal symptoms once their intake is stopped. In accordance with this hypothesis, Koob and Le Moal suggested that such neuroadaptive changes due to chronic drug intake result in establishing a new homeostasis, which depends on the continuation of drug consumption [5]. For example, alcohol stimulates GABAergic inhibition in the brain [10], and a long-term downregulation of GABA-A receptors has been observed in detoxified alcohol-dependent patients [10–12]. This downregulation of GABA-A receptors apparently balances the inhibitory effects of alcohol on GABA-A receptors. However, once alcohol intake is suddenly stopped, for example, in severe alcohol dependence during night sleep, GABA-A receptors remain downregulated, while there is a lack of the inhibitory effect of the drug of abuse. The loss of homeostasis represents a dysbalance between excitation and inhibition and contributes to withdrawal symptoms such as seizures [13]. Furthermore, if such inhibitory drug effects interact with second messenger systems in core areas of the autonomic nervous system including the locus coeruleus, impaired inhibition of this brain area can contribute to vegetative withdrawal symptoms [14–16]. According to Edwards [9], such withdrawal symptoms regularly occur following chronic drug intake and are a hallmark of substance dependence [9]. Moreover, Edwards [9] suggested to focus on the dependence aspect of addictions, because the term "addict" itself can have stigmatizing effects. Today, we see a shift of the research focus away from questions of drug tolerance and dependence towards what is considered to be key aspects of addition, i.e. strong drug craving and loss of control [2]. This shift of focus enabled the American Psychiatric Association to classify pathological gambling as an addiction: Tolerance development and withdrawal symptoms are particularly strong if the consumed drug of abuse has inhibitory effects on certain brain areas including the autonomic nervous system. Gambling and other addictive behaviours, however, are usually not sedative and—unlike drugs of abuse—do not directly interfere with inhibitory and excitatory systems in the central nervous system [5, 17, 18]. While there can be dysphoria and restlessness in gamblers who are suddenly interrupted when participating in their game or when being confronted with gambling machines they are not allowed to use [19, 20], such withdrawal symptoms are usually rather mild and hard to distinguish from some aspects of craving for the behaviour. Therefore, both research on non-substance-related addictions including gambling and a current neurobiological focus on brain

areas and neurotransmitter systems associated with motivation and executive control shifted research on substance dependence towards the "addiction" aspect, i.e. craving and control impairment.

1.3 Key Neurobiological Findings in Addiction Research

Indeed, neurobiological research on addiction has its most consistent findings with respect to the correlates of drug craving and a bias towards drug intake at the expense of other activities, which—when consciously not accepted and accompanied by claims that the person actually wants to do otherwise—often counts as an indicator of control impairment. Further aspects of control impairment include impulsive responding to small rewards that are immediately available instead of waiting for larger rewards and by impaired motor control when having to interrupt a motor tendency to respond to certain stimuli [21]. However, these different aspects of impulsivity often do not correlate at all with each other, questioning the concept of "impulsivity" as a coherent and useful construct in addiction research [22].

More consistent results have been acquired with respect to drug craving and aspects of loss of control associated with dopamine dysfunction in the ventral striatum and further brain areas associated with the so-called reward system [23, 24]. Indeed, all drugs of abuse release dopamine in the ventral striatum and thus reinforce drug consumption [25]. Unlike natural reinforcers, drugs of abuse continue to release dopamine upon re-exposure; thus the dopamine response to drugs fails to habituate. Moreover, dopamine release associated with drugs of abuse is usually much higher than dopamine release associated with natural reinforcers [25–29]. However, direct evidence for sensitized or increased dopamine release, as postulated by some addiction theories [30, 31], is hard to verify in humans, because even functional magnetic resonance imaging with its time frame of seconds is not able to track phasic dopamine release alterations appearing in the range of milliseconds [32]. However, recent research suggest that such short bursts of dopamine release indeed activate the ventral striatum as measured with optogenetic functional magnetic resonance imaging in awake rodents [33], thus suggesting that studies on cue-induced functional activation of the ventral striatum elicited by drug versus neutral or nondrug reward anticipatory cues indeed reflect alterations in dopaminergic signalling.

It is quite plausible that certain gambles and other addictive behaviours repetitively and unphysiologically strongly activate dopamine release in the ventral striatum, and in accordance with this hypothesis, indirect evidence for ventral striatal dysfunction in gambling has been reported [6, 34–37]. In this context, compensatory downregulation of dopamine receptors in the ventral striatum and blunting of functional activation of this brain area elicited by non-addictive reward-indicating cues has repeatedly been observed [6, 35, 38–40]. Such alterations in dopaminergic neurotransmission and the associated functional activation have often but not always been associated with the experience of subjective craving [41–43]. Craving, however, is a conscious process reported by the individual and requires a certain degree of self-reflection and openness towards one's own experiences as well as

interpersonal trust for sharing it with an observer. More direct ways to assess drug craving are measures of implicit drug approach tendencies as observed with the alcohol approach-avoidance task (alcohol AAT [44, 45]), where it has been observed that, for example, alcohol-dependent patients tend to pull alcohol cues towards themselves and need more time when required to push them away compared with nondrug-related stimuli [44, 45].

While there is some evidence that alterations in the so-called reward system contribute to craving for drugs as well as non-substance-related addictions including gambling, there is less consistent neurobiological findings with respect to the clinical symptom of impaired control over drug intake or gambling. An aspect of loss of control that is directly related to reward system alterations in addiction is given by an unconscious bias of behaviour towards drug consumption or pathological gambling at the expense of other activities. Such behavioural biases may be due to the fact that drugs of abuse as well as behavioural addictions activate dopamine release in the ventral striatum more strongly than natural reinforcers [25], thus reinforcing drug consumption or pathological gambling more strongly. Furthermore, increased presynaptic dopamine release can lead to neuroadaptive alterations, e.g. in the availability of dopamine D2 receptors, as observed for chronic alcohol intake in rodents [46] as well as in human alcohol-dependent patients [41, 42, 47]. Downregulation of dopamine receptors may help to explain why natural reinforcers fail to activate the ventral striatum in addicted subjects [39, 40, 48]. However, why do drug-associated cues continue to activate the ventral striatum in many studies [40, 49, 50]? Animal experiments have shown that cues that predict reward are attributed with the same salience and motivational value as the reward itself, due to a shift of phasic dopamine release from reward reception to the surprising presentation of the conditioned cue that reliably predicts reward [32, 51]. Drug cues, which are associated with high drug reward, could thus cause increased ventral striatal activation due to such conditioning processes [52]. Beyond such conditioning processes, Robinson and Berridge [31] suggested that drugs "sensitize" dopamine release [31], with repeated drug use being associated with increased psychomotor stimulant properties of, e.g., cocaine and drug cues, which elicit increased dopamine release in individuals that tend to react strongly to reward-associated stimuli [53, 54]. If such experiments in animals can be transferred to humans, it is quite plausible that some but not all individuals are prone to strongly react to drug-associated cues. On the other hand, salience attribution per se should not be confounded with a strong approach bias towards the drug of abuse. In fact, Beck et al. [49] observed that functional activation of both the amygdala and the ventral striatum was increased in patients who prospectively remained abstinent rather than relapsed to alcohol use [49]. Increased amygdala activation, which was functionally connected to the centre of origin of dopaminergic neurons in the brainstem, may also help to attribute salience towards potentially negative stimuli such as alcohol pictures in patients who consciously decided to remain abstinent. Observing increased activation of the ventral striatum in prospective abstainers but not relapsers was surprising but may also be due to salience attribution rather than eliciting an approach bias by activation of this limbic part of the striatum [55].

The effects of Pavlovian conditioned stimuli on unrelated instrumental choice behaviour can be assessed using Pavlovian-to-instrumental-transfer tasks, in which such Pavlovian cues are presented as background stimuli while performing an unrelated choice. Applying such studies in humans, Garbusow et al. observed that appetitive cues tend to increase approach behaviour and aversive cues tend to decrease approach behaviour to a larger degree in detoxified alcohol-dependent patients compared to healthy controls [56], and assessment of the effects of alcohol cues in such settings is currently carried out. Such studies may help to shed more light on the effects of drug cues on approach behaviour and, in a larger theoretical framework, on reduced control of addictive behaviour due to an unconscious bias of instrumental choice towards drugs or pathological gambling.

1.4 Executive Control and Addictive Behaviour

Neurobiological correlates of reduced control of drug intake or addictive behaviour have also been associated with impaired executive control functions. In alcohol dependence, neurotoxic effects of alcohol intake can contribute to cortical atrophy, particularly in the prefrontal cortex, and thus impair executive functions such as working memory [57, 58]. Again, such neurotoxic drug effects are hard to observe in pathological gambling, where cortical functioning is rather unimpaired on a structural level. Also, studies in subjects at risk failed to reveal impaired frontocortical control functions and rather pointed to a bias of information processing towards drug-associated choices in association with ventral striatal activation [59]. Furthermore, some studies in alcohol-dependent patients suggest that bottom-up information processing from the ventral striatum to the prefrontal cortex rather than top-down control of motivational systems by the prefrontal cortex is impaired in alcohol dependence [60]. These observations are in line with current studies emphasizing the role of the ventral striatum in cortico-striatal-thalamic neurocircuits, which regulate complex behaviour [61, 62]. Altogether, impaired control is a key concept of addictive behaviour; however, whether there are clinically relevant alterations in non-substance-related addictions with respect to cortical control functions remains to be explored in more depth.

1.5 Addictions Versus Compulsions

So far, these considerations suggest that drug addiction is characterized by a bias of information processing particularly in the so-called reward system towards drug consumption and drug-associated cues. With respect to pathological gambling, there has also been observed reduced activation of the ventral striatum by non-gambling-related stimuli predicting financial reward (e.g. [6, 34]), while increased activation of various brain areas including the prefrontal cortex has been observed by drug-associated stimuli in pathological gambling (e.g. [63]). Do these neurobiological correlates suggest that addictions are specific types of compulsions, i.e.

which similarities and differences can be identified when comparing obsessive-compulsive (OCD) and addictive disorders?

It has long been shown that human choice behaviour largely depends upon information processing in fronto-striatal-thalamic neurocircuits [64]. With respect to drug addiction, Volkow and others have repeatedly observed that there is reduced glucose utilization in the frontal cortex in different substance-related addictions [65–67]. OCD, on the other hand, has been associated with increased glucose utilization in the frontal cortex and associative striatum [68–70]. More recent studies with functional magnetic resonance imaging revealed that different obsessive-compulsive behaviours such as washing or hording are associated with specific fronto-striatal-thalamic networks, which also include activation of further limbic brain areas such as the anterior insula [71]. Exposure to drug-related cues has also been associated with brain activation patterns inside and outside of fronto-striatal-thalamic networks [72]. However, the direction of the respective changes appears to be different between OCD and addiction, with increased long-term glucose utilization in the frontal cortex being observed in obsessive-compulsive disorders, while these brain areas are rather hypoactive in addiction except when momentarily activated by drug-associated cues [49, 73]. Clinically, we and others have observed that compulsions are rather permanently manifesting repetitive actions, which phenomenologically differ considerably from cue-induced drug craving and consumption [74, 75]. Therefore, while craving and drug consumption can be experienced as rather "compulsive" by patients, neurobiological similarities are limited and substantial differences are evident.

1.6 Summary and Outlook

Altogether, this review of neurobiological correlates of key symptoms of addiction suggests that tolerance development and withdrawal symptoms constitute core aspects of addiction [9]; however, such symptoms are usually rather mild in non-substance-related addictions due to the rather low sedative effects of such activities. Strong craving and a bias towards addictive behaviour, on the other hand, have repeatedly been associated with altered functional activation of neural circuits known to regulate choice behaviour, which strongly rely on ventral striatal activation embedded in circuits including the prefrontal cortex and thalamus as well as further limbic brain areas [76]. Such alterations can bias behaviour towards the drug of choice or the preferred addictive behaviour at the expense of other activities. However, we should be careful to rely on craving and reduced control as the only indicators of addictive behaviour: Every passion including dedicated research or romantic love can be characterized by strong craving and a certain focus of attention on this activity at the expense of others [77]. Therefore, it does not suffice to label some activities as helpful and others as harmful, because then social and legal tendencies, e.g. to ban a certain drug including alcohol from public consumption or to prohibit gambling, decide whether a certain behaviour is an addiction or not. Conceding this would mean that dominating morals and legislation and not

medical criteria decide what behavioural syndromes constitute a clinically relevant mental malady. To avoid such confounds with changing morals, the diagnosis of a clinically relevant mental malady should, in our view, require that two of three criteria are fulfilled:

The first one is a decision on whether certain symptoms of a disorder are medically relevant, i.e. whether they can *generally* impair human life to a relevant degree (the disease criterion). This decision is not one based on natural science evidence but rather on plausibility and common sense. Not being able to roll your tongue is not a disease, because you do not need to roll your tongue to survive as a human being, while being unable to swallow is a symptom of a disease, because as humans we need to consume food to survive.

The second and third one depend upon the individual assessment of the consequences of these symptoms, i.e. do they harm the person by causing suffering (the illness criterion) or a severe limitation of social participation (the sickness criterion [77]).

With respect to key symptoms guiding the diagnosis of a disease, developing tolerance to a drug of abuse and showing withdrawal symptoms that can be lethal as in delirium tremens are clearly symptoms of a disease, because their manifestation can be life threatening. Other aspects of addictions such as strong craving and loss of control do not directly jeopardize human survival but can severely impair human life with others [77]. Assessing whether this is indeed the case, value judgements play a stronger role than when assessing withdrawal symptoms. Therefore, we have to be careful not to exclusively rely on symptoms such as craving and reduced control when diagnosing an addiction. Kant [78] has suggested that addictions are always characterized by a certain disinterest in another human being as an independent person with his or her own goals and way of life [78], and we suggest that beyond craving and loss of control, behavioural addictions are characterized by such a reduced interaction with other human beings. However, we warn that all these assessments strongly rely on contemporary value judgements and may be revised in the future in more tolerant or less liberal societies. Therefore, diagnosing an addiction in the absolute absence of tolerance development and withdrawal symptoms may not be recommendable and we indeed do not recommend to do so. We have suggested that other criteria to diagnose a medically relevant disease including harm to the person's health or role functioning or an increased amount of time necessary to acquire the drug of abuse or to gamble are of limited value: Physical harm, e.g. resulting from liver toxicity of alcohol intake, can be objectified quite easily, while harmful effects on social interactions depend on legalization or punishment of drug consumption or gambling and also affect the time required to acquire or consume a drug of abuse or to find a place to gamble. Therefore, we feel that at the core of the medical diagnosis of a disease, a general impairment of mental functions relevant for human life needs to be diagnosed, and this diagnosis should rely on core aspects of addictions including tolerance development, withdrawal symptoms, craving and impaired control of the respective behaviour.

Furthermore, we suggest that diagnosing symptoms that indicate that a medical disease is present (the disease criterion) does not suffice to actually diagnose a clinically relevant mental malady. There are human beings who show clear

indications of medically relevant dysfunctions including acoustic hallucinations, who neither suffer from them nor are impaired in their common performance of daily activities [79]. Therefore, beyond the medical disease aspect, the individual has to either suffer from these symptoms (the illness criterion) or be severely impaired in his or her social participation (the sickness criterion), particularly with respect to activities of daily living such as personal hygiene or food consumption etc. [77]. Beyond the assessment of generally relevant medical symptoms, any diagnosis of a clinically relevant disorder thus needs to also assess the personal consequences of such symptoms including individual suffering or the impairment to cope with activities of daily living [77]. We emphasize such a cautious approach to diagnosis in order to avoid that dictatorships or other ideological groups can start defining any unwanted behaviour as an addiction, for example, critical blogging in the Internet or, as was the case in ninetieth century, the attempts to escape from slavery as drapetomania [80, 81]. Behavioural addictions can have a profound negative impact on the life of the afflicted subjects; however, we have to make sure that diagnosing such an addiction is not abused to label socially unwanted behaviour, which is performed by individuals in spite of negative social pressure, as a mental malady. Therefore, the cautious approach of the American Psychiatric Association [2], which only classified gambling as an addictive disorder and abstained from labelling more behavioural syndromes including involvement in excessive sexual contacts or shopping, is quite warranted [2]. We hope that this book and its review of clinical as well as neurobiological findings on behavioural addictions will help to promote such a cautious and rational approach towards behavioural addictions.

References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed., text rev. edn. Washington, DC; 2000.
2. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-5). Washington, DC and London: American Psychiatric Publishing; 2013.
3. World Health Organization. ICD-10, international statistical classification of diseases and related health problems. 10th revision edn. Genf; 2013.
4. Heinz A, Friedel E. DSM-5: important changes in the field of addictive diseases. Nervenarzt. 2014;85(5):571–7.
5. Koob GF, Le Moal M. Drug abuse: hedonic homeostatic dysregulation. Science. 1997;278(5335):52–8.
6. Romanczuk-Seiferth N, Koehler S, Dreesen C, Wustenberg T, Heinz A. Pathological gambling and alcohol dependence: neural disturbances in reward and loss avoidance processing. Addict Biol. 2015;20(3):557–69.
7. Romanczuk-Seiferth N, van den Brink W, Goudriaan AE. From symptoms to neurobiology: pathological gambling in the light of the new classification in DSM-5. Neuropsychobiology. 2014;70(2):95–102.
8. Volkow ND, Fowler JS. Addiction, a disease of compulsion and drive: involvement of the orbitofrontal cortex. Cereb Cortex. 2000;10(3):318–25.
9. Edwards G. Withdrawal symptoms and alcohol dependence: fruitful mysteries. Br J Addict. 1990;85(4):447–61.

10. Krystal JH, Staley J, Mason G, Petrakis IL, Kaufman J, Harris RA, Gelernter J, Lappalainen J. Gamma-aminobutyric acid type A receptors and alcoholism: intoxication, dependence, vulnerability, and treatment. Arch Gen Psychiatry. 2006;63(9):957–68.
11. Clapp P, Bhave SV, Hoffman PL. How adaptation of the brain to alcohol leads to dependence: a pharmacological perspective. Alcohol Res Health. 2008;31(4):310–39.
12. De Witte P. Imbalance between neuroexcitatory and neuroinhibitory amino acids causes craving for ethanol. Addict Behav. 2004;29(7):1325–39.
13. Heinz A, Beck A, Wrase J, Mohr J, Obermayer K, Gallinat J, Puls I. Neurotransmitter systems in alcohol dependence. Pharmacopsychiatry. 2009;42(Suppl 1):S95–S101.
14. Akaoka H, Aston-Jones G. Opiate withdrawal-induced hyperactivity of locus coeruleus neurons is substantially mediated by augmented excitatory amino acid input. J Neurosci. 1991;11(12):3830–9.
15. Maldonado R, Stinus L, Gold LH, Koob GF. Role of different brain structures in the expression of the physical morphine withdrawal syndrome. J Pharmacol Exp Ther. 1992;261(2):669–77.
16. Rasmussen K, Beitner-Johnson DB, Krystal JH, Aghajanian GK, Nestler EJ. Opiate withdrawal and the rat locus coeruleus: behavioral, electrophysiological, and biochemical correlates. J Neurosci. 1990;10(7):2308–17.
17. Koob GF, Volkow ND. Neurocircuitry of addiction. Neuropsychopharmacology. 2010;35(1):217–38.
18. Tsai G, Gastfriend DR, Coyle JT. The glutamatergic basis of human alcoholism. Am J Psychiatry. 1995;152(3):332–40.
19. Chamberlain SR, Lochner C, Stein DJ, Goudriaan AE, van Holst RJ, Zohar J, Grant JE. Behavioural addiction—a rising tide? Eur Neuropsychopharmacol. 2016;26(5):841–55.
20. Grusser SM, Poppelreuter S, Heinz A, Albrecht U, Sass H. Behavioural addiction: an independent diagnostic category? Nervenarzt. 2007;78(9):997–1002.
21. De Wit H. Impulsivity as a determinant and consequence of drug use: a review of underlying processes. Addict Biol. 2009;14(1):22–31.
22. Rupp CI, Beck JK, Heinz A, Kemmler G, Manz S, Tempel K, Fleischhacker WW. Impulsivity and alcohol dependence treatment completion: is there a neurocognitive risk factor at treatment entry? Alcohol Clin Exp Res. 2016;40(1):152–60.
23. Breiter HC, Aharon I, Kahneman D, Dale A, Shizgal P. Functional imaging of neural responses to expectancy and experience of monetary gains and losses. Neuron. 2001;30(2):619–39.
24. Knutson B, Adams CM, Fong GW, Hommer D. Anticipation of increasing monetary reward selectively recruits nucleus accumbens. J Neurosci. 2001;21(16):RC159.
25. Di Chiara G, Bassareo V. Reward system and addiction: what dopamine does and doesn't do. Curr Opin Pharmacol. 2007;7(1):69–76.
26. Di Chiara G, Imperato A. Drugs abused by humans preferentially increase synaptic dopamine concentrations in the mesolimbic system of freely moving rats. Proc Natl Acad Sci U S A. 1988;85(14):5274–8.
27. Fuchs H, Nagel J, Hauber W. Effects of physiological and pharmacological stimuli on dopamine release in the rat globus pallidus. Neurochem Int. 2005;47(7):474–81.
28. Gessa GL, Muntoni F, Collu M, Vargiu L, Mereu G. Low doses of ethanol activate dopaminergic neurons in the ventral tegmental area. Brain Res. 1985;348(1):201–3.
29. Martel P, Fantino M. Influence of the amount of food ingested on mesolimbic dopaminergic system activity: a microdialysis study. Pharmacol Biochem Behav. 1996;55(2):297–302.
30. Flagel SB, Robinson TE, Clark JJ, Clinton SM, Watson SJ, Seeman P, Phillips PE, Akil H. An animal model of genetic vulnerability to behavioral disinhibition and responsiveness to reward-related cues: implications for addiction. Neuropsychopharmacology. 2010;35(2):388–400.
31. Robinson TE, Berridge KC. The neural basis of drug craving: an incentive-sensitization theory of addiction. Brain Res Brain Res Rev. 1993;18(3):247–91.
32. Schultz W, Dayan P, Montague PR. A neural substrate of prediction and reward. Science. 1997;275(5306):1593–9.
33. Ferenczi EA, Zalocusky KA, Liston C, Grosenick L, Warden MR, Amatya D, Katovich K, Mehta H, Patenaude B, Ramakrishnan C, Kalanithi P, Etkin A, Knutson B, Glover GH,

Deisseroth K. Prefrontal cortical regulation of brainwide circuit dynamics and reward-related behavior. Science. 2016;351(6268):aac9698.
34. Balodis IM, Kober H, Worhunsky PD, Stevens MC, Pearlson GD, Potenza MN. Diminished frontostriatal activity during processing of monetary rewards and losses in pathological gambling. Biol Psychiatry. 2012;71(8):749–57.
35. Balodis IM, Potenza MN. Imaging the gambling brain. Int Rev Neurobiol. 2016;129:111–24.
36. Quester S, Romanczuk-Seiferth N. Brain imaging in gambling disorder. Curr Addict Rep. 2015;2(3):220–9.
37. Reuter J, Raedler T, Rose M, Hand I, Glascher J, Buchel C. Pathological gambling is linked to reduced activation of the mesolimbic reward system. Nat Neurosci. 2005;8(2):147–8.
38. Beck A, Schlagenhauf F, Wüstenberg T, Hein J, Kienast T, Kahnt T, Schmack K, Hägele C, Knutson B, Heinz A, Wrase J. Ventral striatal activation during reward anticipation correlates with impulsivity in alcoholics. Biol Psychiatry. 2009;66(8):734–42.
39. Hagele C, Schlagenhauf F, Rapp M, Sterzer P, Beck A, Bermpohl F, Stoy M, Strohle A, Wittchen HU, Dolan RJ, Heinz A. Dimensional psychiatry: reward dysfunction and depressive mood across psychiatric disorders. Psychopharmacology. 2015;232(2):331–41.
40. Wrase J, Schlagenhauf F, Kienast T, Wustenberg T, Bermpohl F, Kahnt T, Beck A, Strohle A, Juckel G, Knutson B, Heinz A. Dysfunction of reward processing correlates with alcohol craving in detoxified alcoholics. NeuroImage. 2007;35(2):787–94.
41. Heinz A, Siessmeier T, Wrase J, Buchholz HG, Grunder G, Kumakura Y, Cumming P, Schreckenberger M, Smolka MN, Rosch F, Mann K, Bartenstein P. Correlation of alcohol craving with striatal dopamine synthesis capacity and D2/3 receptor availability: a combined [18F]DOPA and [18F]DMFP PET study in detoxified alcoholic patients. Am J Psychiatr. 2005;162(8):1515–20.
42. Heinz A, Siessmeier T, Wrase J, Hermann D, Klein S, Grusser SM, Flor H, Braus DF, Buchholz HG, Grunder G, Schreckenberger M, Smolka MN, Rosch F, Mann K, Bartenstein P. Correlation between dopamine D(2) receptors in the ventral striatum and central processing of alcohol cues and craving. Am J Psychiatry. 2004;161(10):1783–9.
43. Volkow ND, Wang GJ, Telang F, Fowler JS, Alexoff D, Logan J, Jayne M, Wong C, Tomasi D. Decreased dopamine brain reactivity in marijuana abusers is associated with negative emotionality and addiction severity. Proc Natl Acad Sci U S A. 2014;111(30):E3149–56.
44. Wiers CE, Stelzel C, Park SQ, Gawron CK, Ludwig VU, Gutwinski S, Heinz A, Lindenmeyer J, Wiers RW, Walter H, Bermpohl F. Neural correlates of alcohol-approach bias in alcohol addiction: the spirit is willing but the flesh is weak for spirits. Neuropsychopharmacology. 2014;39(3):688–97.
45. Wiers RW, Eberl C, Rinck M, Becker ES, Lindenmeyer J. Retraining automatic action tendencies changes alcoholic patients' approach bias for alcohol and improves treatment outcome. Psychol Sci. 2011;22(4):490–7.
46. Rommelspacher H, Raeder C, Kaulen P, Bruning G. Adaptive changes of dopamine-D2 receptors in rat brain following ethanol withdrawal: a quantitative autoradiographic investigation. Alcohol. 1992;9(5):355–62.
47. Volkow ND, Wang GJ, Fowler JS, Logan J, Hitzemann R, Ding YS, Pappas N, Shea C, Piscani K. Decreases in dopamine receptors but not in dopamine transporters in alcoholics. Alcohol Clin Exp Res. 1996;20(9):1594–8.
48. Garavan H, Pankiewicz J, Bloom A, Cho JK, Sperry L, Ross TJ, Salmeron BJ, Risinger R, Kelley D, Stein EA. Cue-induced cocaine craving: neuroanatomical specificity for drug users and drug stimuli. Am J Psychiatry. 2000;157(11):1789–98.
49. Beck A, Wustenberg T, Genauck A, Wrase J, Schlagenhauf F, Smolka MN, Mann K, Heinz A. Effect of brain structure, brain function, and brain connectivity on relapse in alcohol-dependent patients. Arch Gen Psychiatry. 2012;69(8):842–52.
50. Schacht JP, Anton RF, Myrick H. Functional neuroimaging studies of alcohol cue reactivity: a quantitative meta-analysis and systematic review. Addict Biol. 2013;18(1):121–33.
51. Schultz W. Dopamine reward prediction-error signalling: a two-component response. Nat Rev Neurosci. 2016;17(3):183–95.

52. Heinz A, Schlagenhauf F, Beck A, Wackerhagen C. Dimensional psychiatry: mental disorders as dysfunctions of basic learning mechanisms. J Neural Transm (Vienna). 2016;123(8):809–21.
53. Flagel SB, Akil H, Robinson TE. Individual differences in the attribution of incentive salience to reward-related cues: implications for addiction. Neuropharmacology. 2009;56(Suppl 1):139–48.
54. Flagel SB, Clark JJ, Robinson TE, Mayo L, Czuj A, Willuhn I, Akers CA, Clinton SM, Phillips PE, Akil H. A selective role for dopamine in stimulus-reward learning. Nature. 2011;469(7328):53–7.
55. Zink CF, Pagnoni G, Martin ME, Dhamala M, Berns GS. Human striatal response to salient nonrewarding stimuli. J Neurosci. 2003;23(22):8092–7.
56. Garbusow M, Schad DJ, Sebold M, Friedel E, Bernhardt N, Koch SP, Steinacher B, Kathmann N, Geurts DE, Sommer C, Muller DK, Nebe S, Paul S, Wittchen HU, Zimmermann US, Walter H, Smolka MN, Sterzer P, Rapp MA, Huys QJ, Schlagenhauf F, Heinz A. Pavlovian-to-instrumental transfer effects in the nucleus accumbens relate to relapse in alcohol dependence. Addict Biol. 2016;21(3):719–31.
57. Adams RA, Huys QJ, Roiser JP. Computational psychiatry: towards a mathematically informed understanding of mental illness. J Neurol Neurosurg Psychiatry. 2016;87(1):53–63.
58. Pfefferbaum A, Sullivan EV, Mathalon DH, Shear PK, Rosenbloom MJ, Lim KO. Longitudinal changes in magnetic resonance imaging brain volumes in abstinent and relapsed alcoholics. Alcohol Clin Exp Res. 1995;19(5):1177–91.
59. Stuke H, Gutwinski S, Wiers CE, Schmidt TT, Gropper S, Parnack J, Gawron C, Hindi Attar C, Spengler S, Walter H, Heinz A, Bermpohl F. To drink or not to drink: harmful drinking is associated with hyperactivation of reward areas rather than hypoactivation of control areas in men. J Psychiatry Neurosci. 2016;41(3):E24–36.
60. Park SQ, Kahnt T, Beck A, Cohen MX, Dolan RJ, Wrase J, Heinz A. Prefrontal cortex fails to learn from reward prediction errors in alcohol dependence. J Neurosci. 2010;30(22):7749–53.
61. Deserno L, Huys QJ, Boehme R, Buchert R, Heinze HJ, Grace AA, Dolan RJ, Heinz A, Schlagenhauf F. Ventral striatal dopamine reflects behavioral and neural signatures of model-based control during sequential decision making. Proc Natl Acad Sci U S A. 2015;112(5):1595–600.
62. Schlagenhauf F, Rapp MA, Huys QJ, Beck A, Wustenberg T, Deserno L, Buchholz HG, Kalbitzer J, Buchert R, Bauer M, Kienast T, Cumming P, Plotkin M, Kumakura Y, Grace AA, Dolan RJ, Heinz A. Ventral striatal prediction error signaling is associated with dopamine synthesis capacity and fluid intelligence. Hum Brain Mapp. 2013;34(6):1490–9.
63. Lorenz RC, Kruger JK, Neumann B, Schott BH, Kaufmann C, Heinz A, Wustenberg T. Cue reactivity and its inhibition in pathological computer game players. Addict Biol. 2013;18(1):134–46.
64. Alexander GE, DeLong MR, Strick PL. Parallel organization of functionally segregated circuits linking basal ganglia and cortex. Annu Rev Neurosci. 1986;9:357–81.
65. Adams KM, Gilman S, Koeppe RA, Kluin KJ, Brunberg JA, Dede D, Berent S, Kroll PD. Neuropsychological deficits are correlated with frontal hypometabolism in positron emission tomography studies of older alcoholic patients. Alcohol Clin Exp Res. 1993;17(2):205–10.
66. Volkow ND, Chang L, Wang GJ, Fowler JS, Ding YS, Sedler M, Logan J, Franceschi D, Gatley J, Hitzemann R, Gifford A, Wong C, Pappas N. Low level of brain dopamine D2 receptors in methamphetamine abusers: association with metabolism in the orbitofrontal cortex. Am J Psychiatry. 2001;158(12):2015–21.
67. Volkow ND, Wang GJ, Shokri Kojori E, Fowler JS, Benveniste H, Tomasi D. Alcohol decreases baseline brain glucose metabolism more in heavy drinkers than controls but has no effect on stimulation-induced metabolic increases. J Neurosci. 2015;35(7):3248–55.
68. Baxter LR Jr, Phelps ME, Mazziotta JC, Guze BH, Schwartz JM, Selin CE. Local cerebral glucose metabolic rates in obsessive-compulsive disorder. A comparison with rates in unipolar depression and in normal controls. Arch Gen Psychiatry. 1987;44(3):211–8.

69. Baxter LR Jr, Schwartz JM, Bergman KS, Szuba MP, Guze BH, Mazziotta JC, Alazraki A, Selin CE, Ferng HK, Munford P, et al. Caudate glucose metabolic rate changes with both drug and behavior therapy for obsessive-compulsive disorder. Arch Gen Psychiatry. 1992;49(9):681–9.
70. Schwartz JM, Stoessel PW, Baxter LR Jr, Martin KM, Phelps ME. Systematic changes in cerebral glucose metabolic rate after successful behavior modification treatment of obsessive-compulsive disorder. Arch Gen Psychiatry. 1996;53(2):109–13.
71. Mataix-Cols D, Wooderson S, Lawrence N, Brammer MJ, Speckens A, Phillips ML. Distinct neural correlates of washing, checking, and hoarding symptom dimensions in obsessive-compulsive disorder. Arch Gen Psychiatry. 2004;61(6):564–76.
72. Kuhn S, Gallinat J. Common biology of craving across legal and illegal drugs – a quantitative meta-analysis of cue-reactivity brain response. Eur J Neurosci. 2011;33(7):1318–26.
73. Goldstein RZ, Volkow ND. Dysfunction of the prefrontal cortex in addiction: neuroimaging findings and clinical implications. Nat Rev Neurosci. 2011;12(11):652–69.
74. Huys QJM, Beck A, Dayan P, Heinz A. Neurobiology and computational structure of decision making in addiction. In: al. Me, editor. Phenomenological neuropsychiatry: bridging the clinic and clinical neuroscience. Posted at the Zurich Open Repository and Archive, University of Zurich. 2013.
75. Schoofs N, Heinz A. Pathological gambling. Impulse control disorder, addiction or compulsion? Nervenarzt. 2013;84(5):629–34.
76. Heinz AJ, Beck A, Meyer-Lindenberg A, Sterzer P, Heinz A. Cognitive and neurobiological mechanisms of alcohol-related aggression. Nat Rev Neurosci. 2011;12(7):400–13.
77. Heinz A. Der Begriff der psychischen Krankheit. Berlin: Suhrkamp; 2014.
78. Kant I. Anthropologie in pragmatischer Hinsicht. Stuttgart: Reclam; 1983.
79. van Os J. "Schizophrenia" does not exist. BMJ. 2016;352:i375.
80. Beard DJ, Findlay JA. Drapetomania—a disease called freedom. FL: Broward Public Library Foundation; 2000.
81. Naragon MD. Communities in motion: drapetomania, work and the development of African American slave cultures. Slavery Abolition. 1994;15(3):63–87.

Gambling Disorder as a Clinical Phenomenon

2

Christopher J. Hunt and Alexander Blaszczynski

2.1 Games and Gambling in Antiquity

The exact origins of gambling have faded into obscurity but its presence dates to antiquity. Archaeological findings offer evidence of games of chance played as long back as approximately 4000 years BC. Murals and artefacts around this period indicate that board games such as the forerunners of draughts and backgammon and astragals (knucklebones) used as dice thrown to determine the number of steps to move playing pieces [1–3] were commonly accepted as leisure pursuits. The oldest known Eastern games of Wei-kin in China and Go in Japan emerged around 2300 years BC. These games relied on chance as the determinant of outcomes, but the exact point in time when players began to risk items of value either to enhance excitement in competition or for personal gain remains unknown. What is known is that reference to gambling can be found in ancient Egyptian mythical accounts of deities and demigods and in Mediterranean and Eastern culture folklores.

Indications are that many games laid the foundation for activities that subsequently met the definition of gambling, that is, an agreement between two or more participants to risk an item of value on the outcome of an event determined wholly or to some extent by chance for purposes of obtaining a gain/profit. Roulette, for example, has its origins in Grecian and Roman soldiers wagering on the turn of numbered chariot wheels; the throwing of dice and lots in appeal to religious divination represents the forerunner of modern dice games; legends about keno claim a history dating back to efforts to raise money to fund wars and build the Great Wall in ancient China; horse and chariot races later evolved into national wagering events; and simple early card games diverged into the multiple card game formats played today, such as poker, baccarat and blackjack. In contemporary times,

C. J. Hunt · A. Blaszczynski (✉)
Brain Mind Centre, School of Psychology, The University of Sydney, Sydney, NSW, Australia
e-mail: christopher.hunt@sydney.edu.au; alex.blaszczynski@sydney.edu.au

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technological and electronic advances have given rise to sophisticated electronic gambling devices mimicking traditional games, and the Internet offers global opportunities for virtually all forms of gambling.

Societal acceptance of gambling has fluctuated from extremes of widespread indulgence to attempted suppression for as long as gambling has been in existence. For example, Confucius (551–479 BC), whose philosophy formed the basis of much Chinese moral reasoning throughout subsequent centuries, reportedly referred to gambling as unproductive and as violating filial duty [4]. There is then evidence of legal proscriptions against gambling in China during the Warring States period (c. 476–221 BC) and during the Tang dynasty (c AD 618–907 [4]). Similar religious and legal restrictions on gambling in Europe were enacted in response to the social and economic impacts of excessive gambling: public disorder, creation of poverty and personal and familial distress, cheating and exploitation and as it was viewed as an activity contrary to Protestant work ethics or religious tenets [5, 6]. Accordingly, religious edicts prohibiting gambling and statutes banning certain activities, limiting losses or preventing recovery of gambling debts were enacted across many jurisdictions. By 1882, virtually every European province prohibited gambling [7] with the temperance movement in the latter part of that decade temporarily successful in tempering the consumption of alcohol and gambling in America. In the current era, the full circle has turned with gambling, although not universally adopted and accepted, becoming a multibillion dollar global industry, incorporating 24/7 convenient, anonymous and easy access to gaming and wagering products through multiple land-based options and via online devices (smartphones, tablets and laptops).

2.2 Gambling to Excess

Numerous anecdotal and case history accounts of individuals, including historical celebrities, falling prey to the lure of gambling have been chronicled over the ages [6]. Documented in these writings is the extent to which individuals wreaked havoc on their wealth, incurred debt leading to poverty and imprisonment in debtor's jail, destroyed marriages and families and succumbed to suicidal ideation [3, 6]. These accounts are insightful in describing the phenomenology associated with 'compulsive' urges driving an individual motivated by the desire to win to persist despite incurring substantial losses and severe emotional distress. The 'addictiveness' of the behaviour indicated by the presence of tolerance [8] and impaired control and preoccupation comparable to alcohol addiction [9] has been frequently described in the popular literature prior to the twentieth century. Exemplary descriptions of the powerful processes inherent in gambling are contained in Pushkin's *The Queen of Spades* [10], Dostoevsky's *The Gambler* [11], Thackeray's *A Gambler's Death* [12] and Saki's *The Stake* [13], a literature base that depicts the phenomenology of the behaviour in comprehensive detail. However, it was not until von Hattinger's [14] psychodynamic description of gambling was published that scientific consideration was given to the idea of excessive gambling representing a clinical phenomenon reflecting the presence of an underlying psychological disorder.

2.3 Gambling Disorder as a Clinical Phenomenon

Between 1914 and 1957, with continuing pockets of interest, psychodynamic explanations were applied to the aetiology of 'compulsive' gambling. Predominantly based on single case or case series reports, the condition was regarded as the symptomatic expression of an underlying psychoneurosis related to pregenital psychosexual phases and Oedipal conflicts, masturbatory complexes and equivalents or the expression of psychic masochism linked to a tendency for self-punishment resulting from unresolved aggressive feelings [15–17]. Although shaping its intervention, the psychodynamic formulation lacked empirical support, retained untestable hypotheses and failed to explain the transitional shift from recreational to impaired control, a process often taking several years. In addition, the gambling was typically not the primary reason for referral, leaving the causal or interactive relationship between the respective conditions unknown.

Derived from experimental manipulations of behaviour, learning theories gained popularity in the 1960s following the seminal studies of Skinner [18] and Pavlov [19] describing operant and classical conditioning paradigms, respectively. This provided an excellent model explaining how overt gambling behaviours were influenced by contingencies of random ratio-delivered schedules of reinforcement. Anderson and Brown [20] advanced a two-factor theory that incorporated operant and classical conditioning principles with individual differences in autonomic/cortical arousal and sensation-seeking personality traits. This theory was predicated on the assumption that certain individuals had a propensity to respond differently to rewards and punishment, with a proclivity to repetitively seek out risky behaviours to maintain optimal levels of hedonic arousal [21].

Jacobs [22] extended these concepts into his general theory of addictions that contained many of the inherent features of Solomon and Corbitt's [23] opponent process model. Briefly, Jacobs [22] argued that chronically hyper- (anxious) or hypo- (depressed) aroused individuals, in combination with psychological states of low self-esteem and experiences of rejection, placed such individuals at risk for pursuing behaviours that fostered homeostatic levels of arousal. Those hyper-aroused, it was suggested, gravitate to low-skills games where their attention is narrowed and focussed, resulting in negative reinforcement, that is, escaping from states of emotional distress [22, 24]. For those hypo-aroused, preferences were directed to higher skill games that engaged their interests resulting in excitement, boosting their affective states.

These early theories highlighted the central role played by biologically determined differences in psychophysiological arousal, the influence of positive and negative reinforcement and personality traits as vulnerability factors leading to a gambling disorder. Cognitive and motivational variables were recognized but did not attract the primary focus of attention at this point. However, cognitive theories gained prominence with the identification of consistent distorted and erroneous beliefs surrounding illusions of control, misunderstanding the mathematics and statistical basis of gambling and concepts of randomness and mutual independence of chance events [25–27]. Chasing losses as a motivation is one of the overarching

factors defining a gambling disorder as described by Lesieur [28]. Behavioural and cognitive theories are not mutually exclusive but contain behavioural and motivational components that interact with each other to maintain persistence despite serious deleterious consequences.

Given its repetitive persistent nature, it is unsurprising that analogies between gambling and substance addiction have been promulgated. This perspective was formalised in the DSM-IV [29], where the criteria for what was then termed 'pathological gambling' were revised to explicitly draw attention to the presence of many features commonly found in substance use disorders, including withdrawal symptoms, tolerance and preoccupation/dependence and affective disturbances [30].

Irrespective of the explanatory model applied, phenomenological features of emotional dependence on gambling, impaired control over behaviours, concurrent substance use and affective disturbances and persistence in the face of accumulating stresses and distress characterise gambling disorder as a clinical entity. Typical features include the presence of depression, suicidal ideation, anxiety and emotional distress, marital and familial conflicts, impaired work/study productivity, commission of illegal acts to maintain habitual gambling behaviours and substance use. Cognitive distortions result in individuals overestimating personal skills and probabilities of winning and lead to further attempts to recoup losses through continued gambling.

2.4 Current Diagnostic Criteria for Gambling Disorder

Although recognized as a clinical entity for over 40 years since its inclusion within ICD-9 [31] and DSM-III [32], debate regarding inconsistencies in the terminology used, categorization, and criteria used to diagnose a gambling disorder have been prevalent. In particular, gambling disorders have been variably considered to constitute an impulse control disorder, an addictive behaviour or fall on an obsessive-compulsive spectrum (see [33], for an overview). In the following section, the development of the current diagnostic criteria guided by phenomenological features that consolidate gambling as a clinical disorder will be outlined.

With the release of the DSM-5 [34], the following diagnostic criteria were given for the diagnosis now referred to as 'gambling disorder' (the earlier name of 'pathological gambling' was dropped as the term 'pathological' was considered to be pejorative [35]). In order to receive a diagnosis of a gambling disorder, individuals must meet four of the nine criteria over a 12-month period. Their behaviour must also not be better accounted for by a manic episode.

1. Needing to gamble with increasing amounts of money in order to achieve the desired excitement.
2. Feeling restless or irritable when attempting to cut down or stop gambling.
3. Making repeated unsuccessful attempts to control, cut back or stop gambling.
4. Often experiencing preoccupation with gambling (e.g. having persistent thoughts of reliving past gambling experiences, handicapping or planning the next venture, thinking of ways to get money to gamble).

5. Often gambles when feeling distressed (e.g. helpless, guilty, anxious, depressed).
6. After losing money gambling, often returns another day to get even ('chasing' one's losses).
7. Lies to conceal the extent of involvement with gambling.
8. Jeopardising or losing a significant relationship, job or educational or career opportunity because of gambling.
9. Has relied on others to provide money to relieve desperate financial situations caused by gambling.

As well as the aforementioned name change, these criteria represented several changes from the previous DSM-IV-TR criteria for pathological gambling [36]. Firstly, the diagnosis was moved to the section titled 'Substance Use and Related Disorders', where it is the sole member of a grouping titled 'non-substance-related disorders'. The DSM-5 workgroup on gambling cited research that highlighted clinical, neurological, epidemiological and genetic similarities between gambling and substance use disorders as the key reason for the move, although they noted that there were dissenting voices [37]. The research into the similarities and differences between gambling and substance use disorders will be discussed in detail later in this volume (see Chap. 12).

The second change that was made to the criteria in the DSM-5 was the dropping of the criterion included in past editions 'has committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling'. The workgroup reported that this criterion had been removed as only a minority of the treatment population endorsed this criterion, and those who did frequently also reported meeting multiple other criteria, thus diminishing this criterion's usefulness in the diagnosis of gambling disorder [37]. Other writers have disputed this change, noting that illegal acts remain relatively common in treatment samples of gamblers, and the retention of this criterion would draw attention to the relationship between gambling disorder and legal issues [38]. Indeed, regardless of the decision made to exclude this criterion, those working with gamblers should remain aware of the high rates of co-occurrence between gambling disorder and illegal activities. Recent evaluations of the new DSM-5 criteria across various treatment and community samples found that over 40% of those engaged in treatment for gambling-related problems reported engaging in illegal activates [39]. Furthermore, previous work has found that those who have experienced arrests or incarceration as a result of gambling-related crime were more likely to display features suggestive of antisocial personality disorder and substance use disorders [40]. It has also been suggested that gamblers who report illegal activities may also require more intensive treatment than those who do not [41]. Thus, the relationship between gambling and illegality should remain a clinical and research focus despite the illegal acts criterion being removed in the DSM-5.

The final change in the diagnostic criteria for the DSM-5 was the reduction of the number of criteria needs for a diagnosis. In the DSM-IV, meeting five out of the ten listed criteria was necessary in order to obtain a diagnosis of pathological gambling. In the DSM-5, this was reduced to four out of nine criteria. The rationale for this reduction was that it would ensure consistency with previous diagnosis rates

following the removal of the illegal acts criterion [35]. Empirical studies since then have shown that this change in the threshold for diagnosis resulted in either no change or in a very slight increase in the numbers of individuals meeting criteria for disordered gambling [39, 42, 43]. However, comparisons with other measures of gambling severity have led to the claim that the reduced threshold leads to more consistent diagnosis relative to the previous criteria [37]. Taken together, these findings suggest that there does appear to be sound empirical support for the changes made to the diagnostic criteria in the DSM-5.

2.5 Diagnosis of Subclinical Gambling

For many clinicians, diagnostic issues are of secondary importance: when an individual presents to a service asking for treatment for their gambling, they will receive it, and whether they meet strict diagnostic criteria is purely of academic interest. However, in some treatment settings, particularly in the United States where insurance companies often dictate that a current diagnosis is necessary for treatment coverage, ensuring that those who seek treatment would also meet some formal diagnosis can make the difference between those who are experiencing gambling-related harm receiving treatment or not. It is in this context that researchers and commentators have often proposed further changes or additions to diagnostic systems used for gambling-related behaviours that attempt to capture those who may not meet DSM-5 criteria for gambling disorder but who may nonetheless be experiencing significant distress or harm as a result of their gambling.

There have been various proposals for how to classify such 'subclinical' gamblers. One proposal has been to model the criteria for the DSM-5 on the classification system used for substance use disorders, where the endorsement of only two symptoms is required for a diagnosis [38]. Under such a system, gamblers would then be further classified into subgroups by the number of criteria met. For example, individuals endorsing two to four symptoms could be classified as having 'disordered gambling, moderate', while those meeting more than four criteria could be classified as having 'disordered gambling, severe' [38]. Another, which was proposed when developing the National Opinion Research Center DSM Screen for Gambling Problems (NODS), a commonly used population-based screening tool for gambling problems, was to classify those who meet one or two of the previous DSM-IV criteria as an 'at-risk' gambler, those who meet three or four classified as a 'problem gambler' and those who meet five or more as a 'pathological gambler' [44]. Other classification schemes refer to 'levels' of gambling, which are based on both gambling severity and willingness to seek treatment, ranging from 'level 0' representing those who have never gambled, up to 'level 4' representing those who both meet diagnostic criteria for a gambling disorder and show willingness to enter treatment [45].

These and similar suggestions of incorporating previously undiagnosed less severe categorisations of gamblers were rejected by the DSM-5 workgroup as it would result in a large increase in the rates at which gambling disorder was diagnosed [37]. However, whatever terms are eventually settled on ([46], documented

14 different classification schemes), it appears clear that there is a large group of individuals who do not meet full diagnostic criteria for gambling disorder, and yet have come to the attention of researchers and clinicians. Work with individuals in this subclinical group has shown that of the current diagnostic criteria, they are more likely to endorse the more 'cognitive'-type symptoms (i.e. lying, gambling to escape problems, preoccupation with gambling) than they are to endorse other symptom clusters (with the exception of the 'chasing losses' criteria, which almost all treatment-seeking gamblers meet [47]).

Despite the decision not to include a subclinical diagnosis in the current edition of the DSM, there is evidence that those who fall into this category may benefit from clinical attention. It has been demonstrated that adults who report symptoms of disordered gambling but do not meet full DSM criteria for gambling disorder (or its previous incarnation, pathological gambling) show increased rates of other Axis I psychiatric disorders [48], higher rates of alcohol and substance use problems [49] and higher rates of suicidal thoughts [50] than the general population. Gambling disorder symptoms are also associated with problem behaviour in adolescents [51]. Furthermore, rather than progressing in a linear fashion as had been previously assumed, longitudinal research has shown that individuals' gambling frequently moves between severity levels [52]. Taken together, these findings should serve as a reminder to anyone working in the gambling field to not narrow their focus solely on those who meet current diagnostic criteria for a gambling disorder.

2.6 A Harm-Based Classification: The Concept of 'Problem Gambling'

Given evidence that there are many individuals experiencing gambling-related harms who do not meet strict criteria for gambling disorder, it is unsurprising that in many places around the world, a different conceptualisation of difficulties related to gambling is used. Rather than focussing on behavioural symptoms, as is done with both gambling disorder and its predecessor pathological gambling, the notion of 'problem gambling' instead focusses on harm in an individual's life as a result of the gambling. The term problem gambling is generally held to refer to any pattern of gambling that is resulting in disruptions to an individual's social, occupational or psychological functioning [46]. While the precise definition of the term problem gambling can differ between jurisdictions, a commonly cited definition for problem gambling is that put forward by Ferris and Wynne [53], which defines it as 'gambling behaviour that creates negative consequences for the gambler, others in his or her social network, or for the community' (p. 58). With such a definition of problem gambling, the aforementioned difficulties with a symptom-based approach often excluding some individuals who are experiencing gambling-related harms are avoided, as the harm itself becomes the hallmark of the problem. Similar definitions have been used in public health contexts in the United Kingdom, Canada and Australia (see [46] for a brief review). An advantage of the problem gambling approach in public health contexts is that it is useful in identifying individuals with lower levels of gambling-related harms and

encouraging them to seek treatment before they may meet full diagnosis for a gambling disorder or pathological gambling [54].

However, there are also disadvantages of such an approach as well, given its focus on subjective judgements of 'harm'. Walker [55] gave the example of an individual who has with a spouse with strict religious or moral objections to gambling who buys a weekly lottery ticket. While most people would not consider this a behaviour worthy of clinical attention, it is conceivable that such an individual would be experiencing subjective harm as a result of their gambling, if it resulted in arguments with their spouse. Blaszczynski and Nower [54] further note that defining gambling based solely on subjective measures of harm runs the risk of categorising together those with minor levels of gambling-related harm with those with serious difficulties in controlling and regulating their impulses, potentially resulting in a large, heterogeneous group. To overcome such disadvantages, a compromise definition was put forward by Blaszczynski et al. [56], where problem gambling was defined as 'a chronic failure to resist gambling impulses that result in disruption or damage to several areas of a person's social, vocational, familial or financial functioning'. Such a definition includes both the sense of subjective harm, as well as the notion that the individual has a diminished or impaired ability or willingness to resist their impulses to gamble. However, the most important message of this discussion is that researchers, clinicians and policy-makers working in the area need to be aware of the advantages and disadvantages of whatever approach they take to defining gambling-related difficulties and to select that which best suits their purposes.

2.7 Gambling-Related Harm

The centrality of harm to the concept of problem gambling raises obvious questions: How do we define gambling-related harm? And what harms are commonly observed clinically in gamblers? Langham et al. [57] have recently proposed a conceptual framework to assist in answering both of these questions. Based on both a literature review and focus group research with clinical samples of gamblers, a proposed definition of gambling-related harm was given as 'any initial or exacerbated adverse consequence due to an engagement with gambling that leads to a decrement to the health or wellbeing of an individual, family unit, community or population' [57]. Langham et al. [57] then went on to identify seven domains across which gamblers may experience harm: financial, relational, emotional/psychological, health, cultural, work/study and criminal activity. For each of these domains, there is clear evidence of the potential for gambling to cause harms.

Financial harms are one of the easily identified harms as a result of problem gambling, as they are often directly related to gambling losses. They may also contribute to the harms seen in other domains, as financial losses have the potential to result in marital discord, psychological distress, neglect of healthcare, disruptions at work and criminal activity in an attempt to repay debts. For example, gamblers who have declared bankruptcy were significantly more likely to also be experiencing marital, legal, psychological and work-related disruptions [58]. Financial harms

should always be investigated by clinicians working with gamblers, given that they are one of the key motivators for gamblers seeking treatment [59] and are one of the key variables associated with gambling-related suicide [60].

The second identified area of harms caused by gambling identified by Langham et al. [57] were relational harms, which include disruptions in the relationships that gamblers have with their spouse, children or other family members or friends. These harms can be a direct result of the gambler neglecting the relationship due to time spent gambling or due to lack of trust as a result of the gambler lying about their behaviour. Several studies have found that gambling is a potential risk factor for marital discord and divorce [61, 62], domestic violence [63] and child maltreatment [64]. The recognition of such harms has led to the suggestion of providing counselling and treatment directed towards the family members of problem gamblers [65] or for treating problem gambling in the context of family issues [66].

Emotional and psychological distress was the next domain of harm identified by Langham et al. [57]. Emotional distress can result from feelings of hopelessness stemming from poorly controlled behaviour, a lack of security as a result of financial or relational disruptions or shame and stigma associated with gambling. Gambling has been correlated with psychiatric diagnoses generally [48] and with depression and other mood disorders specifically [67, 68]. The existence of stigma and shame around problem gambling should also be noted by clinicians working with problem gamblers, as it may constitute a key barrier to individuals seeking treatment for gambling-related problems [59, 69].

Decrements to health were the fourth domain identified by Langham et al. [57] as an area of potential gambling-related harm. Health problems may result from gamblers neglecting their health due to the time and money they spend gambling, from the stress they experience as a result of their gambling, from living a sedentary lifestyle as a result of time spent gambling or through having no financial resources to engage in more health-positive behaviours. Problem gambling has been associated with poorer physical health and greater numbers of reported physical health problems [70–72]. A large epidemiological survey has specifically found that pathological gambling was specifically associated with higher rates of tachycardia, angina, cirrhosis and other liver diseases, even after controlling for demographic and behavioural risk factors [73]. These findings highlight the toll that gambling may take on physical as well as emotional health.

The fifth domain identified by Langham et al. [57] was cultural harms, which related to the proposal that gambling caused disconnections between gamblers and their cultural beliefs, roles and practices. This process may include distress as a result of going against cultural norms or isolation from a cultural community as a result of gambling. While such harms are more difficult to measure due to their more diffuse conceptualisation, problem gambling has been associated with feelings of loneliness and social isolation [74], and clinicians working with problem gambling should be cognizant of how cultural factors may be impacting on a gambler's psychosocial functioning (for a review on this topic, see [75]).

Reduced performance at work or study was also identified by Langham et al. [57] as an area for potential harm caused by gambling. These harms may result from

being distracted at school, university or work as a result of gambling activities, increased absenteeism as a result of not being able to pay for transportation or not being able to pay for work or study tools. Problem gambling has been associated with poorer grades in adolescents [51] and in college students [76]. Problem gambling is also associated with poor work productivity in adults [77], as are financial losses resulting from gambling [78]. The potential for gambling to lead to problems at work should be of particular attention to clinicians working with problem gamblers, due to the importance of problem gamblers needing to maintain regular work in order to address some of their gambling-related debts.

The final domain identified by Langham et al. [57] was criminal acts. As noted in the previous discussion on the changes in the DSM criteria for pathological gambling/gambling disorder, criminal acts are often a sign of more severe gambling pathology, as they represent a desperate attempt to pay back gambling-related losses, with 40% of those engaged in treatment for gambling-related problems reporting engaging in illegal activates [39].

While the above classification of gambling harms has focussed on harms experienced by gamblers and those in close familiar or work relationships with them, Langham et al. [57] identified the potential for more community-wide harms resulting from problem gambling, in forms such as increased levels of debt and bankruptcies, reliance on government support, decreased community-wide economic productivity or increases in crime rates. They also suggested that harms related to gambling have the potential to cross generations, as children and/or grandchildren of problem gamblers may potentially be impacted in lasting ways (e.g. children of problem gamblers experiencing ongoing psychological disturbances as a result of neglect or homelessness that follows from a parent's gambling). These wider harms, while necessarily more difficult to quantify and measure, require further attention from future research.

2.8 Conclusions

Although both gambling and efforts to control it have long histories, it has only been a focus of clinical attention since the twentieth century. At present, there are several competing accounts that have been put forward to explain gambling behaviour. Given that there is no universally accepted theoretical account of gambling, it is unsurprising that there is still considerable debate over the most appropriate way to define excess gambling and its associated symptoms. Both the behavioural symptom-based DSM-5 diagnosis of 'gambling disorder' and the harm-focussed concept of 'problem gambling' have their advantages and disadvantages, and researchers, clinicians and policy-makers working in the field should be aware of these differences when selecting which conceptualisation is most appropriate to use in their work. What does not appear to be in debate is the recognition that a proportion of individuals gamble to excess, exhibit features of impaired control and suffer psychological distress, supporting the notion that gambling to excess in this subpopulation represents a clinical condition.

References

1. Scarne J. Scarne's new complete guide to gambling. London: Constable; 1961.
2. Schwartz D. Roll the bones: the history of gambling. New York: Gotham Books; 2006.
3. Steinmetz A. The gaming table: its votaries and victims in all times and countries, especially in England and France. Publication No. 96, Patterson Smith Reprint Series in Criminology Law Enforcement and Social Problems, vols I and II. NJ: Patterson Smith; 1969 (orig. 1870).
4. Wu A, Lau JT. Gambling in China: socio-historical evolution and current challenges. Addiction. 2015;110:210–6.
5. Blakey R. The development of the law of gambling 1776–1976. Washington: National Institute of Law Enforcement and Criminal Justice; 1977.
6. France CJ. The gambling impulse. Am J Psychol. 1902;13:364–76.
7. Peterson VP. Obstacles to enforcement of gambling laws. Ann Am Acad Pol Soc Sci. 1950;269:9–21.
8. Orford J. Excessive appetites: a psychological view of addictions. New York: Wiley & Sons; 1985.
9. Squires P. Fydor Dostoevsky: a psychopathographical sketch. Psychoanal Rev. 1937;24:365–88.
10. Pushkin AS. Russian short stories (trans. R.S. Townsend). London: Everyman's Library; 1982. p. 1–37.
11. Dostoevsky F. The gambler. (trans J. Coulson). Middlesex: Penguin Books; 1978. p. 17–162.
12. Thackeray WM. A gambler's death. 1840. https://ebooks.adelaide.edu.au/t/thackeray/william_makepeace/paris/contents.html. Retrieved 9 Jan 2016.
13. Saki. The stake. Champaign, IL: Project Gutenberg; 1995.
14. von Hattinger H. Analerotik, Angstlust und Eigensinn. Int Z Psychoanal. 1914;2:244–58.
15. Bergler E. The psychology of gambling. London: International Universities Press; 1957.
16. Harris H. Gambling and addiction in an adolescent male. Psychoanal Q. 1964;34:513–25.
17. Herman RD. Gamblers and gambling. New York: Harper Row; 1976.
18. Skinner BF. Science and human behaviour. New York: Free Press; 1953.
19. Pavlov IP. Lectures on conditioned reflexes (translated by W.H. Gantt). London: Allen and Unwin; 1928.
20. Anderson G, Brown RIF. Real and laboratory gambling: sensation-seeking and arousal. Br J Psychol. 1984;75:401–10.
21. Zuckerman M. Sensation seeking: beyond the optimum level of arousal. NJ: Hillsdale; 1979.
22. Jacobs DF. A general theory of addictions: a new theoretical model. J Gambl Behav. 1986;2:15–31.
23. Solomon RL, Corbit JD. An opponent-process theory of motivation: I. Temporal dynamics of affect. Psychol Rev. 1974;81:119–45.
24. Blaszczynski A, McConaghy. Anxiety and/or depression in the pathogenesis of addictive gambling. Int J Addict. 1989;24:337–50.
25. Ladouceur R, Walker M. A cognitive perspective on gambling. In: Salkovskis PM, editor. Trends in cognitive-behavioural therapies. New York: John Wiley & Sons; 1996. p. 89–120.
26. Toneatto T, Blitz-Miller T, Calderwood K, Dragonetti R, Tsanos A. Cognitive distortions in heavy gambling. J Gambl Stud. 1997;13:253–66.
27. Walker MB. The psychology of gambling. Sydney: Pergamon Press; 1992.
28. Lesieur HR. The chase: career of the compulsive gambler. MA: Schenkman; 1984.
29. American Psychiatric Association. DSM-IV: diagnostic and statistical manual of mental disorders. fourth ed. Washington, DC: American Psychiatric Association; 1994.
30. Reilly C, Smith N. The evolving definition of pathological gambling in the DSM-5. National Center for Responsible Gaming. 2013. p. 1–6.
31. World Health Organisation. ICD-9: international classification of diseases: revisions 9. Geneva, Switzerland: World Health Organisation; 1975.
32. American Psychiatric Association. DSM-III: diagnostic and statistical manual of mental disorders. third ed. Washington, DC: American Psychiatric Association; 1980.

33. Petry N. Pathological gambling: etiology, comorbidity, and treatment. Washington, DC: American Psychiatric Publishing; 2005.
34. American Psychiatric Association. DSM-5: diagnostic and statistical manual of mental disorders. fifth ed. Washington, DC: American Psychiatric Association; 2013.
35. Petry NM. Pathological gambling and the DSM-V. Int Gambl Stud. 2010;10:113–5.
36. American Psychiatric Association. DSM-IV-TR: diagnostic and statistical manual of mental disorders (text revision). fourth ed. Washington, DC: American Psychiatric Association; 2000.
37. Petry NM, Blanco C, Auriacombe M, Borges G, Bucholz K, Crowley TJ, Grant BF, Hasin DS, O'Brien C. An overview of and rationale for changes proposed for pathological gambling in DSM-5. J Gambl Stud. 2014;30:493–502.
38. Mitzner GB, Whelan JP, Meyers AW. Comments from the trenches: proposed changes to the DSM-V classification of pathological gambling. J Gambl Stud. 2011;27:517–21.
39. Petry NM, Blanco C, Stinchfield R, Volberg R. An empirical evaluation of proposed changes for gambling diagnosis in the DSM-5. Addiction. 2013;108:575–81.
40. Potenza MN, Steinberg MA, McLaughlin SD, Rounsaville BJ, O'Malley SS. Illegal behaviors in problem gambling: analysis of data from a gambling helpline. J Am Acad Psychiatry Law Online. 2000;28:389–403.
41. Grant JE, Potenza MN. Commentary: Illegal behavior and pathological gambling. J Am Acad Psychiatry Law Online. 2007;35:302–5.
42. Denis C, Fatséas M, Auriacombe M. Analyses related to the development of DSM-5 criteria for substance use related disorders: 3. An assessment of pathological gambling criteria. Drug Alcohol Depend. 2012;122:22–7.
43. Temcheff CE, Derevensky JL, Paskus TS. Pathological and disordered gambling: a comparison of DSM-IV and DSM-V criteria. Int Gambl Stud. 2011;11:213–20.
44. Hodgins DC. Using the NORC DSM Screen for Gambling Problems as an outcome measure for pathological gambling: psychometric evaluation. Addict Behav. 2004;29:1685–90.
45. Shaffer HJ, Hall MN. Estimating the prevalence of adolescent gambling disorders: a quantitative synthesis and guide toward standard gambling nomenclature. J Gambl Stud. 1996;12:193–214.
46. Abbott M, Volberg R, Bellringer M, Reith G. A review of research on aspects of problem gambling. London: Responsibility in Gambling Trust; 2004.
47. Toce-Gerstein M, Gerstein DR, Volberg RA. A hierarchy of gambling disorders in the community. Addiction. 2003;98:1661–72.
48. Bischof A, Meyer C, Bischof G, Kastirke N, John U, Rumpf HJ. Comorbid axis I-disorders among subjects with pathological, problem, or at-risk gambling recruited from the general population in Germany: results of the PAGE study. Psychiatry Res. 2013;210:1065–70.
49. Blanco C, Hasin DS, Petry N, Stinson FS, Grant BF. Sex differences in subclinical and DSM-IV pathological gambling: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Psychol Med. 2006;36:943–53.
50. Feigelman W, Gorman BS, Lesieur H. Examining the relationship between at-risk gambling and suicidality in a national representative sample of young adults. Suicide Life Threat Behav. 2006;36:396–408.
51. Potenza MN, Wareham JD, Steinberg MA, Rugle L, Cavallo DA, Krishnan-Sarin S, Desai RA. Correlates of at-risk/problem Internet gambling in adolescents. J Am Acad Child Adolesc Psychiatry. 2011;50:150–9.
52. LaPlante DA, Nelson SE, LaBrie RA, Shaffer HJ. Stability and progression of disordered gambling: lessons from longitudinal studies. Can J Psychiatry. 2008;53:52–60.
53. Ferris J, Wynne H, Single E. Measuring problem gambling in Canada. Draft final report for the Inter-Provincial Task Force on Problem Gambling. Canada: Canadian Centre for Substance Abuse; 1998.
54. Blaszczynski A, Nower L. A pathways model of problem and pathological gambling. Addiction. 2002;97:487–99.
55. Walker M. On defining pathological gambling. National Association of Gambling Studies Newsletter. 1998;10:5–6.

56. Blaszczynski AP, Steel ZP, McConaghy N. Impulsivity and pathological gambling. Addiction. 1997;92:75–87.
57. Langham E, Thorne H, Browne M, Donaldson P, Rose J, Rockloff M. Understanding gambling related harm: a proposed definition, conceptual framework, and taxonomy of harms. BMC Public Health. 2016;16:1.
58. Grant JE, Schreiber L, Odlaug BL, Kim SW. Pathological gambling and bankruptcy. Compr Psychiatry. 2010;51:115–20.
59. Evans L, Delfabbro PH. Motivators for change and barriers to help-seeking in Australian problem gamblers. J Gambl Stud. 2005;21:133–55.
60. Blaszczynski A, Farrell E. A case series of 44 completed gambling-related suicides. J Gambl Stud. 1998;14:93–109.
61. Abbott DA, Cramer SL, Sherrets SD. Pathological gambling and the family: practice implications. Fam Soc. 1995;76:213.
62. Wenzel HG, Øren A, Bakken IJ. Gambling problems in the family—a stratified probability sample study of prevalence and reported consequences. BMC Public Health. 2008;8:412.
63. Muelleman RL, DenOtter T, Wadman MC, Tran TP, Anderson J. Problem gambling in the partner of the emergency department patient as a risk factor for intimate partner violence. J Emerg Med. 2002;23:307–12.
64. Afifi TO, Brownridge DA, MacMillan H, Sareen J. The relationship of gambling to intimate partner violence and child maltreatment in a nationally representative sample. J Psychiatr Res. 2010;44:331–7.
65. Lorenz VC, Yaffee RA. Pathological gamblers and their spouses: problems in interaction. J Gambl Behav. 1989;5:113–26.
66. McComb JL, Lee BK, Sprenkle DH. Conceptualizing and treating problem gambling as a family issue. J Marital Fam Ther. 2009;35:415–31.
67. Clarke D. Impulsivity as a mediator in the relationship between depression and problem gambling. Personal Individ Differ. 2006;40:5–15.
68. Kim SW, Grant JE, Eckert ED, Faris PL, Hartman BK. Pathological gambling and mood disorders: clinical associations and treatment implications. J Affect Disord. 2006;92:109–16.
69. Hing N, Holdsworth L, Tiyce M, Breen H. Stigma and problem gambling: current knowledge and future research directions. Int Gambl Stud. 2014;14:64–81.
70. Burge AN, Pietrzak RH, Molina CA, Petry NM. Age of gambling initiation and severity of gambling and health problems among older adult problem gamblers. Psychiatr Serv. 2004;55:1437–9.
71. Desai RA, Desai MM, Potenza MN. Gambling, health and age: data from the National Epidemiologic Survey on Alcohol and Related Conditions. Psychol Addict Behav. 2007;21:431.
72. Erickson L, Molina CA, Ladd GT, Pietrzak RH, Petry NM. Problem and pathological gambling are associated with poorer mental and physical health in older adults. Int J Geriatr Psychiatry. 2005;20:754–9.
73. Morasco BJ, Pietrzak RH, Blanco C, Grant BF, Hasin D, Petry NM. Health problems and medical utilization associated with gambling disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Psychosom Med. 2006;68:976–84.
74. Trevorrow K, Moore S. The association between loneliness, social isolation and women's electronic gaming machine gambling. J Gambl Stud. 1998;14:263–84.
75. Raylu N, Oei TP. Role of culture in gambling and problem gambling. Clin Psychol Rev. 2004;23:1087–114.
76. Winters KC, Bengston P, Door D, Stinchfield R. Prevalence and risk factors of problem gambling among college students. Psychol Addict Behav. 1998;12:127.
77. Collins D, Lapsley H. The social costs and benefits of gambling: an introduction to the economic issues. J Gambl Stud. 2003;19:123–48.
78. Garman ET, Leech IE, Grable JE. The negative impact of employee poor personal financial behaviors on employers. J Financ Couns Plan. 1996;7:157.