



Behavioral addictions

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Behavioral addictions are slowly becoming recognized as a valid category of psychiatric disorder as shown by the recent allocation of pathological gambling to this category in DSM-5. However, several other types of psychiatric disorder proposed to be examples of behavioral addictions have yet to be accorded this formal acknowledgment and are dispersed across other sections of the DSM-5. This brief review marks this important point in the evolution of this concept and looks to future investigation of behavioral addictions with the theoretical frameworks currently being used successfully to investigate substance addiction and obsessive-compulsive disorder, in a potentially new spectrum of impulsive-compulsive disorders.

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Introduction

The new DSM-5 diagnostic classification has seen significant changes in thinking with respect to entire classes of disorder spanning substance abuse and obsessive-compulsive disorder (OCD) [1]. Thus, in DSM-5, the compulsive aspects of drug-taking now receive greater emphasis than the concept of dependence, and OCD has been shifted from anxiety disorders to occupy a new position as exemplary of obsessive-compulsive spectrum disorder. Formerly, part of ‘Impulse Control Disorders’, Gambling Disorder (originally ‘compulsive gambling’ in the DSM-III) is now associated with substance addictions and characterized as a prototypical example of a ‘behavioral addiction’. It is currently the only example of behavioral addiction recognized in DSM-5, despite widespread public and media interest in such behaviors as ‘internet addiction’, ‘sexual addiction’, ‘compulsive shopping’, and ‘food as an addiction’. What is meant

exactly by the term ‘behavioral addiction’? Its origins clearly have to be examined in terms of its consequences in gaining certain goals, such as pleasant sensory stimulation or information. However, in more extreme form, ‘behavioral addiction’ connotes a form of reinforcement deriving from the performance of a particular behavior itself: in plain words, behavior performed for its own sake.

The inclusion of Gambling Disorder in the ‘Substance-Related and Addictive Disorders’ section of DSM-5 is an important step that recognizes possible commonalities of these conditions. Other disorders possibly to be considered in the same light are dispersed across other sections of DSM-5, including in particular ‘Disruptive, impulse control and conduct disorders’, ‘Obsessive-compulsive and related disorders’, and ‘Feeding and eating disorders’. These sections thus include not only gambling, but also the repetitive playing of video games, use of computers and the internet for diverse purposes including pornography and gambling, excessive exercise, cutting, shopping and tanning, as well as kleptomania. The question of whether such behaviors as trichotillomania and the tics of Gilles de la Tourette syndrome also belong in the broad category of ‘impulsive-compulsive disorders’ is also germane.

Ultimately, we suspect that this issue of classification will be addressed satisfactorily by detailed neurobehavioral and neurocognitive analyses of these disorders. For example, is a behavioral addiction *motivated* in the sense of having, at least at some stage, well-defined goals? Or is the behavior elicited by particular contexts and is apparently without purpose, as appears to be the case for tics, for example? In fact, we believe that this distinction may be difficult to apply categorically and that *all* of these behaviors may fall into a superordinate category of ‘impulsive-compulsive disorders’ [2,3], albeit where each disorder is associated with behavioral outputs of variable, and sometimes considerable, complexity.

By ‘impulsive’, we mean behavior that is apparently disinhibited to the degree that it is premature and inappropriate to the context, with likely adverse consequences. Impulsive behavior is frequently risky in nature, although it is not necessarily synonymous with ‘sensation seeking’. Hypothetically, the personality trait of impulsivity is an important concept for vulnerability, initiation and relapse in impulsive-compulsive disorders. However, impulsivity itself is not a unitary construct; one distinction is between impulsive action [4] (as in Go-No Go paradigms) and impulsive choice (as in the temporal discounting of reward and delayed gratification). Another

is between 'waiting' and 'stopping', which refers to whether the inhibitory process occurs before or after response initiation, respectively [5]. Impaired 'stopping' may also be a component of compulsive behavior, which is defined as the continued repetition of behavior despite adverse consequences. Compulsive behavior clearly arises in different contexts and may involve distinct behavioral mechanisms. For example, OCD is commonly conceived as a harm-avoidant behavior maintained by *negative* reinforcement [6]. In contrast, compulsive drug taking is often hypothesized to arise from an aberrant *positive* reinforcement system [7]. However, this distinction may be less clear-cut than first appears to be the case; the influential opponent motivational hypothesis [8] accounts for drug-seeking and taking in terms of negative reinforcement obtained from postponement or escape from aversive withdrawal symptoms (whether physiological or subjective). Moreover, some forms of obsessive-compulsive behavior (including hoarding for example) may not involve negative reinforcement at all. Within pathological gamblers, similar heterogeneity is observed in motivations to gamble, with dimensions representing gambling for enhancement (i.e. positive reinforcement, linked to excitement and physiological arousal) and gambling for coping (i.e. negative reinforcement, linked to relief of anxiety, low mood and boredom) [9].

The criterion of adverse consequences is clearly important for defining compulsive behavior, but requires careful consideration. Many substances of abuse have neurotoxic effects which may impair cognitive functioning and also lead to ill-health. Some compulsive behaviors (e.g. kleptomania) are obviously a hazard to society and represent criminal activity. However, a mere disproportionate allocation of time to a specific activity at the expense of others can also be construed as an adverse consequence, if for example, a compulsive behavior infringes on performance in the workplace or in family relationships. 'Behavioral addictions' are thus of considerable theoretical interest because their outcomes do not, like drugs, lead to direct neurotoxic effects and only rarely directly to ill-health, but do in the case of problem gambling lead to the clear adverse consequence of debt.

One question to be posed from the outset is epidemiological in nature. How reliable is the evidence for occurrence of disorders that could be categorized as 'behavioral addictions' with disastrous consequences for the individual? A model study in this respect investigated pathological internet use (PIU) among 11 356 adolescents in 11 European countries [10^{••}]. The incidence of PIU was 4.4%, and was higher in males, who had significantly higher rates of online solo gaming than females, who preferred social networking. Overall, PIU was associated with reduced parenting and parental unemployment. PIU has been associated with both psychological (i.e. depression, anxiety, ADHD as well as obsessive symptoms) and physical problems, although the

issue of causality is still to be resolved [11]. Analogous surveys will be required to quantify the burdens associated with other 'behavioral addictions'. This brief review considers how this new category of behavioral addictions might be investigated in light of what we have gleaned from the study of drug addiction, OCD [12], and more recently, pathological gambling.

Substance abuse as the prototypical addiction

Drug or substance addiction is a prototypical disorder of the neural 'reward system'. Drugs have been suggested to 'hijack' this system and induce potent forms of reinforcement learning, resulting in aberrant and escalating sequences of drug-seeking and drug-taking behavior [13]. Within this framework, the theory of instrumental behavior and its division into action–outcome and stimulus–response habit learning has been proposed to contribute to the development of compulsive drug seeking behavior, beginning from its hypothesized origins, in the case of stimulant drug abuse, for example, in individual differences in impulsive behavior [14]. Hypothesized transitions from action–outcome (i.e. goal-directed) behaviors to habitual responding in addiction have been related to the cascading loop circuitry of the basal ganglia [15]. An alternative, but possibly related perspective, more relevant perhaps to opiate and alcohol addiction, is the opponent motivational hypothesis, which also invokes such physical and subjective phenomena as craving, tolerance and withdrawal [8]. These hypotheses, based on reinforcement theory and derived largely from a large body of studies in experimental animals in model systems, are gradually being applied to human drug abusers, with a resultant profusion of illuminating parallels. The burgeoning analysis of compulsive gambling is taking much the same course [16,17,18[•],19^{••}]. We suggest that the consideration of future behavioral addictions should be most informative if conducted within a similar theoretical framework.

Gambling as the prototypical behavioral addiction

Impulsivity is a core feature of pathological gambling. Recent neurocognitive studies in pathological gambling have explored the facets of impulsivity with respect to the dichotomy between impulsive choice and impulsive action. There are numerous illustrations of impulsive choice in pathological gamblers: for example, community-recruited problem gamblers showed a similar increase in risk-taking on the Cambridge Gamble Task (CGT) to patients with alcohol dependence who did not gamble regularly [20]. Elevated risk-taking on the CGT was recently seen to covary with steeper delay discounting tendencies and higher scores on a mood-related impulsivity ('urgency') questionnaire [21]. These studies converge with work taking a behavioral economics perspective to gambling. Pathological gamblers showed

impairments on an array of decision-making tasks assessing both choice under uncertainty (i.e. where the probabilities can only be estimated through sampling from the task) and under explicit risk, where the probabilities were explicitly stated [22]. A further study [23] modelled the probability weighting function from Prospect Theory in pathological gamblers, showing a global over-estimation of probabilities across the full range (from 0 to 1).

Impulsive choice in pathological gamblers is also subject to Pavlovian influences from gambling-related cues. A recent study has examined these influences by presenting gambling-associated images in the background during functional neuroimaging of delay discounting decisions [24^{*}]. The cues were subsequently categorized on the basis of subjective craving. The high-craving cues were associated with a greater preference for the impulsive, immediate rewards, and on these trials the value coding in midbrain and striatum was reversed compared to the low craving condition. It is well-recognized that the irrational cognitions that are evident in problem gamblers seem to be enhanced during games themselves ('the action') [25], which may either be mediated by gambling Pavlovian cues or the physiological arousal that gambling induces. The insula is emerging as playing a specific role in these gambling distortions: a recent neuropsychological study found that patients with brain injury affecting the insula were insensitive to the effects of gambling 'near-misses' on a slot machine task, and did not display the gambler's fallacy in sequential roulette predictions, compared to various comparison groups including patients with vmPFC and amygdala damage [26^{**}]. The increased levels of these cognitions in pathological gamblers — itself linked to steep delay discounting [27] — may therefore be underpinned by excessive recruitment of the insula.

Studies of impulsive action in pathological gambling have been less consistent. We detected no impairments on the stop signal task in community-recruited problem gamblers [28] and a second study saw that any deficit depended upon co-morbid ADHD [29]. One possible interpretation is that deficits in response inhibition are only present in a subset of pathological gamblers; for example Blaszczynski and Nower's [30] influential 'Pathways Model' points to a subgroup of 'antisocial impulsivists' with ADHD comorbidity and neuropsychological sequelae of a dysexecutive syndrome. Two studies that directly compared measures of impulsive choice and impulsive action found that impulsive action (on the stop signal task) was only disrupted by the most severe level of pathological gambling, whereas impulsive choice was incrementally sensitive to subclinical problematic status as well as full-blown diagnosis status [19,31].

Trait impulsivity and impulsive choice on the Iowa Gambling Task also predict treatment dropout in pathological gamblers, whereas impulsive action (Stroop test)

and executive inhibitory tasks including the Wisconsin Card Sort Test did not [32]. One laboratory has included a valuable comparison with cocaine dependence, with a view to dissociating vulnerability markers from neurotoxic consequences. Impulsive choice on a delay discounting task was in fact stronger in the PGs than the cocaine group, and differences in Stroop inhibition and trait urgency were present in both groups. Importantly, the cocaine users showed an additional disruption of working memory, which correlated with measures of cocaine exposure, consistent with a neurotoxic effect [33]. Impulsivity as an early risk marker cutting across the addictions is supported by major prospective studies dating back as far as early childhood [34].

Impulsivity in pathological gamblers has long been suspected to have a dopaminergic basis, given the link with dopamine agonist medications in the context of Parkinson's Disease [35,36]. Recent work has begun to examine this hypothesis. One study used H₂O¹⁵ PET study to examine the effects of the D2/3 agonist pramipexole in PD patients [37^{*}]: pramipexole increased impulsivity on a delay discounting task, with the change in *k* scores correlating with drug-induced activation of medial PFC and posterior cingulate. Strikingly, there were no dopaminergic effects on a Go-No go task of impulsive action administered in the same session. Other studies corroborate that delay discounting is particularly sensitive to the presence of behavioral addictions in patients with Parkinson's Disease [35,38,39] than markers of inhibitory control [40], with a recent computational analysis proposing a core alteration in a parameter that represents the value of uncertain future rewards [41]. Perhaps surprisingly, there is less work examining dopamine transmission in pathological gamblers without Parkinson's Disease, although a notable recent study used the [11C]PHNO dopamine ligand, in combination with amphetamine challenge, to show that dopamine release in the dorsal striatum was increased in pathological gamblers compared to healthy controls [42^{**}]. Dopamine release is reported to be attenuated in stimulant addicts, and as such the PET work in pathological gambling is beginning to highlight some provocative differences with substance abuse [43].

How well is the construct of compulsivity itself reflected in pathological gambling? Some evidence exists for slower contingency learning [44] as well as perseveration [19,45] in pathological gamblers, which have also been associated with treatment outcomes [46]. Loss chasing, a prototypical instance of compulsivity, is sometimes considered to be the hallmark of the problem gambler. These tendencies have only recently begun to be explored at a neurobiological level; loss-chasing in healthy volunteers has shown sensitivity to serotonergic and dopaminergic manipulations [47]. Influential models of drug addiction have also generated a strong focus on the dorsal striatum underlying the transition from recreational (goal-directed) to compulsive

usage. There are limited data on dorsal striatal involvement in PG, although one study using card guessing task found hyperactivity in the dorsal striatum of pathological gamblers during the anticipation of gain outcomes, and as a function of gain-related expected value [48^{*}]. Future work may fruitfully focus on separating goal-directed and habitual processes in pathological gamblers.

Other behavioral addictions

Impulsivity is undoubtedly associated with other behavioral addictions. A recent review compared findings for kleptomania, compulsive buying/shopping and internet addiction, as well as pathological gambling [49^{**}]. All three of the former disorders were found to exhibit deficits in inhibitory control as measured by the stop-signal reaction time task. Studies with impulsive choice paradigms have been less frequent, although they might be expected to show even greater deficits greater given the findings for pathological gambling reviewed above. In a recent study, Voon [50] has shown in parallel studies of obese patients, compulsive binge eating as well as substance use and OCD, that impulsive choice deficits tend to be the most reliable occurring across the impulsive-compulsive spectrum as a whole. A functional neuroimaging study demonstrated increased ventral striatal activity in the Monetary Incentive Delay task in a sample of obese individuals who did not exhibit binge eating, whereas a subgroup of obese people with binge eating disorder exhibited reductions relative to the obese group [51] suggesting that binge eating may be a distinct behavioral disorder compared to obesity. To what extent impulsivity is a predisposing trait, as appears to be the case for stimulant abuse [52], remains unclear, although it seems unlikely that deficits in SSRT result from behavioral addictions. Compulsivity has been far less investigated for behavioral addictions other than gambling which thus remains a top priority for future research, especially as this construct is perhaps closest to the notion of addiction itself, as well as treatment outcome.

The DSM-5 describes internet gaming disorder as the condition most likely candidate next to join pathological gambling in the category of behavioral addictions, although there were insufficient research data available at the time of publication to warrant its inclusion. Many publications on this syndrome to date have used the broader term 'internet addiction', which could also include excessive engagement in online gambling, social media, or pornography. Given that the internet is merely the portal by which these stimuli are accessed, we would support their clinical differentiation. Particular risks are associated with the 'Massively Multiplayer Online Role Player Games' (MMORPGs) like World of Warcraft, and an international consensus group has recently established a 9-item assessment instrument for the gaming syndrome [53].

The evidence base for cognitive and neurobiological data on internet gaming disorder is growing rapidly. As for

pathological gambling, pathological video gaming is closely tied to high trait impulsivity [54] and cognitive impairments on tasks of both impulsive choice and impulsive action [50,55,56]. Imaging studies of inhibitory tasks point to impaired recruitment of frontal cortical regions in particular [57], and a multi-centre European study in 154 adolescents found that regular video gaming involvement was associated with increased striatal grey matter volumes and faster decision-making latencies on a risk-taking task [58^{*}]. Some changes in dopamine transmission have been reported [59] and a seminal PET study described striatal dopamine release in healthy volunteers during video game play [60].

Future prospects

Over the last period comparisons of the behavioral addictions with drug addiction have enriched and strengthened the field of enquiry for impulsive-compulsive disorders more generally. This is exemplified by the development of animal models of behavioral addictions, given that models of drug addiction have arguably have been among the most useful within neuropsychiatry. Thus, several groups have begun to develop paradigms suitable for measuring gambling-like behavior in rodents, beginning with the classic Iowa Gambling Task [61,62,63^{*}]. Additionally, novel paradigms have been produced to measure compulsive eating behavior in rodents, relevant to the current controversy concerning 'food addiction' [64]. These paradigms have been used to show how compulsive eating may be exacerbated by stress and alleviated by specific drugs [65–67]. Such developments herald a new phase of research in this field that will more readily make contact with basic neuroscience. Another important focus will be on treatment (see [68^{*}]), as an increasingly dimensional analysis of impulsive-compulsive syndromes may identify medications that can be used for different expressions of what may, in fact, be common disorders resulting from dysfunction of specific neural circuitries. Possible 're-purposing' of such agents as naltrexone, SSRIs and atomoxetine may provide an initial impetus for this enterprise. Finally, a focus on individual differences and endophenotypes amongst this diverse and fascinating group of disorders may inform genetic approaches to understanding their aetiology.

Conflict of interest statement

TWR consults for Cambridge Cognition, E. Lilly, Lundbeck, Chempartners, Teva, Shire Pharmaceuticals. He receives royalties from Cambridge Cognition for CAN-TAB. He has recently held research grants from Lilly, Lundbeck and GSK. He receives editorial royalties from Springer-Verlag and Elsevier.

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