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Review

Applying incentive sensitization models to behavioral addiction

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ABSTRACT

The incentive sensitization theory is a promising model for understanding the mechanisms underlying drug addiction, and has received support in animal and human studies. So far the theory has not been applied to the case of behavioral addictions like Gambling Disorder, despite sharing clinical symptoms and underlying neurobiology. We examine the relevance of this theory for Gambling Disorder and point to predictions for future studies. The theory promises a significant contribution to the understanding of behavioral addiction and opens new avenues for treatment.

Contents

1. Introduction

Gambling Disorder[1] (GD) was recently classified as a *behavioral addiction* (American Psychiatric Association, 2013), due to overlap with drug addiction regarding symptoms and underlying neurobiology. Accordingly, it is pertinent to take a look at some of the influential theories that have formed our understanding of drug addiction. Incentive sensitization (Robinson et al., 2013; Robinson and Berridge, 1993) is one such theory. Here we examine the relevance of this theory for GD by reviewing the relevant studies and outlining its predictions for future studies. Last, we discuss implications for treatment. By stressing the role of unconscious craving the theory points to new treatment strategies such as mindfulness-based interventions that aim at enhancing awareness of bodily and emotional signals.

2. The incentive sensitization theory

Most people have experimented with recreational drugs at some point in their life (if we include alcohol), and for the vast majority this does not raise serious concerns. But for a small group of people the casual use leads to compulsive patterns of abuse with detrimental consequences. The incentive sensitization theory offers a promising explanation of how drug-induced alterations in psychological functioning can cause a transition to addiction, and pose a major risk for relapse.

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[1] In the recently released DSM-V the term 'Pathological Gambling' has been replaced by 'Gambling Disorder' (American Psychiatric Association, 2013).

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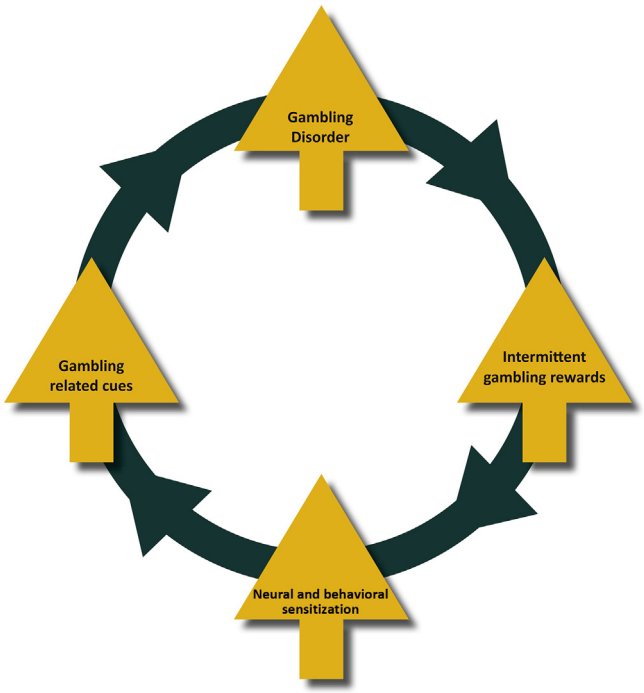

Fig. 1. Vicious circle of incentive sensitization and enhanced addiction. The incentive salience theory can be illustrated as a vicious circle where drug abuse/addiction sensitizes neural systems underlying incentive salience, leading to over-attribution of incentive salience to drugs and drug-related cues (and a blunted response to other types of rewards, e.g. social, sensory and sexual). As the addiction progresses, the cues associated with drug-intake obtain progressively greater efficacy, and at the same time, the number of cues associated with drug-intake increases and may later trigger strong 'wanting' – all of which increases severity of the addiction and makes it increasingly difficult to break the circle. As illustrated in the figure we hypothesize that the same mechanisms apply to behavioral addiction, such as Gambling Disorder. ▲ illustrates a progression or increase over time e.g. in severity of the addiction.

According to this theory, strong craving for drugs is governed by a sensitized neural system that normally functions to attribute incentive salience to reward cues. This system consists (primarily) of mesocorticolimbic dopaminergic neurons that connect the ventral tegmental area with the nucleus accumbens, neostriatum, amygdala, ventral pallidum and prefrontal cortex (Robinson et al., 2013). It "transforms ordinary stimuli, such as cues associated with rewards, into incentive stimuli, making them motivationally attractive and able to trigger an urge to pursue and consume their reward" (Robinson et al., 2013, p. 392). This is highly adaptive under normal circumstances by promoting behavior to obtain fundamental rewards (such as food and sex), ensuring survival and procreation (Kringelbach and Berridge, 2009). However, the system can be *sensitized* by drugs of abuse, if the drug is taken repeatedly and at high doses, and especially if the individual is predisposed to be susceptible to sensitization. Once sensitized, the system reacts more powerfully to the drug and drug-related cues by eliciting progressively greater neural or psychological response of *incentive salience* attribution. Hence, when the addict encounters these cues the urge to take the drug is strongly amplified, leading to increased automatic control of behavior by drug-related incentive stimuli (Robinson et al., 2013). With time, this can create a vicious circle where drug abuse and sensitization of incentive salience systems enhance each other, making it increasingly difficult to break the cycle (see Fig. 1).

Importantly, the ability of a cue to trigger a momentary desire to consume the reward ('wanting') is independent of the rewarding effects ('liking'). Although, 'wanting' and 'liking' of the drug are typically strongly linked in the initial phases of drug use, only 'wanting' becomes sensitized and consequently increases as the addiction develops (Robinson et al., 2013). Accordingly, "Craving is defined as pathologically intense feelings of wanting, which can be produced when incentive salience (or core 'wanting') is translated into conscious awareness" (Robinson et al., 2013, p. 392). Importantly, the over-attribution of incentive salience to drug-related stimuli can occur without conscious feelings of wanting, thereby directing the addict's behavior even when he/she is not aware of it (please note that *wanting* refers to the subjectively experienced feeling of desire while '*wanting*' refers to the core reactions that can happen without conscious awareness, likewise with *liking* and '*liking*'). The role of unconscious 'wanting' may have implications for treatment, because traditional cognitive behavioral therapy (CBT) does not directly target unconscious urges. It can also help explain the impaired insight that characterize addicted individuals, including lack of insight into what motivates them and severity of their disorder (Goldstein et al., 2009).

There is convincing evidence to support the model in drug addiction (see Boxes 1 and 2). Although most studies have been conducted in animals there is increasing interest in applying the model to human drug addiction (for reviews see Leyton and Vezina, 2013; Vezina and Leyton, 2009).

It is important to stress that the incentive sensitization theory is compatible with theories of deficient self-control and self-awareness, with associated dysfunctions in cortical regions such as prefrontal-, anterior cingulate and insular cortices (Changeux and Lou, 2011; Goldstein et al., 2009; Noel et al., 2013). *Excessive craving* and *deficient self-control* may even be seen as two sides of the same coin and improved self-control is expected to decrease control of behavior by drug-related incentive stimuli. As suggested by Noel and colleagues the mechanisms highlighted by the incentive sensitization theory may represent one of three systems that can lead to deficient 'willpower' in addiction (Noel et al., 2013). Furthermore, evidence supports an important role of automatic stimulus-response habits in the transition from casual to compulsive drug consumption (Everitt and Robbins, 2005). Although we acknowledge these contributions our focus here is on the role of incentive sensitization in addiction.

3. Gambling Disorder

GD is a disabling disorder characterized by repeated maladaptive gambling behaviors that persist despite negative consequences and impaired social functioning. Until very recently GD was classified as an impulse control disorder (American Psychiatric Association, 1994). However, there has been growing agreement to view GD more specifically as a behavioral addiction, similar to drug addiction (e.g. Frascella et al., 2010; Grant et al., 2010), which has now been implemented in the DSM-V (American Psychiatric Association, 2013).

The main argument for classifying GD as a behavioral addiction is the extensive overlap in clinical symptoms and underlying neurobiology between GD and drug addiction. Like drug addicts, problem gamblers struggle with symptoms such as craving, tolerance and withdrawal. They often gamble when feeling distressed and experience repeated unsuccessful attempts to cut back or stop. On a neural level, increasing evidence supports shared impairments in the mesolimbic reward circuitry (Frascella et al., 2010; Grant et al., 2010; Potenza, 2008). For example, evidence suggests that similar changes in brain activity underlie gambling urges and cocaine cravings (Potenza, 2008). Further, both types of addiction are associated with impairments in decision-making processes (in particular in self-control) and parallel changes in the functional anatomy of prefrontal-, anterior cingulate- and insular cortices (Bechara, 2005;