Learning to lose control: A process-based account of behavioral addiction

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Abstract

Learning psycho(bio)logy has developed a solid corpus of evidence and theory regarding behavior control modes. The present article briefly reviews that literature and its influence on recent models in which the transition from goal-directed to compulsive behavior is identified as the main process underlying substance use disorders.

This literature is also relevant to non-substance addictive disorders, and serves as basis to propose a restricted definition of behavioral addiction relying on the presence of behavior-specific compulsivity. Complementarily, we consider whether some activities can become disordered while remaining mostly goal-driven. Based on reinforcement learning models, relative outcome utility computation is proposed as an alternative mechanism through which dysfunctional behaviors (even not qualifying as *addictive*) can override adaptive ones, causing functional impairment.

Beyond issues of conceptual delimitation, recommendations are made regarding the importance of identifying individual etiological pathways to dysregulated behavior, the necessity of accurately profiling at-risk individuals, and the potential hazards of symptom-based diagnosis. In our view, the validity of these recommendations does not depend on the position one takes in the nosological debate.

Keywords: Behavioral addiction; Non-substance addictive disorders; Behavioral control modes; Learning; Compulsivity; Reinforcement learning

1. Background

Recent debate on behavioral addictions has been dominated by discussions as to whether dysregulated behavioral patterns such as excessive video gaming, social networking, pornography use, or exercising, should be categorized as mental disorders (Aarseth et al., 2017; Billieux et al., 2017; Dullur and Starcevic, 2018; King et al., 2018; Stein et al., 2018) and, more specifically, as addictive disorders (Billieux et al., 2015c; Blaszczynski, 2015; Griffiths et al., 2016; Mihordin, 2012; Saunders et al., 2017).

In the most recent edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), a new category was created for behavioral addictions, within the section *Substance-related and Addictive Disorders*. So far, this category only includes gambling disorder, as it was decided that internet gaming disorder should be rather included in Section III (*Emerging Measures and Models*). More recently, gaming disorder has been included in the International Classification of Diseases (ICD-11, World Health Organization, 2019), and defined as a pattern of (video) game use "characterized by impaired control […], increasing priority given to gaming over other activities […], and continuation or escalation of gaming despite the occurrence of negative consequences". Furthermore, to be considered as a disorder, excessive gaming must be of sufficient severity to result in significant functional impairment (Billieux et al., 2017).

The reclassification of gambling disorder as an addictive disorder has been supported by clinical, experimental, and neurobiological evidence (e.g., Fauth-Bühler et al., 2017), although discrepancies in brain findings between gambling and substance-use disorders exist (Clark et al., 2019). Conversely, the concept of (video) gaming disorder has been criticized by some scholars (Aarseth et al., 2017; van Rooij et al., 2018) on the basis of its allegedly excessive emphasis on symptoms (and their correspondence with those of other addictive disorders), and neglecting the psychological processes underlying the problematic use of video games.

This dispute about the conceptualization and nosology of addiction-related disorders has mostly overlooked etiological mechanisms. Although inclusion of a candidate disorder in DSM and ICD classifications has important practical consequences, its complete characterization has never been presumed to end there. Crucially, a thorough characterization would require the ability to account for clinical manifestations as arising from specific mechanisms. As we will discuss here, the failure to describe etiology, and employing a definition based on the similarity of observable manifestations can limit the potential benefits of prevention and treatment (Billieux et al., 2015c; Dudley et al., 2011; Kinderman and Tai, 2007; Stein et al., 2010). To overcome this problem, we propose a process-based conceptualization of behavioral addictions grounded in two neurocognitive mechanisms: *domain-specific compulsivity* and *computation of relative outcome utility*. The practical (e.g., clinical) implications of this conceptualization will be discussed.

2. From symptoms to learning mechanisms

Research on behavioral addictions remains directed at elaborating tools for the assessment and diagnosis of new addictions. These tools (and, eventually, the very conceptualization of the putative addictive disorders they are intended to assess) are usually based on the reclaiming of symptoms from substance-use and gambling disorders (e.g., Andreassen et al., 2012; Meerkerk et al., 2009). Validity is thus established by analogy and, subsequently, these measures are often incorporated into more complex structural models involving side constructs and clinically relevant features of behavior. Paradoxically, this research agenda could with time become detrimental for theoretical and practical development in the field (Billieux et al., 2015c).

Underlying this approach are the assumptions that behavioral addictions and substance addiction "should be defined by their similarities not their dissimilarities" (Griffiths, 2017; p. 1718), and that substance use and behavioral addictions involve the alteration of similar brain areas (i.e., the dorsolateral, orbital, and ventromedial parts of the prefrontal

cortex, and the mesocorticolimbic system; see Brand et al., 2014; Long et al., 2018). However, this 'shared features and structures' approach is vulnerable to a confirmatory bias, as contradictory findings are likely to be overlooked (Billieux et al., 2015c; Clark et al., 2019; He et al., 2017; Kardefelt-Winther, 2017a; Turel et al., 2014). Furthermore, the common involvement of similar brain structures cannot be considered a proof that they are playing the same role, or in case they are, that such role is causal (Passingham and Rowe, 2016).

Although undoubtedly valuable, etiological theorization remains underrepresented (Bickel et al., 2011; Engel and Cáceda, 2015; Kardefelt-Winther, 2017b; Kuss and Billieux, 2017). Still, the attempts to identify causal mechanisms for behavioral addictions may provide some clues on where to look, if we want to overcome the symptom recycling approach and its potential caveats. In line with an initial proposal by James and Tunney, (2017) this review places an emphasis on learning mechanisms.

Research on the learning mechanisms underlying behavioral control and decision making has the potential to provide crucial evidence for understanding addictive disorders (Bickel et al., 2018; Lewis, 2018; O'Doherty et al., 2017). According to developments in related fields (i.e., learning and decision-making psychology, behavioral and cognitive neuroscience), two control modes are responsible for two broad types of behavior: *goal-driven* and *compulsive*. Terminological alternatives for these constructs and their nuances are discussed later.

From a biobehavioral perspective, addictive substances or activities are hypothesized to exploit Pavlovian and instrumental learning mechanisms in such a way that the choices underlying these activities gradually shift from being goal-driven to being compulsive (Ersche et al., 2016; Everitt and Robbins, 2016, 2005; Holton and Berridge, 2013). The cognitive mechanisms and neuroadaptations underlying such a transition (and whether a single mechanism can account for compulsivity across different addictive disorders, experimental protocols, and species) are now the focus of an intense debate (Hogarth et al., 2019; Singer et

al., 2018). Unfortunately, with the exception of gambling disorder (Clark, 2010; van Timmeren et al., 2018), clinical research on behavioral addictions has, to date, remained oblivious to advances in the understanding of the learning mechanisms underlying behavior control.

Importantly, compulsivity as used here applies to particular behaviors, i.e., is domain specific. In other words, it is not the individual who becomes compulsive, only certain behaviors do, as a consequence of their specific history of interactions with reinforcers in the environment. Malfunctioning or weakened domain-general processes (prepotent response inhibition, working memory, cognitive flexibility, planning, resistance to interference and perseverance in demanding tasks) can facilitate such a transition, but are neither necessary nor sufficient for it. Indeed, there is considerable scientific agreement that signs of alterations in these processes are markers of addiction, but, simultaneously, a proportion of patients suffering from addiction show no domain-general alterations of goal-directed behavior, and present intact executive functions (Grant and Chamberlain, 2014; van Holst et al., 2010; Yücel et al., 2019). In patients with a severe substance use disorder, executive dysfunction is more prevalent than at the population level, but remains far from being generalized (Billieux et al., 2012; Fernández-Serrano et al., 2010; Verdejo-García et al., 2007; Yan et al., 2014). Besides, control and inhibition failure is present in other psychopathologies (Iacono, 2014; Shanmugan et al., 2016). Hence, domain-general alterations of executive functions that make individuals compulsivity-prone should be seen as a vulnerability factor contributing to altered content-specific Pavlovian and instrumental learning, and/or as a consequence of chronic addiction-related neuroadaptations.

3. Learning to lose control

The addictive process can be defined as a transition between behavioral control modes. Various models have been proposed to describe the differences between control modes, and how this transition occurs (e.g., Berridge and Robinson, 2016; Everitt and Robbins, 2016,

2005). The main differences between these models are discussed below. Still, similarities are no less important, and provide common ground for the arguments that follow.

A first approach distinguishes between goal-directed and habitual behaviors (see Dolan and Dayan, 2013, for a review of the several variations of this dichotomy). In the goal-directed mode, individuals purposefully use their knowledge of the contingencies between actions and consequences. The seminal evidence showing that the cognitive representation of a goal can drive a specific behavior came from reward devaluation procedures: if, after instrumental learning, the goal is devalued, the individual stops pursuing it (or pursues it with less intensity) from the first trial in which she is given the chance to perform the instrumental behavior again (Balleine and Dickinson, 1998). Numerous studies have shown that the ventromedial prefrontal cortex (vmPFC) is an important hub in the network representing the utility of goals[1] (Valentin et al., 2007) while the ventral striatum plays a key role in maintaining reward-based learning flexibility (Voon et al., 2015). Moreover, evidence suggests that the subjective value of different rewards is represented in the form of a common currency in a subregion of the vmPFC/orbitofrontal cortex (Levy and Glimcher, 2012; McNamee et al., 2013), allowing the decision-maker to weigh them with the same scale and decide which one to pursue.

In the habit-driven mode, instrumental behavior is no longer controlled by goals and their subjective values; namely, it becomes habitual, i.e., stimulus-triggered and stereotyped. This is a process that has been observed to concur with a transition from prefrontal to striatal control over behavior as well as a progression from the ventral to the dorsal striatum. (Everitt and Robbins, 2005, 2016). In animals, transition from goal-directed to habitual behavior requires extensive instrumental training of that specific behavior (Dickinson et al., 1983; Dickinson and Balleine, 2002; Tricomi et al., 2009). In humans, however, recent research has

[1] Here and in the remainder of this article, utility is not used with the standard meaning in Economics and formal Choice Theory, but refers to the mental representation of the combined probabilities and values of the anticipated consequences of a given course of action (as in the Prospect Theory and related models; [Fox and Poldrack, 2009]).

shown the difficulties in experimentally generating habits by mere massive repetition (de Wit et al., 2018; Watson and de Wit, 2018). Moreover, related research has also revealed that habit formation could be insufficient to fully account for addictive behavior. First, addiction symptoms in animals also develop in self-administration paradigms whereby complex and novel instrumental behaviors are required to obtain the drug (Singer et al., 2018). Second, goal-directed control can be reinstated or habit formation can be thwarted by modifying the original "one-response one-reward" paradigm so that the experimental situation becomes more similar to drug accessibility in real life (Hogarth et al., 2019). In view of this evidence, our position here is that compulsivity should not be equated with habit formation.

In the best-established alternative to the habit formation hypothesis of behavior control in addiction (at least among the learning-based proposals, Holton and Berridge, 2013), the representation of the hedonic value of rewards relies on a 'liking' system (Berridge and Robinson, 2016; Robinson et al., 2015; Robinson and Berridge, 2001) distributed across neocortical and limbic areas labelled as *hedonic hotspots* (Berridge and Kringelbach, 2015). However, drugs of abuse have the capacity to mobilize plasticity in a second system (the 'wanting' system). The learning process by means of which 'wanting' ends up overriding 'liking' as the main motivation to use the drug is called *incentive sensitization*, and the signals responsible for this progressive transition are hypothesized to originate in the mesolimbic dopamine pathway (Wyvell and Berridge 2001, 2000). Note, however, that mesolimbic dopaminergic prediction-error signals have also been implicated in other functions (see Schultz, 2016, for a review), and their role in cue cached values have been recently challenged (see Gardner et al., 2018; Sharpe et al. 2017). A complementary debate concerns whether dopaminergic signals play a role in the learning component of incentive accrual (i.e. *incentive sensitization*; Flagel et al., 2011), or in the very power of incentive cues to elicit approach

behaviors (i.e., in the expression of cue-induced behavior, or *incentive salience*, Saunders and Robinson, 2012)[2].

Behaviorally, incentive sensitization has been proposed to lead to (1) an attentional bias towards the cues that indicate the proximity of reward, (2) automatic approach tendencies to those cues, and (3) an intense affective state characterized by an overwhelming desire for the reward that grows progressively detached from its hedonic value (Berridge and Robinson, 2016). These three effects are the components of a multifaceted *craving* response. The approach component of craving (investigated in animal models using sign tracking paradigms; Anselme, 2016; Srey et al., 2015) should not be equated with habit, as defined earlier, but shares with it the disconnection of behavior from goals, since interacting with signaling cues carries no instrumental utility.

To recap, according to the habit formation hypothesis, drug use becomes compulsive as it transitions from being goal-driven to habitual. According to the incentive sensitization hypothesis, however, what becomes compulsive is not drug use or drug seeking, but craving, and this, in turn, increasingly motivates drug use. In spite of their differences, it is noteworthy that the two theories are similar in hypothesizing that (1) people who suffer from an addictive disorder feel increasingly impelled to seek and use the drug, although they are aware that these acts are not in line with their overall goals, and (2) environmental cues associated with the drug through repeated pairing play a growing role in triggering drug seeking and consumption.

This twofold conceptualization of compulsivity is fully compatible with a recent Delphi review (Yücel et al., 2019), in which a panel of experts concluded that compulsivity is the main driving force of addiction chronicity. According to it, habits can be defined as "sequential,

[2] In the words of Schultz, "*the evidence for a strictly differential dopamine role in approach behaviour versus learning is at best inconclusive. The incentive-salience hypothesis and the prediction-error account are difficult to compare and might not be mutually exclusive: incentive salience concerns dopamine's influences on behaviour, whereas prediction-error coding concerns the properties of the dopamine prediction-error signal itself, which can have many functions."* (2016, p. 192)

repetitive, motor or cognitive behaviors elicited by external or internal triggers that, once initiated, can go to completion without constant conscious oversight" (p. 8), whereas compulsivity "can also be repetitive, or automatic behavior", but is also "associated with negative outcome expectancy that contributes to the experience of being 'forced' or 'compelled' to act despite negative consequences" (p. 9). Crucially, in the context of addictive disorders, this contraposition of compulsivity and declarative goals introduces an element of irrationality, as affected people often invest significant amounts of time and resources in both the addictive behavior and efforts to cease it (Camerer, 2006; Luigjes et al., 2019; Reith, 2007).

Some contemporary models of reinforcement learning (RL) subsume different notions of compulsivity into *model-free* control, whereas goal-directed behavior is further developed as *model-based* control (O'Doherty et al., 2017; Otto et al., 2015). Model-based control relies on a learned internal model of the world to make decisions, and is computationally taxing, but allows for behavioral flexibility in rapidly changing or novel environments. Model-free control, on the contrary, relies on stored 'summary' values of reinforcement history of each available option, so that decisions are based on past history instead of anticipated consequences. In these models, such summary values are stored as associative strengths and are automatically activated by the choice options or the cues present at the moment of making a decision (Gläscher et al., 2010; Lucantonio et al., 2014). Crucially, not only habit formation, but also incentive sensitization and aberrant motivational valuation of drug rewards can result from model-free learning mechanisms (Huys et al., 2014, 2016)

In summary, irrespective of whether the main mechanism of compulsivity is described as the development of a habit or incentive sensitization (or, more generally, a progressive imbalance between model-based and model-free control of the potentially addictive activity), individuals end up losing full intentional control over that activity. A non-problem recreational gambler, for instance, would start gambling because they expect it to be pleasurable or beneficial in some way (positive reinforcing), or help curb their negative affect (negatively

reinforcing). This could progress into an addiction to gambling as certain conditioned cues acquire the power to trigger the compulsion to gamble. Thus, some substances are addictive not only because they are positively or negatively reinforcing, but also because they boost compulsivity via direct or indirect neurochemical pathways (Koob, 2009). Importantly, in the absence of a substance, that effect has been proposed to depend on the features of the reinforcement schedule through which reward for a specific behavior is delivered. In the case of gambling, and in some experimental protocols, certain intermittent reinforcement schedules seem to be maximally effective at generating compulsive-like behaviors (Anselme et al., 2013; Rømer Thomsen et al., 2014; Ross et al., 2012).

From this argument, the inference seems to be that, as addictive behaviors are compulsive (or controlled in a model-free mode), they are also completely independent of model-based control. That is not the case; the RL model-free/model-based framework actually assumes that no behavior is completely controlled in a single-mode fashion, but control is better defined as resulting from a combination of modes, in which one or the other can be more influential. Decisions –including those involving a potentially addictive behavior– result from the competition between the representations of the different available actions for behavioral expression, and each of these is subject to positive and negative forces from model-free and model-based systems of behavioral control. Consequently, although instrumental behaviors may become *less sensitive* to contingency manipulations as they turn into addictive behaviors, they never turn *completely insensitive* to such manipulations (Lamb and Ginsburg, 2018). In fact, some elements of addiction treatment rely on providing alternatives to drug use, and training in the skills necessary to attain them (e.g., McHugh et al., 2010).

This notion that addiction does not result in a complete lack of agency, and that compulsive and rational desires can interact at a common neurocognitive level has been described in detail by Holton and Berridge, (2013). Accordingly, in our framework, the key question is not whether or not people suffering from an addictive disorder have 'free-will' (i.e.,

whether or not their addictive behavior is sensitive to reflection on costs and benefits [Heyman, 2017]) in a dichotomous manner. Instead, the key issue is about the relative weight of cost-benefit analysis and compulsivity and the subsequent balancing between the two in decision-making. The evidence that behavior control is influenced by mutually opposing forces opens the possibility for different problematic behaviors to be more or less controlled in one mode or the other. This also allows model-free/model-based contributions to such behaviors to vary across individuals or lifetime trajectories (Lopez-Quintero et al., 2011). Understanding the factors accounting for that variability remains an open question. Still, as it will be described below, one of the aims of this work is to provide guidelines for individual assessment and to tailor treatment in function of individual differences.

4. Ancillary processes

According to the general framework outlined above, the *signature* of addiction is the increasing role that compulsivity plays in the activity that one becomes addicted to. Importantly, several ancillary processes can facilitate this process and thus increase vulnerability to addiction, or contribute to its maintenance or aggravation. For example, it has been reported that sensitization of the wanting system is accompanied by a progressively reduced hedonic response to the addictive agent (see Volkow et al., 2014 and Wang et al., 2014, for examples regarding substance use disorders and obesity). This hedonic decline can be accounted for by habituation and Pavlovian conditioning of homeostatic regulation (Koob and Le Moal, 2008) and has been proposed to underpin the pattern of behavior known as "chasing the remembered high" (Volkow et al., 2017).

Also related to the functioning of reward systems, blunting of the pleasure derived from a broad range of rewards and activities (other than the addictive ones) has also been proposed to be characteristic of addiction. *Reward deficiency* (Clark et al., 2019; Parvaz et al., 2011; Sescousse et al., 2013), that is, generalized hypo-responsivity to rewards, can occur as a

consequence of addiction, but can also predate it and increase vulnerability in terms of uncontrollably pursuing artificial rewards (van Holst et al., 2010).

Also in a domain-general manner, as pointed out earlier, the weakness of executive control can result in various forms of impulsivity (Nigg, 2017; Sharma et al., 2014). The RL framework outlined above can help understand how this link operates. In the model-based mode, when faced with a decision, the individual needs to foresee a map of possibilities including future states and actions (see O'Doherty et al., 2017, for a detailed review of the neurobiological bases of these *cognitive maps*). The best course of action is selected by exploring paths on the map and accumulating utility along each path. Every imagined potential pathway will accrue positive and negative utility (i.e., potential outcomes with positive and negative subjective value), and the probability of selecting one of them will depend on its utility, as well as on the utility of the alternative pathways under consideration. Crucially, this projection is cognitively taxing, and planning and working memory capacity determine how far one can look into the future and how many alternatives one can explore. In other words, limitations in executive capacity can result in an imbalance between the two control modes, in favor of the model-free control, and thus more reflexive behavior. Moreover, brain abnormalities compromising prefrontal integrity can potentially weaken affect model-based control, and thus behavior regulation. Thus, studies showing prefrontal functional or structural alterations in putative behavioral addictions are no evidence by themselves that such conditions should be considered addictive in nature. As noted earlier, domain-general executive dysfunction is neither necessary nor sufficient for development of an addictive disorder.

Finally, not only overt behaviors, but also covert ones can be under model-based or model-free control. For instance, Etkin et al., (2015) have used the model-free vs. model-based control dichotomy to classify *emotion regulation* processes and hypothesize the involvement of executive functions in model-based emotion regulation. Given the well-established role of

emotion dysregulation in addictions (Cheetham et al., 2010; Navas et al., 2019, 2017; Volkow et al., 2016), this involvement reveals a new path by means of which anomalies of executive functions can contribute to addictive processes.

5. Abnormal relative utility computation

Up to this point, we have argued that as the addictive process progresses, behavior-specific compulsivity plays an ever-increasing role. Still, there is evidence that certain activities can become problematic, excessive, or dysregulated, despite remaining oriented towards declarative motives. In other words, some problematic behaviors can remain mostly under model-based control, and still become a source of distress and cause significant harm.

As noted above, not all activities need to be pleasurable to be rewarding, but can be reinforced in indirect ways (Caretti et al., 2018; Khantzian, 1997). First, the putative addictive activity can be a source of negative reinforcement: virtually all potentially addictive activities are mood modifiers and can be used to cope with negative emotional states, which is consistent with the common observation that clinical or subclinical distress is a vulnerability factor for a variety of problematic behavior patterns (Devos et al., 2018; Kardefelt-Winther, 2017a; Lister et al., 2015; Plante et al., 2018). Second, under certain circumstances, an otherwise dysfunctional activity can become the preferred strategy for the individual to attain (or retain) a personally relevant state. If certain desirable states cannot be attained by other means except through a potentially problematic activity, the probability of falling for that activity will rise, in spite of its negative consequences.

A recent review by King et al. (2020) has shown that most maladaptive player-game relationships in (video) gaming disorder can be defined in this way, with the contents and features of different games being more or less suited to the motives for playing. For instance, excessive involvement in Massively Multiplayer Online Role Playing Games (MMORPGs) can be largely accounted for by motives related to the maintenance of self-esteem, coping with negative affect, replacement of the real identity by a virtual one, or within-game achievements

(Billieux et al., 2015a). Some of these motives are 'negative' as they are related to coping with negative emotional states. Others however, are 'positive' in the sense that they are related to obtaining rewards that the individual is unable or unwilling to pursue in other ways.

Another example of this mechanism is the *secondary dependence* hypothesis of 'exercise addiction' (Landolfi, 2013), according to which excessive exercising emerges as a strategy to cope with symptoms of other clinical or subclinical disorders. In the best-known case, excessive exercising is used to maintain weight loss, so that individuals with excessive exercising (particularly those with lengthy aerobic sessions) are about two times more likely to present clinical symptoms of eating disorders compared to individuals without such a pattern of exercising (Cardol et al., 2019).

Importantly, these potentially problematic goal-oriented behaviors are not controlled only by the absolute value of their expectancies, but also by their comparative value, relative to the set of outcomes that can be achieved with alternative behaviors[3] (*relative outcome utility*; see Borders et al., 2004, for a similar proposal). Across a variety of domains, problematic behaviors can be weakened by increasing the reinforcement rate of alternative behaviors or training skills to access new sources of reward, as predicted by the matching law (Correia, 2004). This is the case of problematic activities that have been conceptualized by some authors as behavioral addictions or impulse control disorders, as, for example, self-injury behavior (Symons et al., 2003), overeating (Epstein et al., 2005; Lappalainen and Epstein, 1990), or excessive gaming (Torres-Rodríguez et al., 2018). It could even be the case that

[3] These competitive processes can also contribute to some components of putative behavioral addictions. For example, problematic activities can greatly diminish naturally rewarded activities. This turns the latter into weaker competitors and makes the problematic activity even more intense. Complementarily, according to the *bliss point hypothesis*, when behavior is not externally constrained, individuals distribute their time among the available activities to reach an equilibrium point and thereby maximize total utility (Allison, 1993; McFall et al., 2019). That equilibrium can nonetheless be 'pathological', when a single activity occupies a disproportionate amount of the individual's time. In that case, inability to engage in such an activity and the subsequent breakdown of balance trigger a motivational state to resume this activity.

externally constraining one potentially problematic behavior leads to an increase in another (Castro-Calvo et al., 2018).

In summary, in many cases of putative behavioral addictions, the individual resorts to problematic activities to achieve goals that are not evident from an external observer's point of view. Moreover, the utility of these outcomes can override others that can be achieved by means of non-problematic behaviors. Importantly, this overriding can occur because the individual lacks alternative skills (e.g., face-to-face social skills) or cannot engage in functional behaviors (e.g., healthy eating) to attain the same or similarly valuable goals. There is little doubt that the resulting behavior pattern, despite remaining mostly goal-driven, can be severely dysfunctional.

6. So, what is a behavioral addiction?

To sum up, there are at least two pathways through which positively or negatively reinforcing activities can become dysregulated and eventually problematic: domain-specific compulsivity and relative outcome utility computation. Although the most recent conceptualizations of these pathways allow both of them to simultaneously influence response selection, domain-specific compulsivity seems to be the predominant mechanism in well-established addictive disorders (Ross et al., 2012).

The presence of compulsivity seems thus necessary for a behavior to be considered addictive. So, ascertaining the presence of compulsivity in putative behavioral addictions appears crucial for their precise characterization. Indeed, it has been proposed that the features of reinforcement schedules that drive compulsivity in gambling disorder could account for the attention-grabbing effects of notifications and other cues in social networks (Rosen et al., 2013), as well as for the time-wasting effects of the infinite scroll feature in many apps (Fishman, 2018). Relatedly, commercial video games are being progressively *gamblified* through the insertion of compulsivity-enhancing reinforcement schedules (e.g., loot boxes), which could partly explain their alleged addictive potential (Clark, 2015; King and Delfabbro,

2018). Yet, this evidence is still very preliminary, with no evidence of the same kind for other candidate addictions, which has led some authors to suggest that such conditions should better be conceptualized as maladaptive coping or disorders related to emotion dysregulation (Aarseth et al., 2017; Kardefelt-Winther, 2017a).

Still, growing evidence supports the second mechanism –relative outcome utility computation– as an important factor in behavior dysregulation (Billieux et al., 2015b; Cardol et al., 2019; Landolfi, 2013; Mathieu et al., 2018). For example, as noted by Sztainert et al., (2014): "pathological gamblers should find treatment seeking aversive because doing so would remove a route to reward" (p. 901). In other words, even after gambling has become compulsive, utility in the form of potential expected reward (which cannot be attained using other behaviors) can continue to play an important role in maintaining disordered gambling (see also Sescousse et al., 2013).

Our main proposal here is that both (1) the degree of compulsivity, and (2) the relative utility of behavior outcomes can contribute to functional impairment, mostly by displacing adaptive goal-directed behaviors. As a consequence, we contend that problematic behaviors customarily labelled as behavioral addictions are better conceptualized as belonging to a spectrum of related, yet distinct, conditions in which these behaviors are driven by compulsivity or relative outcome utility, whereby their clinical threshold is defined by significant functional impairment. However, and to establish an unambiguous terminology, a key issue remains to be resolved: do all these types of dysregulated behavioral patterns qualify not only as disorders, but as *addictive* disorders? Whatever the best answer is, the behavior-based, process-blind use of the term should be avoided (Starcevic et al., 2018). This is so because unveiling etiology (i.e., the balance between relative outcome utility and compulsivity) is crucial for tailoring prevention and treatment.

According to Ersche et al. (2016), individuals suffering from a substance use disorder seek drugs in an automatic fashion, irrespective of the consequences, so addictive behaviors

are unlikely to be affected by cognitive interventions based on punishment of drug seeking or enhancement of alternative outcomes[4]. In other words, as far as compulsivity plays a significant role in an addictive behavior, treatment should address compulsivity using controlled exposure[5] (e.g., Park et al., 2015), implementation intention (e.g., Gollwitzer, 1999), or craving management skills (e.g., Naqvi et al., 2015), or aim to reduce harms via stimulus control and relapse prevention (see Rash and Petry, 2014). In contrast, behavior modification techniques based on contingency management (Coleman and Pasternak, 2012) are probably more promising for problematic behaviors mainly driven by relative outcome utility.

Unfortunately, to date, no simple diagnostic tool exists to quantify the relative contribution of these two forces to any putative behavioral addiction. For example, questionnaire items pertaining to the difficulty of staying focused on activities unrelated to the problematic behavior (e.g., Cho et al., 2014) can reflect both a loss of interest in the alternatives (relative outcome utility) and the hijacking of attention by cues related to the problematic behavior (compulsivity). In the absence of standardized assessment and diagnostic tools, the only available way to design a treatment plan is to capitalize on an individualized functional analysis, where environmental, cognitive, affective, interoceptive, and motivational triggers and consequences are clearly identified (Billieux et al., 2015c; Dudley et al., 2011; Rochat et al., 2019; Wéry et al., 2019). On the one hand, automaticity, lack of control despite awareness of the negative consequences, and dissociation between pleasure and urges could be taken as signs of compulsivity. The identification of (sometimes hidden) functionally relevant sources of positive or negative reinforcement, on the other hand, could

[4] This position has been recently subjected to criticism, as insensitivity to punishment, at least in animal research, may characterize drug seeking in a small proportion of vulnerable individuals (Pelloux et al., 2015). Alternatively, from an incentive sensitization perspective, punishment insensitivity would result from an inability of punishment expectancy to override compulsive 'wanting' (Kawa et al., 2016).

[5] Exposure techniques remain controversial in the addiction treatment literature (and particularly in the case of gambling disorder). Although, in theory, exposure to sensitized cues is necessary for extinction of craving to occur, it is also true that exposure strongly triggers cravings, putting the patient in a serious risk of relapse that can extend beyond the exposure session (Giroux et al., 2013; Smith et al., 2015). The risks of relapse and therapy dropout must thus be carefully addressed and assessed against the potential benefits of exposure therapy (relative to other treatment alternatives).

reveal the influence of relative outcome of utility computation. Importantly, these possibilities are not mutually exclusive but can coexist in different proportions in various individuals and be simultaneously relevant for the same behavior.

Nevertheless, the development of etiology-sensitive diagnostic tools remains a challenge (for an attempt in this direction, see Cornil et al., 2018). Using the overarching 'addiction' label has probably contributed to perpetuating a tendency to flood the literature with psychometric tools for new addictions that are far from optimal for the aims of prevention and treatment (see, for example, Long et al., 2015). Hence, we advocate to restrict the use of the concept of *addiction* to excessive behavioral patterns in which there is clear evidence of domain-specific compulsivity. Specificity, however, must not come at the cost of neglecting conditions in which compulsivity plays a less relevant role, but in which clinically significant impairment levels are nonetheless caused by non-adaptive relative outcome utility computation.

7. Facing the challenge of new disorders for which evidence remains controversial

In spite of the issues raised in previous sections, provisional recognition of a problematic behavior pattern as a mental disorder may be justified from a public health perspective. As a prototypical example, the inclusion of gaming disorder in the ICD-11 has been claimed to be of public health importance, although some gaps in the existing evidence – regarding for example underlying learning processes or effective treatments – warrant critical attention and further research (Fineberg et al., 2018; Rumpf et al., 2018; Stein et al., 2018). According to some authors, the recognition of the condition is necessary to address the increasing demand of treatment, promote information and prevention, or facilitate the implementation of regulations that limit gambling features to be incorporated into video games (King and Delfabbro, 2018). Critics of this position, however, have argued that recognizing conditions that deserve clinical attention does not imply the necessity to generate a clinical diagnosis for that condition (van Rooij et al., 2018). For the sake of maximum possible consensus, our stance

in this controversy remains neutral, thereby discouraging terminological distractors and fostering crucial research collaborations instead.

For problematic behaviors with equivocal evidence, and to avoid the exponential growth of new symptom-based disorders, it is important to clearly define functional impairment as a criterion for distinguishing between dysfunctional behavior and high, but non-problematic participation in a specific activity. If high engagement in a certain behavior does not lead to impairment, there is no reason to consider it problematic. Indeed, some authors have proposed the term *passion* for referring to that type of high, non-pathological engagement (Vallerand et al., 2003). In accordance with this view, a significant proportion of individuals identified as addicted by current behavioral addiction scales – usually developed by recycling substance use disorder items – should be regarded as individuals strongly committed to their favorite activity (Calvo et al., 2018; Deleuze et al., 2018; Fuster et al., 2014). Importantly, the implausibly high prevalence rates of putative behavioral addictions reported in some studies (typically involving Internet-related disorders, [see Kuss et al., 2014]) can be partially attributed to screening tools failing to distinguish high commitment from problematic involvement. As already noted, this distinction depends on the clear conceptualization of functional impairment, which constitutes a research area itself, and includes significant reductions of satisfaction and functioning in the recreational, school/work, self-care, socioemotional, and responsible behavior domains (Colburn et al., 2018).

8. Final remarks

To some extent, the controversy around the concept of behavioral addiction has been a semantic conflict, which has obscured the discussion on the causes of behaviors that lead to psychological distress. Arguably, the unrestricted use of the term 'addiction' may increase the risk of stigma for people with high degrees of commitment to leisure activities, generate false positives, and render treatment guidelines ineffective. On the other hand, overly restrictive use – or opposing the intermediate position that some behaviors that do not qualify as

addictions can still be considered disordered[6] – can neglect a real public health problem and discourage the investment of effort and resources to address it. Therefore, both of these positions are vulnerable to criticism, calling for an effort to reach an agreement that would incorporate key elements of each position.

A partially overlapping dispute has developed around the brain disease model of addiction, and whether such a model could (or should) apply to behavioral addictions (see Lewis, 2018). Although Berridge (2017) noted that in the learning approach "neural sensitization changes are arguably extreme enough and problematic enough to be called pathological" (p. 29), the term 'disease' may suggest that addictions do not emerge from originally adaptive learning processes and may obscure the importance of the social context and the active agency of the individual in such learning processes. Consequently, we find the brain disease model of addiction too restrictive in its scope.

In line with Berridge's proposal, we conceptualize behavioral addictions as lying at an extreme end of the continuum of normal psychological-neural processes, which leads to functional impairment. The same learning mechanisms underlying compulsivity have adaptive functions when they remain within certain limits. So, beyond terminology, and as suggested by James and Tunney (2017), theorists are encouraged to turn their attention to the specific processes that underlie acquisition and maintenance of addictive behaviors, while clinicians are urged to incorporate such processes into diagnosis and treatment. In our view, the learning-based approach to addiction is already developed enough to provide specific, causality-informed and practical fieldwork hypotheses, as described in this paper.

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[6] This position is exemplified by authors who accept the existence of (video) gaming disorder, but not its consideration as an addictive disorder.

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Contributions

JCP wrote the first draft of this manuscript. JCP, JB and JFN conceived the main ideas. All authors made substantial conceptual contributions, and revised the manuscript in successive rounds.

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