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Neurocognitive components of gambling disorder: Implications for assessment, treatment and policy

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Aims and scope

The present chapter aims to describe the psychobiological bases of gambling disorder (GD), and to identify how neuroscience research could inform better prevention and treatment strategies.

In the first section, we describe the characteristics shared by patients with gambling disorder (PGD), and revisit the literature showing that GD is in essence a disorder of learning. Among vulnerabilities, we highlight factors incrementing the allure of gambling, making it more rewarding, or strengthening its negatively reinforcing properties.

Second, we pinpoint the variables contributing to individual differences within the PGD population, with a particular focus on emotion regulation. Dysregulation of automatic (model-free) emotion regulation is suggested to be a complicating factor of GD, and a transdiagnostic vulnerability factor for psychopathology beyond GD. Dysregulation of controlled (model-based) emotion regulation strategies, along with gambling-related cognitive distortions, are hypothesised to contribute to self-deceptive thinking in some gamblers.

Lastly, all these variables are integrated into a dimensional model (the *Gambling Space Model*), aimed at updating previous cluster-based proposals to subtype PGD, by incorporating recent neurocognitive evidence. The implications of the model are discussed, and we address its implications on policy and regulation. Additionally, we discuss whether or not other putative behavioural addictions should be ascribed the same consideration. Eventually, we analyse how better understanding individual differences could contribute to better treatment and prevention designs.

Homogeneity in gambling disorder: Incentive sensitisation as the mechanism of gambling conditioning

Gambling research has flourished in recent years, and can be considered an example of integration of knowledge from different disciplines. The recent reconceptualisation of GD as an addictive disorder (American Psychiatric Association, 2013) and the ensuing translational advances would have not occurred without such cross-talk.

In a joint attempt to define addictive disorders, animal and human research, behavioural neuroscience and cognitive neuroscience have converged in stressing the importance of progressive detachment of addictive behaviour from instrumental goals. According to some etiological models, addictive behaviours are problematic because they become *habit-driven* or *compulsive* (Everitt & Robbins, 2016). Other models stress that individuals with addictive disorder cannot help *craving* their addictive substance or activity (Skinner & Aubin, 2010). Beyond the subtleties of these two approaches, here we will stress their commonality; namely, the fact that wanting (to gamble, to use the drug) and seeking behaviour, once a person meet criteria for addictive disorder, have little to do with the hedonic properties of drug use/gambling consequences.

Craving is thus best defined as a multifaceted construct, manifested by the urge to engage in the addictive behaviour, automatic hijacking of attention and cognitive resources by cues reminding or signalling the availability of the object of desire, and imperative approach responses to such cues. With regard to its proximal and distal causes, there is also convincing evidence that craving is cue-driven, and acquired through exposure to the addictive agent (Sayette, 2016).

In keeping with the importance of craving in substance use disorders (SUDs; Kober & Mell, 2015) considerable efforts have been made to prove the existence of gambling craving (Ashrafioun & Rosenberg, 2012). Additionally, recent evidence suggests that craving may involve a common brain circuitry, with an important hub in the insula, independently of the type of addictive disorder (Garavan, 2010; Limbrick-Oldfield et al., 2017). The association between this area and cravings accords with its status as an important node in interoceptive representation, and the use of such information in decision-making (Garavan, 2010).

From a practical point of view, however, the key question is how gambling *becomes* compulsive, and wanting detaches from hedonic value. A leading model in accounting for drug craving acquisition is the *incentive sensitisation* (IS) hypothesis. The IS model posits that all addictive drugs directly or indirectly sensitise dopamine release in the mesolimbic system, which is responsible for attributing *incentive salience* to cues signalling the availability of reward (Robinson & Berridge, 1993).

Incentive salience is hypothesised to be the inner engine of craving acquisition. In normal circumstances, when an unexpected reward is encountered, a mismatch between predicted and experienced utility generates error signals in the mesocorticolimbic system, and particularly in the ventral striatum (VS; Humphrey & Richard, 2014). However, rewards become more predictable as instrumental learning progresses, so the magnitude of error signals decreases, and incentive salience reaches asymptote. Drugs of abuse alter this system by producing supra-threshold stimulation and precluding habituation, and thus causing "irrationally strong motivation urges that are not justified by any memories of previous reward values (and without distorting associative predictions)" (Berridge, 2012, p. 1124). In other words, substance use disorders (SUDs) are normally accompanied by a subjective and behavioural dissociation between *liking* and *wanting* the drug (Pool, Sennwald, Delplanque, Brosch, & Sander, 2016).

In view of the success of the IS hypothesis to account for some of the seemingly irrational features of SUDs patients' behaviour, the question arises whether the hypothesis can be also tested in behavioural addictions (Rømer Thomsen, Fjorback, Møller, & Lou, 2014). That is, in the absence of an external chemical agent, what misleads dopaminergic error signals?

This question can be addressed by revisiting the literature on reinforcement schedules. According to recent analyses, most gambling behaviours are under random ratio (RR) schedules (Haw, 2008). These are characterised by intermittent reward, such that the probability of reward in any single trial does not depend on the previous density of rewards. Uncertainty in RR

schedules is irreducible, and the rates of responding they generate are particularly stable and free of breaks after reward (Schoenfeld, Cumming, & Hearst, 1956).

Irreducible uncertainty in gambling scenarios can be regarded as a constant source of prediction error for the mesocorticolimbic dopaminergic system to feed incentive salience. Supporting that hypothesis, Anselme, Robinson, and Berridge (2013) have indeed shown that increasing the uncertainty level in the relationship between a cue and a reward enhances incentive salience of this cue, as measured by a sign-tracking response. Further evidence supports the involvement of the incentive salience dopamine system in the effect of uncertainty on the ability of contextual cues to behave as motivational magnets (Anselme & Robinson, 2013).

Sources of heterogeneity in gambling disorder

Differences in gambling disorder vulnerability

If IS resulting from RR schedules is the main learning mechanism underlying gambling conditioning, any factors fuelling this mechanism will contribute to GD vulnerability. More specifically, any factors increasing exposure to gambling or its rewarding properties will facilitate transition from recreational to problematic gambling. Accordingly, research shows that early wins have a particularly strong effect on behaviour under RR schedules and in gambling scenarios (Haw, 2008).

Complementarily, people differ in the degree to which they are sensitive to the various appetitive and aversive properties of different types of events. Gray's (1994) psychobiological model of personality proposes reward sensitivity (RS) and punishment sensitivity (PS; the overt manifestations of two biological systems referred to as behavioural activation and behavioural inhibitions systems) as the main foundation of motivation and personality. In the framework we are starting to sketch here, RS and PS easily enter the equation as individual differences that modulate IS. However, reinforcement-related sources of individual vulnerability could be less general than PS and RS traits, and more circumscribed to the types of rewards that occur in gambling scenarios.

There is evidence, for instance, that some gamblers experience gambling-triggered arousal or uncertainty as intrinsically rewarding (Megias et al., 2017; Sharpe, Tarrier, Schotte, & Spence, 1995), a result that converges with studies on the biological basis of individual differences in risk proneness in animals (Fiorillo, 2011). Complementarily, individuals presenting high levels of neuroticism and punishment sensitivity, or proneness toward negative mood, are more likely to use gambling to cope with psychological distress (Balodis, Thomas, & Moore, 2014).

The role of basic emotion regulation mechanisms

A growing corpus of evidence suggests that craving management, that is, succeeding in keeping IS below a given threshold, can be viewed as an instance of emotional regulation (Loewenstein,

1996) that can be implemented at different levels.

IS is subject to influences from same-level learning mechanisms (e.g. extinction, counter-conditioning, cue-interaction; Kober et al., 2010). Etkin, Büchel, and Gross (2015) have recently proposed that, at this level, emotion regulation proceeds in a model-free, automatic manner. This could be the case for loss-related learning processes necessary to compensate IS. Supporting this idea, a recent study has showed casino gamblers to underestimate how much money they spend on gambling in the long run. And, that their gambling expenditures could be reduced just by providing them with a player account with their personal spend (Wohl, Davis, & Hollingshead, 2017). Interestingly, behaviour changed with limited or no awareness. Accordingly, manipulations that reduce loss awareness increase wagering, in a similar, mostly automatic way (Monaghan, 2009).

Etkin and colleagues (2015) have identified the ventromedial prefrontal cortex (vmPFC) and the ventral anterior cingulate (vACC) as the main regions in the circuit for model-free emotion regulation, although their review mostly focuses on fear regulation and it is unclear whether these would also constitute the most important structures for craving regulation. A discussion on the exact brain implementation of model-free emotion regulation goes beyond the scope of the present chapter. Medial and ventral parts of the PFC, the insula, and their connections with the amygdala and the VS are, however, the most frequently mentioned structures (Phillips, Ladouceur, & Drevets, 2008).

With regard to the model-free regulation of craving in GD, the evidence to date remains indirect. For example, Contreras-Rodríguez et al. (2016) found a common pattern of hyperconnectivity in PGD and cocaine-dependent individuals, mostly between the orbitofrontal cortex (OFC) and VS, and between the insula and the amygdala. Complementary evidence comes from studies showing that PGD perform worse than controls on the Iowa Gambling Task, in which successful performance is known to depend on balanced emotion-driven learning (Buelow & Suhr, 2009).

Malfunctioning of basic, model-free emotion regulation will be subjectively experienced as a pervasive influence of craving on behaviour, and if such malfunctioning is extensive enough, as a disproportionate impact of emotions in other areas of decision and action. This resonates with similar findings in the SUDs literature that craving correlates with negative urgency (NU), namely the tendency to act rashly under the influence of strong negative emotions (Cyders et al., 2014; Doran, Cook, McChargue, & Spring, 2009).

Higher-order emotion regulation mechanisms

Model-free emotion regulation is complemented by model-based emotion regulation strategies. These form a category of learned goal-directed responses through which people act upon their own emotional processes. Not surprisingly, then, specific cerebral areas involved in this type of

emotion regulation (lateral PFC, pre-supplementary and supplementary motor areas [pSMA, SMA], and parts of the parietal cortex) overlap with those involved in model-based instrumental behaviour (O'Doherty, Cockburn, & Pauli, 2017).

Emotion regulation strategies are varied. The emotion regulation questionnaire (ERQ; Gross & John, 2003) distinguishes between *expressive suppression* (suppressing the external manifestations of emotion), and *reappraisal* (reprocessing of the causes of the emotion), with use of the latter being considered adaptive and the former maladaptive. The more comprehensive cognitive emotion regulation questionnaire (CERQ, Garnefski & Kraaij, 2007) identifies nine cognitive strategies to deal with negative affect (*blaming oneself*, *blaming others*, *acceptance*, *rumination*, *positive refocusing*, *refocus on planning*, *positive reappraisal*, *putting into perspective*, and *catastrophising*).

In psychobiological terms, reappraisal has been shown to downregulate the activity of the VS and the amygdala, altering the balance in favour of either continuing or interrupting gambling (Kober et al., 2010; Sokol-Hessner et al., 2009). Accordingly, studies on craving regulation have focused on this cognitive strategy (Giuliani & Berkman, 2015), and have observed that successful downregulation of craving is associated with increased activity of the lateral and dorsomedial PFC, and dampened activity of the ventral striatum, subgenual cingulate, amygdala, and ventral tegmental area (Kober et al., 2010).

GD can progress with malfunctioning of the basic mechanisms necessary to regulate craving and other undesirable emotions, and this can have an influence on how model-based strategies operate. In a recent study, we tested the hypothesis that regulation of negative emotions in PGD imposes an extra burden on cognitive control mechanisms, relative to healthy controls (Navas et al., 2017b). Downregulation of emotions triggered by negative pictorial stimuli activated the control network in controls and PGD, but the latter showed further hyperactivation of an area comprising parts of the premotor cortex and the dlPFC. Additionally, activation of dlPFC correlated with NU. In a separate sample, NU significantly correlated with the proneness to use expressive suppression as a (maladaptive) strategy to regulate negative affect.

The Gambling Space Model

So far, we have suggested a number of psychobiological processes (1) to account for the transition from recreational to problem gambling, (2) to facilitate that transition and contribute to GD vulnerability, and (3) to underlie individual differences in PGD. In the next section these constructs are integrated into a coherent model (Table 1), and their contribution to the behavioural manifestations and clinical implications of disordered gambling are explicated.

Table 1. The Gambling Space Model

Construct	***Sensitivity to positively reinforcing properties of gambling***	***Sensitivity to negatively reinforcing properties of gambling***	***Generalized affect dysregulation***	***Cognitive elaboration and self-deception***
Psychobiological basis	Reward system, uncertainty-sensitive dopaminergic projections	Fronto-amygdalar systems of escape and avoidance	Model-free emotion regulation systems	Model-based emotion regulation system, cognitive control structures
Behavioural manifestations	Positive motives for gambling, reward seeking	Negative motives, poor mood, neuroticism, boredom	Affect-driven impulsivity, disinhibition, deficits in decision making	Exaggerated expectancies, interpretative biases, motivated reasoning
Clinical implications	Vulnerability to risk gambling, low motivation to quit gambling, dropout risk	Emotional vulnerability, internalizing comorbidity, risk of relapse	Low problem awareness, externalizing comorbidity, dropout risk	Cognitive distortions, preference for skill-based games, low change motivation, treatment reluctance
Common construct	***Incentive sensitization driven by random ratio schedules***			

The first construct in the Gambling Space Model (*sensitivity to appetitive properties of gambling*) is related to reward sensitivity. The relationship between RS and gambling has been lingering in the literature for decades, yet it has been difficult to identify it as a strong and independent predictor of disordered gambling behaviour (Goudriaan, van Holst, Veltman, & van den Brink, 2013). More consistently, gamblers have been found to differ from non-gamblers in how they respond to the different sources of reward present in gambling scenarios (Sescousse, Barbalat, Domenech, & Dreher, 2013). Still, reward sensitivity can interact with gambling features in shaping individual gambling preferences. In a recent article, Navas et al. (2017a) found that recreational and disordered gamblers preferring card, skill and casino games show higher RS scores than those preferring slot machines, lotteries, and bingo. These gamblers are more strongly motivated by positive reinforcers, and also more sensitive to the positive features of the gambling

experience.

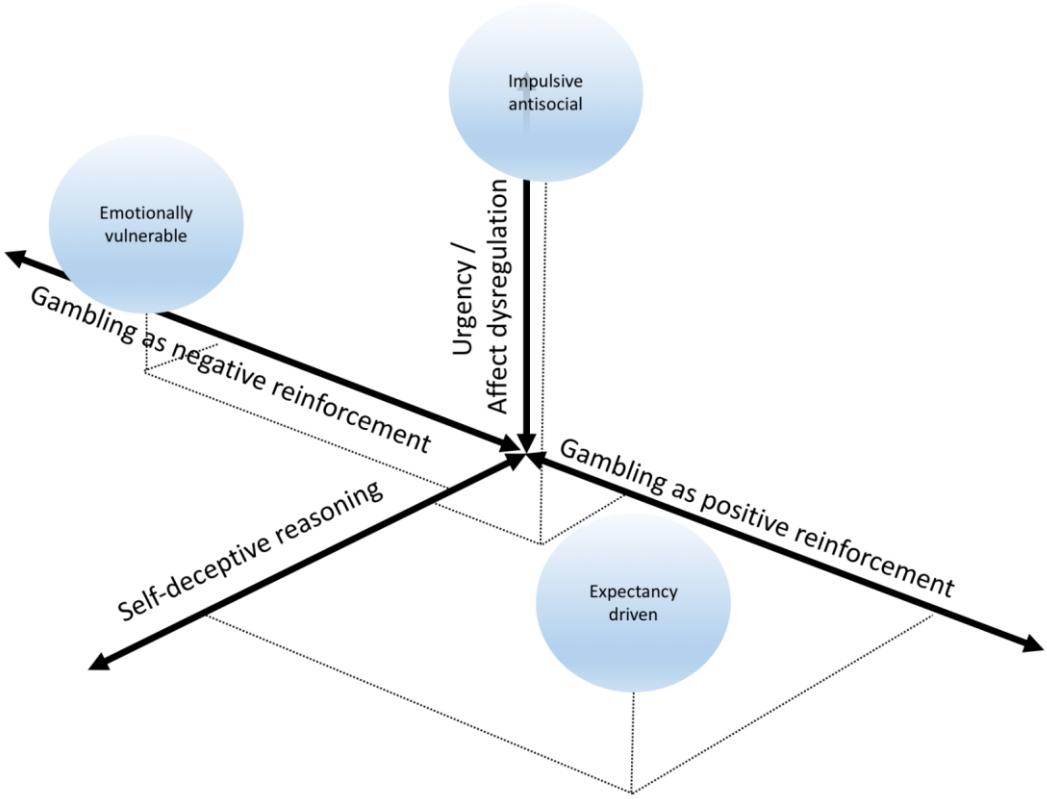
The second putative construct relies on the *negatively reinforcing properties of gambling*. Negative trait emotions can interact with sensitivity to the mood-modifying properties of gambling. In practical terms, gambling-to-cope has been observed to correlate with comorbid depression and relapse risk (Ledgerwood & Petry, 2006; Lister, Milosevic, & Ledgerwood, 2015)

The third construct, *generalised emotional dysregulation*, captures deterioration of model-free emotion regulation mechanisms. Weakness of low-level regulation mechanisms necessary to limit gambling conditioning are hypothesised to characterise all disordered gamblers. However, extensive malfunctioning of basic emotion regulation mechanisms is likely to be responsible for differences among PGD. Unfortunately, to date, there is a dearth of reliable and psychometrically sound neurobehavioural tasks that could be used as tools to assess the extent and severity of this type of dysregulation. Provisionally, we propose NU as the most promising available proxy to evaluate it across disorders (Berg, Latzman, Bliwise, & Lilienfeld, 2015).

Finally, the fourth construct has to do with the use of strategic, model-based emotion regulation. Recent evidence shows the existence of a subgroup of PGD who effectively use putatively adaptive forms of emotion regulation (Navas, Verdejo-García, López-Gómez, Maldonado, & Perales, 2016). However, in these patients, such strategies correlate *directly* with gambling severity and gambling-related cognitive distortions. In a similarly counterintuitive fashion, gamblers with high dispositional optimism have been found to be more prone to maintain positive expectations and remain motivated to gamble after negative outcomes (Gibson & Sanbonmatsu, 2004). In our model, gamblers' use of model-based emotion regulation strategies in combination with certain gambling-related cognitive distortions forms part of a *self-deceptive reasoning style*. This ego-protective mechanism has been established as a factor contributing to drug use perseverance, and to reluctance to treatment (Martínez-González, Vilar López, Becoña Iglesias, & Verdejo-García, 2016). Here, we posit that self-deception has an emotional regulation function, in line with models of motivated reasoning (Kunda, 1990).

According to the present model, certain types of cognitive distortions thus reflect *spared* cognitive control, rather than cognitive dysfunction. Indeed, cognitive distortions are more frequently encountered in young, educated, skill-game gamblers (Myrseth, Brunborg, & Eidem, 2010). Furthermore, and importantly, they are not systematically accompanied by signs of cognitive/non-planning impulsivity or lack of conscientiousness (Navas et al., 2017a). This relationship between elaborate distorted cognitions and planning abilities could partially account for the inconsistency of findings regarding the link between GD and executive tasks (Goudriaan, Yücel, & van Holst, 2014). In self-deceptive gamblers, preserved executive function would contribute to false mastery, whereas in patients with less elaborated gambling beliefs weaker executive functions would contribute to inflexible behaviour and unconscientious gambling.

Figure 1. A simplified depiction of the mapping of gamblers subtypes onto a dimensional model.



It is worth noting that there are important connections between our Gambling Space Model and the *Pathways Model* (Blaszczynski & Nower, 2002). Figure 1 displays a simplified depiction of the mapping of the Pathways Model onto the Gambling Space Model. In this space, all PGD are *conditioned gamblers*, and subtypes would arise from the combination of conditioning processes with sources of heterogeneity. In individuals with high levels of neuroticism, poor mood or susceptibility to boredom, the negatively reinforcing properties of gambling would give rise to the *emotionally vulnerable gambler*, whereas in cognitively spared individuals, motivated reasoning and elaborated emotion regulation could give rise to *self-deceptive gamblers*. The latter are not specifically considered in the Pathways Model, but are easily identifiable in emerging profiles (Griffiths, Wardle, Orford, Sproston, & Erens, 2009).

With regard to the *impulsive-antisocial gambler* type, our model depicts a slightly more complex scenario. Given the partial overlap between RS and impulsivity (Knezevic-Budisin, Pedden, White, Miller, & Hoaken, 2015), reward-sensitive GD patients have remained partially confounded with impulsive-antisocial ones. RS, however, reflects the hyper-reactivity of the behavioural activation system to potential sources of, whereas other relevant aspects of emotion-driven impulsivity reflect a more generalised regulatory dysfunction. Hence, it would be possible to distinguish between predominantly reward- or sensation-seeking impulsive gamblers, and gamblers with high levels of urgency, with the latter presenting a higher incidence of problematic behaviours (Vachon & Bagby, 2009).

What has neuroscience ever done for us? Summary and implications

According to a recent opinion article by Markowitz (2016), "*there is such a thing as too much neuroscience*" in psychopathology and psychotherapy research. In the context of GD research, it is true that clear-cut biomarkers are still lacking, drug trials have yielded inconclusive, mixed or unspecific results (Alexandris, Smith, & Bowden-Jones, 2015; Yip & Potenza, 2014), and other manipulations of the brain (e.g. transcranial magnetic stimulation, neurofeedback) are still matters of ongoing research (Goudriaan et al., 2014). Still, neuroscientific research has contributed to change the way we conceptualise GD, and such a change is having consequences on the way we deal with it.

Implications of conceptualising gambling disorder as an addictive disorder

Conceptualising GD as an addictive disorder implies endorsing the dissociation between wanting to gamble and liking gambling, and thus the view of gambling as economically inconsistent. Individuals with nicotine use disorder, for example, can invest considerable effort and money in purchasing tobacco *and* trying to quit smoking (Reith, 2007). The liking/wanting dissociation thus provides ethical ground for some degree of political paternalism. Given that *likes* also belong to individuals, consideration of likes beyond and above *wants* actually sanctions what has been called liberal paternalism (Camerer, 2006).

In other words, understanding the centrality of IS, its key role in the development of craving, and how loss-based learning fails to compensate it, justifies product- and offer-centred interventions regarding pervasiveness of gambling-triggering cues, and product design aimed at reducing features that enhance their addictive potential (Parke, Parke, & Blaszczynski 2016). And the other way round, if evidence does not support the consideration of a putative addictive disorder as a genuine one, there would be less ethical ground to justify intervention. If we adopt the same 'addiction' model for hypersexuality, dysregulated food intake or excessive video gaming, there would be no reason not to implement similar rules in those markets. Previous attempts to define addictive disorders based on the analogy between the excessive behaviour in question and a previously accepted addictive disorder, based on the application of DSM diagnostic criteria to the new putative 'addictions', has led to overdiagnosis and overtreatment. As stated by Billieux, Schimmenti, Khazaal, Maurage, and Heeren (2015), behavioural addiction research should shift "from a mere criteria-based approach toward an approach focusing on the psychological processes involved" (p. 119). As reviewed in this chapter, neuroscience definitely has a role in defining such processes.

Implications of a psychobiological approach to heterogeneity among patients with gambling disorder

Complementarily to the coexisting ways in which gamblers' heterogeneity has been approached to date, neuroscientific work can already provide a set of core dimensional constructs with practical use.

Individual treatments are likely to benefit from the reviewed evidence. First, in accordance with the Gambling Space Model, gambling motives should be assessed in order to draw a profile of the reinforcement sources that patients find in gambling, which could become targets of intervention. The identification of reinforcement sources linked to gambling could be useful to implement individual and process-oriented psychological interventions aimed, for instance, at developing skills to cope with high relapse-risk situations (anxiety, low mood, money-related thoughts, or boredom; Ledgerwood & Petry, 2006).

Second, gamblers with deficits in basic emotional dysregulation have been found as especially refractory to treatment attempts. For these cases, a better prospect is provided by studies in which mindfulness-based training has shown promising results in comorbid addictive and emotion disorders (Hoppes, 2006), and positive effects on decision-making neuropsychological tasks linked with basic emotion regulation (Alfonso, Caracuel, Delgado-Pastor, & Verdejo-García, 2011).

Third, intervening on planning executive functions is likely to benefit gamblers in the low end of the elaboration-self deception continuum (as it has been shown with SUDs patients, Verdejo-Garcia, 2016), whereas people in the high end would probably benefit more from metacognitive training skills aimed at making them aware of the connection between their dysfunctional beliefs and their motives to gamble (see Lindberg, Fernie, & Spada, 2011).

Complementarily, secondary prevention efforts in community populations could also be enriched with this dimensional-psychobiological vision, through the implementation of screening techniques aimed at identifying high-risk profiles (although we are aware that extra measures must be taken to avoid stigmatisation and stereotyping; O'Leary-Barrett et al., 2013). Neurobiologically-informed risk profiling has already gone a step further than traditional personality profiling, in delineating a common vulnerability factor for externalising problems in early adolescence, and dissociating it from other factors with differential loadings in separate disorders (Castellanos-Ryan et al., 2014). Prevention programs could thus be directed to individuals in general populations (not necessarily current gamblers) identified to have poorer basic emotion regulation. These individuals could benefit from interventions aimed at improving general emotional regulation and self-control, and thus see their risk of externalising problems, including disordered gambling, reduced.

Final remarks

The sociodemographic and behavioural map of gambling is changing rapidly. New gambling opportunities and media (e.g., mobile gambling) are generating new gambler profiles, so

understanding the mechanisms that generate the evolving variability of vulnerabilities, symptoms, and outcomes is necessary to be proactive at providing the best possible clinical and political response to eventually diminish the public health burden of disordered gambling.

As depicted in the current chapter, a combined psychobiological and behavioural-cognitive framework has shown some capacity to capture at least some of these sources of variability. The four proposed constructs are not necessarily exhaustive but are grounded in sufficient evidence to have clear implications for policy, prevention and psychological interventions. Still, further evidence should be gathered to help delineate or reconfigure this set of dimensions and evaluate its predictive power.

In parallel, it is important to acknowledge that theories of psychopathology have important, and potentially negative, consequences in real life. Biological approaches to psychopathology are often accused of crystallising abnormal behaviours that would be better understood as dynamically evolving and distributed in a continuum. As we have tried to illustrate here, psychobiological models can be learning-based and dimensional and, simultaneously, able to incorporate biological factors. At the same time, such models must be discriminative enough to allow identifying genuine addictive disorders. So, the risk of overpathologisation and psychiatrisation actually exists, in particular for many putative behavioural addictions. Probably, misleading and overinclusive definitions are already creating more harm than good.

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