Chapter 4
EFs in Pathological Gambling Disorder

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4.1 Introduction and Definition of Pathological Gambling Disorder

Gambling behaviour can be defined in different ways, such as "compulsive", "pathological", or "problematic" (Caretti & La Barbara, 2009). In the DSM-V (American Psychiatric Association, 2013) Gambling Disorder (GD) is placed in the "Substance-Related Disorders" section as "Non-Substance Related Disorder" and is referred to as "gambling disorder". It implies a significant compromise in family, work, and interpersonal life of the subjects. In order to be diagnosed, the person must present four or more of the following symptoms within a period of 1 year:

1. Needs to gamble with increasing amounts of money in order to achieve the desired excitement.
2. Is restless or irritable when attempting to cut down or stop gambling.
3. Has made repeated unsuccessful efforts to control, cut back, or stop gambling.
4. Is often preoccupied with gambling (e.g. having persistent thoughts of reliving past gambling experiences, handicapping, or planning the next venture, thinking of ways to get money with which to gamble).
5. Often gambles when feeling distressed (e.g. helpless, guilty, anxious, depressed).
6. After losing money gambling, often returns another day to get even ("chasing" one's losses).

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7. Lies to conceal the extent of involvement with gambling.
8. Has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling.
9. Relies on others to provide money to relieve desperate financial situations caused by gambling

Over the years, several authors have developed assessment and screening tools for the diagnosis of GD. Many of them have been developed by examining the diagnostic criteria of DSM (*Diagnostic and Statistical Manual of Mental disorder, DSM*). For example, Winters et al. (2002) have developed the *Diagnostic Interview for Gambling Schedule*, a 20-question interview to investigate the age of onset of gambling, symptoms, course and impairments in family, and interpersonal life (Winters et al., 2002).

From the point of view of symptoms, the psychological and physiological symptoms that occur in people with GD are very similar to the symptoms that can be found in people with substance addiction. Indeed, individuals with GD show symptoms of: abstinence, such as restlessness and irritability, when they are not gambling; craving, or the impressible desire for gambling behaviour; tolerance, when they need to play more and more in order to reach the desired pleasant effect; inability to control impulses, since subjects declare that they experience strong instincts to play and they are not able to resist them; and pervasive and constant thought of the game, which obscures the concentration of the subjects (Marazziti et al., 2015).

An important element to take into account is the impairment of family, work, and interpersonal life caused by gambling. In fact, individuals with GD tend to spend a lot of time playing and concentrating all their energies in the game, with the consequence of neglecting, reducing, or even interrupting other activities of daily life, such as work, family, or social ones (Rosenberg & Feder, 2014). Furthermore, a recurring problem in the life of gamblers is economic instability. Indeed, they often have financial problems caused by gambling, such as debt or bankruptcy.

In addition, GD presents several comorbidities with other addictions and psychopathological profiles: it has often been associated with a substance-related disorder. In this sense, it is important to consider that the use of psychoactive substances influences development and course of GD, as these substances have negative consequences on decision-making and impulsivity. In addition, gambling was found to be associated with other personality disorders, mood disorders, in particular depression and anxiety (American Psychiatric Association, 2013; Erbas & Buchner, 2012).

Over the years, several authors have tried to differentiate the subjects, applying terminological distinctions such as “pathological”, “problematic”, “social”, or “at risk” players. In particular, Custer (1984) identified six types of players: professional players, for whom gambling is not an addiction but a real job; antisocial gamblers, who play illegally; casual social players, for whom gambling is merely occasional entertainment; constant social players, who make gambling their main entertainment or leisure activity, but do not let it interfere with their family or work life; neurotic players without addiction syndrome, who use the game to soothe boredom, anxiety, depression; and compulsive gamblers, who have no control over their

own behaviour, cannot manage their impulses and continue to gamble despite negative repercussions on their family, work, and interpersonal life.

According to Custer, in this last type of player, a mechanism is established that he describes as "model of the career of the player" (Custer & Milt, 1985). In fact, they would go through three stages: a first phase of winning, in which they experience intense feelings of pleasure following the winnings of money; a second phase of loss, in which they begin to lose and therefore experience negative emotions due to the loss itself, thus trying to compensate by playing further; finally, a third phase of despair, in which the situation becomes increasingly serious to the point of causing a strong feeling of despair as a result of the different repercussions of the game on family and work life.

Finally, it was observed that, in pathological gamblers, it is possible to find neurobiological and neuropsychological alterations similar to those found in individuals with substance use disorder (SUD). In fact, several neuroimaging studies have been conducted that confirm the similarity between SUD and behavioural addictions, specifically GD. For example, a reduction in the activity of the ventromedial prefrontal cortex (vMPFC) was observed in individuals with GD during certain tasks, such as Stroop test or during the presentation of signals associated with gambling (Potenza et al., 2003b). As we have seen, the vMPFC plays a key role in the decision-making circuit and in risk assessment (Potenza, 2006). In addition, as regards subjects with GD, they show impairments in the performance of the Iowa Gambling Task (IGT) as much as individuals with SUD (Bechara, 2003). The IGT is used to analyse the effect of reward sensitivity and to identify predictive indicators of GD. In particular, the factors that influence the choices of the individual in the decision-making process are analysed, distinguishing between high and low risk decisions (Balconi et al., 2015, 2014a), as we will explore in the next paragraphs.

4.2 How Are EFs Involved in Pathological Gambling Disorder?

Because Executive Functions (EFs, for a definition see Chap. 1) deficits frequently underlie addictive behaviours (Hester & Garavan, 2004), it is essential to study potential EFs dysfunction in GD. This is especially important because EFs deficits may have also implications for the capacity of individuals to benefit from psychosocial treatments for GD (Leblond et al., 2003).

As we underlined in the previous chapters, EF involves higher-level cognitive processes implicated in the formation of successful goal-directed behaviour (Lezak et al., 2004), including planning and initiating behaviours, anticipating (positive and negative) consequences of actions, and the ability to adjust behaviours based on environmental feedback.

Specifically, planning, judgment, decision-making, set shifting, anticipation, and reasoning are the cognitive processes required for the successful completion of any

complex behavioural or cognitive task. Also required in this context are the suppression of unnecessary input and output, and the inhibition of inappropriate responses.

As we have previously observed, EFs were first described as central executive by Baddeley and Hitch (1974) and have been characterized by Lezak as the dimension of human behaviour that deals with how behaviour is expressed (Lezak, 1982). Therefore, the definition of "executive functions" includes a large umbrella of multiple processes [such as decision-making, response inhibition, conflict monitoring, cognitive flexibility, and their possible relationship with reward-related decision-making processes (Moccia et al., 2017)] and several, different definitions of EFs exist, which refer to different cognitive and neuropsychological models. Accordingly, in studying EFs in GD, authors referred to different models and adopted different tasks to analyse this family of functions. The problems of the absence of a homogenous definition of EFs and the large variety of tools used to assess them in the clinical population has already been underlined in some meta-analyses (see Kerns et al., 2008).

In this regard, studies have identified cognitive deficits in GD across a variety of domains (van Holst et al., 2010). Specifically, response suppression is indexed by stop-signal and Go/No-Go tasks, which require subjects to withhold simple motor responses when a stop-signal occurs (stop-signal tasks) or when a particular kind of stimulus is presented (Go/No-Go tasks). The ability to suppress responses is dependent on distributed neural circuitry, including the right inferior frontal gyrus and bilateral anterior cingulate cortices (Aron et al., 2004; Hampshire et al., 2010). The majority of studies have reported impaired response inhibition performance (i.e. increased motor impulsivity) in GD.

Several studies indicate a general trend towards EF impairment in GD. Specifically, GD performance in various neuropsychological tasks compared to non-GD revealed impairment in planning (Goudriaan et al., 2006b; Ledgerwood et al., 2012), cognitive flexibility (Goudriaan et al., 2006b; Odlaug et al., 2011), and behavioural inhibition (Goudriaan et al., 2006b; Grant et al., 2012; Kalechstein et al., 2007; Odlaug et al., 2011; Potenza et al., 2003b; Roca et al., 2008). Other studies found deficits in episodic and working memory, as well as verbal fluency in GD (Leiserson & Pihl, 2007; Roca et al., 2008; Zhou et al., 2016). Finally, performance on IGT, which was designed to assess decision-making capacity under ambiguity and risk, is impaired in GD (see Goudriaan et al., 2006b; Brevers et al., 2012b; Ledgerwood et al., 2012).

Brain imaging data appear to be consistent with these findings, revealing aberrant patterns of hemodynamic responses in prefrontal cortices in GD (for a review, see Grant et al., 2016). Given that the lateral prefrontal cortices have a central role in the neural substrate of EFs and working memory (Wager & Smith, 2003; Zakzanis et al., 2005), taken together this evidence points to a dysexecutive cognitive basis for GD, possibly attributed to lateral prefrontal dysfunction (for a review, see van Holst et al., 2010).

GD may experience significant deficits in EFs compared with non-GD, meaning that GD may be associated with significant comorbid neurological dysfunction in

many individuals with gambling tendency. This is clinically significant when considering appropriate treatment strategies for this population, as EF difficulties may hinder an individual's ability to benefit from treatment for GD (Ledgerwood et al., 2012).

As outlined previously, problems with cognitive functions dependent on cortico-subcortical circuitry have long been implicated in the manifestation of GD. Behaviours in people with GD are often repetitive, hard to suppress, and are impulsive in that they result in negative long-term outcomes. Furthermore, people with the disorder often have difficulty shifting their thoughts and behaviour away from gambling towards other areas of life that may be less damaging. Therefore, the study of Hinson et al. (2003) is particularly interested in two cognitive domains often reported to be deficient in patients compared with controls in the extant literature: response inhibition and cognitive flexibility. In prior cognitive studies, there has been a lack of clarity regarding whether deficits stemmed from the pathophysiology of recurrent gambling itself or rather reflected deficits that can pre-date symptoms and exist in people "at risk". In this study, authors attempted to address this issue in part by recruiting a group of subjects with "at-risk" gambling, viewed as being in an intermediate state between health and disease.

A second main and relevant factor that could be implicated in EF deficit in GD is the impulsivity control and related impaired behaviour. Many studies have found correlations between GD and behavioural and self-report measures of impulsivity. Specifically, impulse control is thought to be associated with underlying deficits in function in particular areas of the brain (e.g. prefrontal cortex) that are related to EF (Hinson et al., 2003).

Indeed, GD has been associated with impulsivity and attention deficit: GD patients were found to perform significantly worse than control subjects on attention measures and showed more childhood behaviours related to attention deficits (Rugle & Melamed, 1993). More recently, neuropsychological measures of impulsivity, such as the reaction time and number of errors at Go/No-Go tasks, as well as the scores at the Barratt Impulsiveness Scale, were higher in GD patients than healthy control subjects, while highlighting the importance of this dimension in the clinical picture of GD (Fuentes et al., 2006).

4.2.1 Brain Correlates of EF in GD Deficits

As we have underlined before (see Chaps. 1 and 2) prefrontal cortex (PFC)-dependent neurocognitive functions have been of particular interest in addiction research (Goldstein & Volkow, 2011).

Although the function of the PFC is highly integrated, two partially distinct PFC networks have been implicated in different aspects of neurocognitive function. The anterior cingulate cortex (ACC), lateral inferior cortex, and dorsolateral prefrontal cortex (DLPFC) have been linked to so-called "cool" EF, including working memory, response inhibition, task switching, and conflict monitoring (Badre &

D'Esposito, 2009; Koechlin et al., 2003), and the ventral, medial, and orbitofrontal structures (VMPFC, OFC) manage the so called "hot" EF, more involved in reward/emotion-related functions, including valuation, emotion regulation, and decision-making (Bechara & Van Der Linden, 2005; Peters & Büchel, 2010).

Also, GD patients may share a common dysfunction at the level of the vMPFC. In line with this hypothesis, a recent study using a comprehensive neuropsychological battery measuring EFs, demonstrated that GD and alcohol-dependent patients showed a reduction of executive functioning performance on inhibition, time estimation, cognitive flexibility and planning tasks (Goudriaan et al., 2006a, b).

The first neuroimaging studies in GD indicate that abnormalities exist in the vMPFC and cortico-basal ganglionic-thalamic circuits (Potenza et al., 2003a, b). Neuroimaging studies have shown that EF tasks activate a variety of areas within the prefrontal cortex (Coull et al., 2004) and, in addition to this, activate areas with important connections to the PFC, such as the caudate nucleus, the putamen, thalamic areas (Monchi et al., 2001), cingulate and parietal cortex (Van Den Heuvel et al., 2003).

The deficits in EFs as found in GD and SUD groups are therefore likely to be associated with dysfunctions and clusters of abnormal activation of these brain structures and brain circuits (for a recent review, see Moccia et al., 2017).

More recently, abnormal activity of the right Middle Frontal Gyrus (MFG), consistent with previous research (De Ruiter et al., 2009; van Holst et al., 2012a, b; Potenza et al., 2003a, b; Tanabe et al., 2007), and increased activity of the left dorsal ACC has been observed in GD (Quaglieri et al., 2020). The neural reward system encompasses both subcortical and cortical areas (including frontal lobes) and through the release of dopamine can stimulate food consumption, social reproduction, but also neural responses for "unnatural rewards" (such as monetary rewards), that contribute to compulsive behaviours like for instance gambling (the same occurs for substances) (Comings & Blum, 2000). Indeed, the striatum has been frequently reported to be involved in the expectation of monetary rewards (Crockford et al., 2005; Miedl et al., 2012; Power et al., 2012; Reuter et al., 2005): individuals with GD displayed greater activation in the bilateral dorsal striatum, related to stronger associations between the action and its outcome (van Holst et al., 2010), which could be accounted for by an overestimation of the gambling outcomes. The hyperactivity of dorsal striatum regions appears to be linked to a higher degree of reward-seeking behaviour, which could be a compensatory mechanism correlated to reward gaps in GD (van Holst et al., 2010); whereas the ventral part of the striatum appears to be more involved in the processing of the rewards (Miedl et al., 2012).

Regarding the involvement of the frontal lobe, the fronto-striatal cortical circuit is crucial for EF (Robbins, 2007), encompassing reward processing, control, and motor planning (Meng et al., 2014). When the clinical syndromes of GD are more severe, a hyperactivation of the striatum leads to impaired ability to control gambling behaviour. This impairment may contribute to fronto-striatal dysfunction in GD, with individuals showing deficits in self-regulation and higher degree of reward-seeking behaviour. The loss of control over gambling conduct is therefore due to an imbalance of the dopaminergic system and the neural circuits connecting

subcortical structures, such as basal ganglia and limbic areas and frontal regions (Moccia et al., 2017).

4.2.2 *Empirical Studies About Behavioural Deficits in GD: Measurement Evidence*

A recent study showed that patients affected by GD undergoing a battery of neurological tests, namely, the Wisconsin Card Sorting Test (WCST), the WMS-R (Wechsler Memory Scale revised) and the FAS (Verbal Associative Fluency Test), had sufficient or normal intellectual, linguistic, and visual-spatial abilities. As far as the WCST is concerned, GD patients showed qualitative but not quantitative deficits: in fact, although no differences were found between GD patients and healthy control subjects in the total number of categories completed, different abnormalities were detected at some subscales. As compared with healthy subjects, the thinking of GD patients appeared perseverant, because when they tried to resolve a problem while using an incorrect method, they tended to continue beyond that point at which other subjects would have looked for alternative solutions. A similar behaviour has been observed in GD patients at both the card-choosing tests (Goudriaan et al., 2006a, b) and the Go/No-Go task (Fuentes et al., 2006).

The difficulty that GD patients showed in learning from their mistakes and in redirecting themselves in the appropriate direction represents one of the most characteristic features of patients with alterations of the prefrontal lobe. This aspect has been observed in a significant number of experimental paradigms, in particular, patients with lesions of the prefrontal lobe are sometimes able to identify correct answers, while nevertheless still continuing to produce wrong answers (Drewe, 1975; Lurija & Homskaya, 1964). These findings are also compatible with other studies reporting worse performances in cognitive "risk-taking" tasks in patients with prefrontal lesions, as compared with healthy control subjects or patients with temporal lobe excision (Miller, 1992). In addition, these data would suggest a more generalized frontal lobe impairment. This is also supported by a recent study showing behavioural evidence of an alteration of both DLPFC and orbitofrontal cortex (OFC) in GD (Brand et al., 2005). However, it is still unclear whether the observed frontal lobe abnormalities should be considered a primary phenomenon linked to the aetiology of GD, or secondary to some symptomatologic features, or to the comorbid psychopathological conditions.

Flexible responding has traditionally been assessed with the WCST and its variants, which are dependent on distributed neural circuitry, including the ventromedial and ventrolateral prefrontal cortices (Buckley et al., 2009; Hampshire & Owen, 2006). Consequently, the majority of available studies have reported on WCST performance in GD compared with healthy controls.

Goudriaan and colleagues (2006a, b) concluded that comprehensive EF deficits were present in the GD group compared to normal controls. The deficits found in

EFs in the GD group could not be explained by deficits in basic cognitive functions, which are proposed as a prerequisite for performance of EF tasks. Also, their results indicate that the GD group resembled the alcohol dependence group, suggesting that comorbid symptoms had limited influence on EF performance.

While regarding the impairment of decision-making observed in GD might be explained by the inability to inhibit irrelevant information: in a recent study, the performances on the reverse Stroop task, which highly discriminates the ability to inhibit interferences, were significantly impaired in GD patients than in healthy subjects (Kertzman et al., 2006). Moreover, neurocognitive indicators of decision-making and disinhibition, such as the Card Playing Task and Stop Signal Reaction Time, respectively, seem to be powerful predictors of relapse in GD (Goudriaan et al., 2008).

4.2.3 Behavioural Addiction, GD, and Substance Addiction: What Kind of Brain Correlates Relationship?

The current state of knowledge from neuroscience studies suggests that there may exist a common pathological pathway between SUD and non-substance-related disorder (e.g. gambling or Internet gaming disorder), involving dysfunctional reward mechanisms and deficit in cognitive decisional processes (for an in-depth description, see Chap. 1). Previous studies observed that the neurobiological patterns of the addictive behaviours are similar: for instance, there is a reduction in dopamine (DA) receptor on compulsive feeding (Wang et al., 2002) and gambling related to deficits of the frontal cortex in GD (Potenza, 2008).

Many of the features central to GD are similar to those of SUD and implicate common underlying dysregulation of frontostriatal circuitry (Clark, 2010; Grant et al., 2010). Notable features that share commonality between GD and addiction include persistent engagement in a behaviour despite negative consequences, loss of self-control, compulsive engagement (“drive”), craving, tolerance, and withdrawal (Potenza, 2008). As such, GD represents a valuable model for studying the neurobiology of addiction, without the potential confounding pernicious brain effects from chronic alcohol or illicit substance abuse.

Apart from the diagnostic similarities that GD shares with SUD and Impulse Control Disorders (ICDs), these disorders are all characterized by behavioural deficits in self-regulation, as manifested in an impaired ability to inhibit the urge for the desired behaviour or drug. Deficits in EFs are proposed as important mediators in drug bingeing (Goldstein & Volkow, 2002), and several studies suggest that impairments in EFs have a negative impact on treatment success and relapse in substance dependence (Bates et al., 2004; Fals-Stewart & Schafer, 1992).

4.2.4 Some Limits in EFs Studies Applied to GD

Despite the relevance of EFs in GD, research in this field is still scarce and findings are inconsistent. In addition, most studies did not investigate whether deficits in EFs were independent of deficits in basic cognitive functions. A closer look at the literature reveals a number of potential weaknesses in this notion. Firstly, there is evidence against a generalized EF impairment in GD (Manning et al., 2013). Secondly, several studies have a number of methodological limitations. The most important reason for these inconsistencies concerns the fact that some studies targeted only a single EF, most studies were restricted to small groups and studies often failed to assess and control for comorbid disorders and medication use. In addition, the specificity of EF deficits in GD is not known, because clinical comparison groups were not included in most of these studies. Sampling bias, mainly due to inclusion of treatment-seeking patients only, may provide non-representative groups (Lorains et al., 2011). Additionally, it has been argued that the majority of GD seek treatment for a co-morbid disorder rather than gambling per se (Winters & Kushner, 2003). Moreover, small sample size prevents the use of parametric statistics and limits generalizability of results. Finally, a large proportion of the relevant studies lack a thorough neuropsychological assessment, thus drawing conclusions on the basis of limited data.

The above limitations stress the need for further studies utilizing comprehensive cognitive batteries on representative, unbiased, ecological samples of individuals with GD.

4.3 Theoretical Models to Explain SUD and GD

Some recent neurocognitive models were introduced to explain drug dependence. However, they can be applied and extended also to GD, based on previous evidence on both behavioural deficits and neurocognitive correlates. We summarize some main directions of these models in the following paragraphs.

4.3.1 Aberrant Learning Theory

Chronic drug exposure leads to long-term associative memory processes occurring in several neural circuits that receive input from midbrain DA neurons (reward learning). Specifically, cues predict-rewards can strongly activate NAcc related circuitry in both animals and humans even better than the reward itself (Schultz, 1998). It was argued that explicit learning (declarative memory) could reinforce the addiction: usually people who take drugs since the first time learn, at conscious level, predictive relationships between some cues in the environment and rewards.

Abnormally strong explicit learning might distort declarative memories or expectations; such addicts make inaccurate predictions about the consequences of taking drugs. Even so, drugs cause strong implicit learning which is not directly accessible to conscious. *The Stimulus-Response (S-R) habit learning hypothesis* (Everitt & Robbins, 2005) proposed that the progression to addiction involves at first controlled behaviour by explicit and cognitive expectations about Act-Outcome relationships (memory of drug pleasure), and then occurs the automatic behaviour consisting of Stimulus-Response habits. Although habits are not intrinsically compulsive, the addiction is due to the development of very strong S-R habits. Considering the neural system of reinforcement for addiction, the changing from voluntary drug use to habitual and compulsive abuse represents a transition from PFC to striatal control, involving its dopaminergic innervation.

A similar explicative approach describing the transition from voluntary gambling behaviour to pathological and compulsive behaviour may be adopted for GD (Brevers & Noël, 2013). In this case, on the one hand, there are some structural factors of gambling games that could promote the repetition of gambling behaviour to the point that in some people it could lead to a dysfunction of controlling gambling conduct. On the other hand, there are three crucial neural systems whose dysfunction may lead to an impairment in controlling gambling conduct, and that will be described in the following paragraphs. Starting from the structural peculiarities of gambling behaviour, authors underlined that there are at least two properties of gambling that promote the repetition of playing behaviour: they are (a) the intermittent schedule for reward and loss, and (b) the illusion of control over the game (Brevers & Noël, 2013).

4.3.1.1 The Intermittent Schedule for Reward and Loss

Gambling is characterized by irregular wins and losses delivered on a variable ratio, which entails imperfect reward estimation. This may be one behavioural reason for why gamblers engage in gambling despite growing losses (Schultz, 2002). In fact, in previous studies, it has been demonstrated that behaviours learned after a primary learning phase featured by intermittent rewards are carried over time and far more resistant to extinction than conducts learned under continuous rewards (individuals stop the activity when it is no longer rewarded) (Schultz et al., 2003). Hogarth and Villeval (2010), for example, found that participants in the continuous-reward-schedule condition leave as soon as payment stops, while irregular monetary incentive schedules result in greater conduct persistence displayed by the participants at the end of the payment phase.

In line with the *Reward Prediction Error Models of Learning* (Montague et al., 1996; Schultz et al., 1993), a behaviour learned under intermittent reward learning requires imperfect reward prediction and it is much more resistant to extinction. According to the model, rewarding events that entail a better result than predicted (i.e. a positive reward prediction error) produce highly positive emotional activations, and these feelings remained stable if followed by a good prediction, and/or may

vary and be diminished by a reward that is worse than predicted (Schultz et al., 2003). Also, the release of dopamine co-varied according to the uncertainty of the reward, with higher amount of release for rewards with maximal uncertainty (Fiorillo et al., 2003). Therefore, when the roulette wheel spins and players win some money during gambling, they can experience a powerful emotional positive state, because the reward was so unpredictable or unforeseen.

4.3.1.2 Illusory Perceived Control

The second structural property of games supporting gambling behaviour consists of players option of arranging their own wagers (like picking a number at the lottery or selecting a colour at the roulette), which can boost players' belief that he/she could win (Ladouceur & Sévigny, 2005). The term adopted to describe this mechanism is "illusion of control", since none of the actions cited above have an effect on the probability of winning, and it has been described also in diagnostic manuals as a peculiarity of GD (American Psychiatric Association, 2013).

4.3.2 The Triadic Neurocognitive Model

As previously mentioned, a recent neurocognitive theoretical model includes gambling structural features in a more complete and exhaustive view (Brevers & Noël, 2013). Indeed, in addition to gambling games' characteristics, the model posits there are three crucial neural systems whose dysfunction may lead to an impairment in controlling gambling conduct:

- *A hyperactivation of an "impulsive" system* that is immediate, unaware, and unconscious and promotes automatic and repetitive actions.
- *A hypoactivation "reflective" system* that is slow and deliberative, predicting the potential implications of a behaviour, response inhibition, and metacognition.
- *The interoceptive system*, which transforms bottom-up bodily sensations into a subjective state of craving, accordingly, boosting the impulsive system, and/or weakening the normal functioning of the reflective system.

We distinctly consider these three neural systems and their implications in gambling behaviour.

4.3.2.1 The Hypersensitization Toward Gambling-Related Stimuli and the "Impulsive System"

Firstly, the authors try to answer to the following question: "how is it possible that individuals keep gambling despite growing monetary losses?" Authors advanced the hypothesis of a hypersensitization toward gambling-related stimuli and actions,

that is in line with the Incentive Sensitization Theory developed for SUD (Robinson & Berridge, 2003). Over time, gambling-related cue can activate disruptive motivational states, able to hinder high-order cognitive and affective systems adopted for controlling the behaviour and preventing the person from addiction-related conducts (Verdejo-Garcia & Bechara, 2009).

Through classical conditioning processes, the repeated gambling experience may promote the formation of associative learnings between gambling-related cues, the positive emotions derived from wins and gains, and the behavioural actions of gambling (Hofmann et al., 2009). These learned associations can be easily re-activated when the individual is confronted with gambling related cues, in the sense that his/her brain-body system is able to answer immediately to these attractive and salient stimuli, based on previous learning experiences, and may in a suitable way trigger the positive emotions and the behaviours linked to gambling (Hofmann et al., 2008, 2009). As for SUD, even gambling-related stimuli (considered as "unnatural rewards") may promote these quick and implicit activations (both at the memory and emotional level) and capture the attention of individuals with GD, leading to the so-called "attentional bias" (Robbins & Ehrman, 2004).

4.3.2.2 The Disruption of the Reflective Function

Although impulsive processes and hyperactivation toward gambling stimuli may explain individuals with addiction incentive to look for rewarding cues, it does not appear to explain the deficit in individual's capability to control the impulsive and immediate tendency to gamble, to implement a more functional and long-term goal-directed behaviour, a function that is mainly operated by the so called "reflexive system".

The integrity of the two following sets of neural systems is needed for the reflexive system to function: the "cool" and "hot" EF systems (previously described in Sect. 4.2.1). Also, successful decision-making represents the convergence of these two cognitive and affective processes, which results in the ability to optimally balance short-term benefits against long-term losses, or to predict the possible consequences of a given decision (Damasio et al., 1996). In contrast to the "impulsive" system, the functions of the reflexive system are managed through comparatively slow, monitored, conscious, aware, and self-regulated processes (Smith & DeCoster, 2000).

An impairment in "hot" EF could have an impact mainly in decision-making situations in which emotion regulation is involved, since there is no information related to reward probability (i.e. decision-making under ambiguity; Brand et al., 2006; Krain et al., 2006). In these conditions, previous associative memories of win or losses must be recalled foreseeing both short- and long-term positive or negative outcomes of any given option (Bechara, 2004) and an impairment of this ability in GD will be extensively described below.

Additionally, regarding the disruption in "cool" executive functioning, recent research on excessive gambling indicates that the capability to inhibit unconscious

immediate responses could be the critical element in the development and maintenance of gambling addiction. Indeed, impaired inhibitory control has been associated with the onset of addiction by exacerbating problem gambling (Brevers et al., 2012a) and sabotaging gambling withdrawal (Goudriaan et al., 2008).

4.3.2.3 The Role of Interoceptive Processes: Halfway Between Impulsive and Reflective Systems

As third system of the model, Brevers and Noël (2013) included the interoceptive system, as a halfway system that may play a role in the onset and maintenance of addiction by transforming bodily signals into feelings of desire, anticipation, or urge (Goldstein et al., 2009; Goldstein & Volkow, 2011). At the neural level, the area that mainly processes the interoceptive signals is the insular cortex (Craig, 2009). For further information on interoception and addiction, see also Chap. 9.

Furthermore, some recent theoretical discussions (Goldstein et al., 2009; Goldstein & Volkow, 2011) propose that the inability to grasp the interoceptive signals can affect the metacognitive capacity (i.e. the ability to reflect on one's own actions and thoughts, but also to assess one's own performance at the behavioural level, discriminating its success or failure (Cleeremans et al., 2007); for this concept, see also Chap. 1) in an individual with addictions. The deficiency of metacognitive capability in addicts has been well documented and it is extremely relevant for the clinical relapses, since the individual fails to understand the seriousness of the condition (Goldstein et al., 2009). The underestimation of addiction severity and a disconnection between self-perception and actual behaviour have been detected in different categories of substance users (cocaine, nicotine, methamphetamine, and cannabis users) (Chiu et al., 2008; Hester et al., 2009; Moeller et al., 2010; Payer et al., 2011); as well as GD (Brevers et al., 2013; Brevers & Noël, 2013).

4.3.3 Frontocortical Dysfunction Theory

A more neurocognitive model posits that the cortical impairment may strongly support the cognitive function impairment in both drug addiction and GD (Quaglieri et al., 2020). Chronic exposure to drugs can modify neural processing in frontal regions and distort functions of the PFC (Volkow et al., 2013). Dysfunctional changes in fronto-cortical activity have been described during intoxication for many of the drugs and in polysubstance abusers and a decrease of the volume of the PFC was also found in these populations (Volkow et al., 2013). Evidence show that fronto-striatal projections are important in regulating emotions and providing inhibitory control behaviour (Davidson et al., 2000). Furthermore, neurobiological studies report that some addicts show a variety of neuropsychological deficits shared with patients with frontal dysfunction (Bechara et al., 2000), such as deficit in decision-making (Verdejo-García & Pérez-García, 2008). It is widely accepted that PFC

is an important contributor to decision-making, assignment of value, and to maintenance of goal-directed behaviours (inhibitory control).

In our recent study, we focused on the metacognitive representation in Cocaine Addicts (CA) about the strategies they used during the IGT decision-making task (Balconi et al., 2014d). The IGT (Bechara et al., 1994) is a sensitive measure of decisional processing that simulates a real-world decision-making situation under uncertain conditions, and it implies some factors like: immediate rewards, delayed punishments, risk and uncertainty of outcomes. In the IGT, participants are instructed to try to gain as much money as possible by drawing selections from a choice of four decks; two of the decks are disadvantageous (DD), because they produce immediate large rewards and also significant money loss; the other two decks are advantageous (AD), because rewards and punishments produced are lower. In general, insensitivity to punishment, together with a strong reward dependence, results in a disadvantageous pattern of decision-making, and more reward-dependent individuals should make more risky and disadvantageous choice (Balconi et al., 2014b, d). Data showed different behavioural options and opposite strategies on the IGT comparing CA and healthy subjects: addicts demonstrated a more dysfunctional behaviour in their choice of strategy; moreover, they were unable to evaluate and reconstruct a realistic thinking about the cognitive strategy they adopted during the IGT performance (Balconi et al., 2014d).

It is widely accepted that the frontal lobes are involved in cognitive and metacognitive functions, and also the OFC and VMPFC, which are part of PFC, are networked with the amygdala, dorsal striatum, NAcc, hypothalamus, and insula. Thus, it has been hypothesized that addictive drugs produce a distorted and excessive DA signal in the OFC and other regions of the PFC, and this excessive DA signal can produce overlearning of drug-related cues. In general, impairments in executive function and increased impulsivity have been correlated with the diminished ability to recruit high cognitive functions of the PFC in drug abusers. Thus, pathological over-evaluation of drug related cues and impairment of some functions of top-down control could make significant deficits, such as loss of control and absence of coherent meta-representation about their own strategy in decisional making processes in addiction.

4.3.4 The Cortical Unbalance Model and Lateralization Effect

Previous neuroscientific literature demonstrated an association between addiction and the abnormal functioning of neural systems supporting motivation and reward processing.

As previously underlined in Chap. 1, the development of a problematic addiction disease (related or non-related to substances) has been mainly linked to deficit in reward pathways, neurocognitive deficits, attribution of value to salient stimuli (Balconi et al., 2014b, d; Bechara, 2005; Goldstein & Volkow, 2002), neural changes in memory structures (Volkow et al., 2003), and impaired metacognitive processes (Balconi et al., 2014b, d; Goldstein et al., 2009). Regarding SUD, previous works

indicated that addiction to substance is linked to the salient properties of drugs, which are strictly connected to a rewarding effect (Balconi et al., 2014b, d).

One of the main characteristics of SUD and behavioural addiction is the dysfunctional preference for instant gratification (i.e. reward) rather than a delayed gratification, which is observable in behaviours characterized by impulsivity. Several fMRI studies supported this dysfunctional process displaying higher amygdala activation to addiction-related cues (Volkow et al., 2013). For this reason, individuals with addiction have been compared to patients with VMPFC damage, highlighting how both clinical categories are characterized by insensitivity to future consequences (Bechara, 2005): in fact, as previously mentioned, they display the so-called "myopia for the future", being mainly compelled in obtaining a short-term gain, and unconscious of long-term beneficial or adverse outcomes (Balconi et al., 2014a, b). This aspect has been extensively studied by adopting decision-making tasks, such as the IGT. Interestingly, the repetitive use of substances and problematic gambling could also induce individuals not previously displaying deficit in decision-making, to develop an impairment in evaluating the long-term adverse consequences of their actions and prefer short-term rewards for having relief from the negative mood.

4.4 Behavioural Study and EFs in GD

As mentioned in several points in the chapter, the IGT is one of the most used behavioural tasks for assessing decision-making deficits in multiple categories of patients, from patients with frontal lesions to SUD individuals, to patients with GD. Previous studies demonstrated that GD-impaired performance at the IGT task is comparable to that of individuals with SUD (Goudriaan et al., 2006b).

A more recent work sought to classify decision-making deficits in GDs and investigate distinct features in two types of decision-making; under uncertainty and under risk, with two different versions of the IGT (Ochoa et al., 2013). As key findings, the authors indicated that the majority of GDs had general decision-making deficiencies, which were characterized by myopia for the future rather than aversion to punishment. Also, GDs mainly showed abnormal choice behaviour in relation to decisions made under risk on the IGT (linked to the explicit understanding of the task, EF, control processes, and impulsiveness) more than decision-making under ambiguity. It is worth noting that the authors highlighted that different pattern of deficits are involved in GD decision-making processes, and the predictors vary depending on the reinforcement schedule (Ochoa et al., 2013).

Moreover, basic research studies on the IGT demonstrated that decision-making under ambiguity features the first phases (trials) of the task, when the understanding of the rules is less explicit to the subjects (and the game depends primarily on emotional feedback processing), while decision-making under risk characterizes the final phases of the task, when the rules become more explicit (and the game relates with other complex mechanisms of EFs, such as categorization, task monitoring, and cognitive flexibility) (Brand et al., 2007).

Therefore, despite Bechara (2001) claiming that to obtain a good performance on this task, individuals should listen to and follow their feelings and intuitions (in line with *Somatic Marker Hypothesis*), we agree with previous studies stating somatic signals are essential for decision-making processes, but the integrity of the cognitive processes also depends on EFs (Brand et al., 2007).

Overall, findings described above suggest the need for specific clinical approaches based on learning techniques to support people to deal with decreased inhibitory control and impaired decision-making ability (Goudriaan et al., 2008). For treating GDs effectively, it has been also suggested that interventions should include methods for identifying the impulsive reaction before acting, in order to support them in reflecting on the long-term consequences of their actions, to control their behaviour, and to find possible alternative solutions (Álvarez-Moya et al., 2011).

4.4.1 Reward Sensitivity and IGT

Theory and past research using monetary incentive tasks, such as IGT, suggest that individuals' sensitivity to reward and loss plays a role in their ability to anticipate positive versus negative consequences that may result from their actions (Bjork et al., 2004).

As we know, and we already described in Chap. 1 (Sect. 1.6.2 on reward mechanisms in behavioural addiction) in the IGT, participants choose from four decks of cards across 50 trials, with the goal of acquiring as much money as possible. Decks vary in both the magnitude and frequency of rewards and losses. As such, the task can be used both to assess sensitivity to reward as well as sensitivity to loss. Importantly, the IGT is sufficiently complex that participants are unable to calculate the net gains and losses that each deck affords (Damasio et al., 1996). Rather, according to the hypothesis of somatic markers, participants must rely on covertly and overtly occurring marker signals to sense which decks are good, and which are bad, with correspondingly better versus worse likely future outcomes. For example, one study found that healthy subjects exhibited a Skin Conductance Response (SCR) prior to selecting a card from a bad deck, whereas patients with ventromedial frontal damage, who typically perform poorly on the task, did not (Bechara et al., 1996). Poor performance on the task is hypothesized to indicate individuals' less effective cue detection of these marker signals regarding possible future outcomes, which in turn may affect real-time decision-making.

Healthy participants will learn which decks are advantageous and will select more often from these decks, while patients with VMPFC lesions will persist in selecting from the DD that provide a large immediate reward (Bechara et al., 1996, 1997). More interestingly, healthy comparisons showed anticipatory SCRs when they choose decks, and the SCRs were higher when choosing disadvantageous decks; however, the VMPFC patients did not show the same anticipatory SCRs (Bechara et al., 1996, 1997).

Based on the studies in VMPFC patients (e.g. Damasio et al., 1991; Damasio, 1994), Damasio proposed the famous Somatic Marker Hypothesis: he argued that these patients had decision-making deficits because they were not able to use somatic markers to guide their decision-making. The somatic markers are body-generated, emotion-based signals (see also Dunn et al., 2006).

However, there are several limitations of the SCR studies. First, in the psychophysiology analysis, the deck that participants selected at last was used to designate each anticipatory "somatic marker"; however, in the deck selection phase, participants were free to shift their attention across all decks prior to selecting one. This procedure meant that the anticipatory SCRs may not reflect attention to a single card but shifting attention across all decks before making a choice (Dunn et al., 2006). Second, a study using the IGT in rhesus monkeys showed that SCRs were associated with the anticipation of a reward after a decision had been made rather than reflecting the decision-making process directly (Amiez et al., 2003). Thus, due to the low temporal resolution of SCRs, it was difficult to separate the signal related to response selection from the anticipation of feedback after the response (Dunn et al., 2006). One solution is to use other psychophysiological responses with a faster time course, such as Event-Related Potentials (ERPs).

4.5 Electrophysiology of Pathological Gambling behaviour

4.5.1 ERP Evidence for GD

To examine the electrophysiological correlates of GD, some research has explored widely-known ERPs, which have been documented to mark brain activity variations associated with selective attention and inhibition (for a review see, Luijten et al., 2014).

Some specific deflections were studied, mediating different cognitive processes. Two main ERP components have been reported to reflect changes in brain activity related to inhibitory control (Kok et al., 2004). Specifically, accumulating evidence suggests that the N2 and P3 reflect functionally distinct processes associated with inhibitory control. Accordingly, less pronounced N2 or P3 amplitudes in addicted populations relative to controls can be considered markers for neural deficits in inhibitory control.

4.5.1.1 N200

The first component, the N200 (or N2), is a negative-going wave emerging 200–300 ms after stimulus presentation. The neural generators of the N2 appear in the ACC (Huster et al., 2010; Nieuwenhuis & Yeung, 2003) and the right inferior frontal gyrus (IFG) (Lavric et al., 2004). The N2 is believed to index a top–down mechanism needed to inhibit the automatic tendency to respond (Falkenstein, 2006;

Kaiser et al., 2006) and corresponds to behavioural outcomes of inhibitory control (Dimoska et al., 2006; Falkenstein et al., 1999; Van Boxtel et al., 2001). The N2 has further been associated with conflict detection during early stages of the inhibition process (Falkenstein, 2006; Nieuwenhuis & Yeung, 2003). Consequently, the N2 can be interpreted as an index for early cognitive processes necessary to implement inhibitory control rather than the actual inhibitory brake.

ERP findings in behavioural addicted individuals (excessive Internet users) showed reduced N2 amplitudes, suggesting a deficit in the conflict detection stage of the inhibition process. In contrast, N2 amplitudes in people with excessive gaming behaviour were enhanced in a parietal cluster (Luijten et al., 2014).

To go into more detail, various and different N2 subcomponents have been reported according to the generation sites, the experimental tasks, and the underlying cognitive process (Patel & Azzam, 2005): the N2a is mainly generated in frontal sites by conscious attention to an oddball stimulus; the N2b is mainly evoked in central sites and is related to conscious stimulus attention; the N2c arises in frontal and central regions, in relation to classification tasks; finally, the N2pc, with a posterior distribution, is evoked during visual perceptual tasks involving the discrimination of a featured target showed in a field with distractors, it is an indicator of attentional selectivity (Treisman & Sato, 1990).

4.5.1.2 P300

The P3, the second ERP component involved in inhibitory control, is a positive-going wave emerging 300–500 ms after stimulus onset. The source of the P3 has been found to be close to motor and premotor cortices (Ramautar et al., 2006). Hence, P3 amplitudes appear to reflect a later stage of the inhibitory process closely related to the actual inhibition of the motor system in the premotor cortex (Band & Van Boxtel, 1999).

Some studies show that the reduced amplitude of P3 may be an indicator of the neurobiological vulnerability underlying disorders such as addictions (Patrick et al., 2006). In this regard, a recent study found a neural index underlying the response inhibition difference between individuals with Internet Addiction Disorder (IAD) and a control group by using an ERP technique (Dong et al., 2010). As discussed above, N2 is believed to be related to the process of conflict monitoring, and P3 to response evaluation: these two mental processes are fundamental abilities in the impulse inhibition process, and these two ERPs are frequently examined together in electrophysiological studies. Internet-addicted participants were expected to show some difference in N2 and P3 compared with their normal peers. Indeed, significant difference was found between IAD and normal groups in No-Go condition, the IAD group elicited significant lower N2 mean amplitude than normal group. The difference was largest at the central sites, as compared with frontal sites and parietal sites. In addition, the peak latencies in No-Go conditions were significantly longer than Go conditions in both IAD group and normal group.

Further analysis between groups showed that IAD group showed significantly higher P3 amplitude than normal group in No-Go items. In peak latencies of P3, IAD group elicited significantly longer P3 latency than normal group in No-Go condition, but no significant difference was found in Go condition. Thus, the size of P3 amplitudes in the present experiment might reflect the degree of cognitive endeavours when the participants successfully inhibited their impulse to respond. The IAD group elicited higher P3 amplitude than the normal group, and this evidence was interpreted as the need for more cognitive endeavours for behavioural addicted participants to successfully inhibit their response impulses. The NoGo-P3 latency was longer in IAD-afflicted participants compared with that of normal subjects. Peak latency is associated with cognitive efficiency. P3 latency is an indicator of processing speed suggesting that IAD had less efficient information processing function than their normal peers (McEvoy et al., 2001; Polich & Criado, 2006). On the other hand, the longer P3 amplitude may be related to impaired impulse control: evidence from studies on impaired inhibitory ability shows that individuals with Post Traumatic Stress Disorder and Parkinson's disease have longer NoGo-P3 latency compared with control groups (Bokura et al., 2005; Shucard et al., 2008). In summary, IAD participants displayed less efficient brain function not only with respect to information processing, but also response inhibition. Taking all features of N2 and P3 components into consideration, we can comprehensively understand impulse control in the IAD individuals.

In other studies, reduced P3 amplitudes to rewarding stimuli have been found for frequent gamblers compared to non-gamblers (Oberg et al., 2011), and in individuals with SUD (Goldstein et al., 2008). It is also of value to confirm whether problem gamblers abnormally process the significance of positive outcomes. A recent study revealed that the P3b subcomponent is likely to be driving the observed valence differences in global P3 amplitude (Lole et al., 2013). From this point on, references to the P3 will relate to the traditionally conceptualized global P3 component that comprises various subcomponents, including the P3a/novelty P3, P3b, and Slow wave, and it will be identified by its topography, latency, and experimental determinants.

4.5.1.3 ERN and FRN

The examination of the feedback-related negativity (FRN) ERP component was also considered a relevant effect in GD. Similar to the error-related negativity (ERN) that is elicited by commission errors in reaction time tasks (Falkenstein et al., 1999; Gehring & Willoughby, 2002; Miltner et al., 1997), the FRN provides insight into how feedback on reward and non-reward/punishment outcomes are evaluated in the brain. This component has been consistently shown to be sensitive to valence and context manipulations. Specifically, larger FRN magnitudes are observed when feedback signals monetary loss compared to gain (San Martín et al., 2010; Toyomaki & Murohashi, 2005; Yeung et al., 2005) or the least desired outcome within a particular context (Holroyd et al., 2004) during tasks that resemble gambling

activity. The reinforcement learning theory (Holroyd & Coles, 2002) postulates that the ERN and FRN reflect the activity of a high-level error-processing system within the mesolimbic–dopaminergic pathway, a system believed to be involved in the evaluation of environmental stimuli, the activation of motivated behaviours, and association formation.

Little and colleagues' (2012) study showed increased error rates for No-Go trials in people with excessive gaming behaviour compared with controls (Littel et al., 2012). Lower ERN amplitudes were found in participants with excessive gaming for error trials, suggesting that initial error processing in excessive gamers may be less pronounced than in controls, whereas error awareness may not be related to increased error rates.

Our recent research explored the main factors able to influence the subjects' choices in the case of decisions and distinguish between high- and low-risk decisions. Behavioural responses at the IGT, meta-cognitive strategy, and two ERP (FRN and P3) effects were used as predictive markers of gambling behaviour. Behavioural activation system (BAS) reward measure was applied to distinguish between participants with high-BAS and low-BAS levels. It was found that higher-BAS participants opted in favour of the immediate reward, with a concomitant dysfunctional metacognition of their strategy: a consistent "reward bias" affected the high-BAS performance reducing the P3 and FRN in response to unexpected (loss) events.

Regarding the EFs and metacognition, it was shown that impaired working memory can lead to poor decision-making capacity, with a consequential inability to plan the best long-term strategy, to inhibit the immediate reward-seeking, and to organize a functional behavioural response (Bechara & Martin, 2004; Verdejo-Garcia & Bechara, 2009). In particular, these functions under uncertain conditions, flexibility, and adaptation in behaviour were required to preserve the processing of consequences of previous decisions and actions (Perry et al., 2011). Recently, some research contributed to clarify the role of cognition and metacognition in gambling behaviour, and some specific ERP effects, such as the FRN and P3 effect, were considered the neurocognitive correlates of decisional behaviour in case of both functional and dysfunctional conditions.

The first ERP effect related to FRN is involved in performance monitoring, and it was observed that it is probably cortically generated near the MFC, mainly the ACC (Hewig et al., 2007). In addition, processing underlying the FRN are triggered by phasic dopaminergic signals, which code reward prediction error. These prediction error signals may then be conveyed to the ACC where they lead to adjustments in subsequent action selection and FRN production as an ERP effect (Holroyd & Coles, 2002).

A second relevant ERP deflection, the P3, was used to explore the impairment of the EFs in decisional processes (i.e. the difficulty in updating the incoming contextual information.) The P3 is the ERP component commonly investigated during feedback processing; it has been shown to be sensitive to the significance and occurrence probability of a stimulus (Hajcak et al., 2005; Oberg et al., 2011) as well as task complexity (Duncan-Johnson & Donchin, 1977). The increasing amplitude

of this positive deflection might represent the necessity to restore adjunctive information to updating the context (Balconi & Crivelli, 2010; Isreal et al., 1980; Johnson & Donchin, 1980) when an unattended event is observed. Thus, it was found that more unexpected outcomes (as in case of losses) generated an increased P3 in comparison with more expected (gains) outcomes.

Therefore, when considered together, these two ERP measures could signal the increased inability to adopt an adequate cognitive strategy in response to a decisional context.

4.5.2 EEG and Lateralization Effect

In line with the reward and lateralization model (for this concept, see also Chap. 1, Sect. 1.6.3 on the cortical unbalance model), we propose that a similar cortical left "unbalance" could be suggested in GD as for SUD.

Previous research works based on Gray's BIS/BAS model (Gable et al., 2000), indicated that behavioural motivational responses related to personality characteristics are essential for two main aspects: for generating emotions, and approach (reward) and withdrawal (inhibition) behaviours in the decisional process (Gray, 1981; Yu & Dayan, 2005). With respect to reward mechanisms, the BIS/BAS scale is a valuable instrument for evaluating possible anomalous reward sensitivity in neuropsychiatric populations, such as addictions, relative to healthy subjects (Gray, 1981; Gray & Naughton, 1987; Yu & Dayan, 2005). It permits to quantify the prevalence of BIS or BAS in individuals. As we have seen, the BAS motivational component has been conceived as a mechanism sensitive to compensation, incentive stimuli, reward, and non-punishment, involving actions directed towards a gain and away from a loss (Gray & Naughton, 1987).

Therefore, approach behaviour is promoted by reward, which induces a positive reinforcement for action, whereas avoidance behaviour (withdrawal) is reinforced by punishment. A normal level of BAS has a functional influence on positive emotional attitudes, while severe BAS and reward sensitivity levels have been related with impulsivity disorders (Fowles, 2000), and high levels of BIS have been associated with anxiety disorders (Balconi et al., 2014c; Balconi & Mazza, 2009; Yu & Dayan, 2005).

A crucial aspect of the BIS/BAS system (as previously explained in Chap. 1) is its cortical correlation with the PFC structures: while the left PFC activity was shown to be involved in approach-related motivations (appetitive) and positive emotions (reward processing), it was found that the right PFC activity was involved in withdrawal-related motivations (aversive) and negative emotions (punishment) (Gray, 1987; Quay, 1998).

Former studies showed that individuals with SUD, GD, or high-level of BAS reward sensitivity exhibited substantially more risky decision-making, preferring a greater possible reward even at a higher penalty risk. In addition, in these populations, their electroencephalographic behaviour showed a left PFC (DLPFC and ACC)

frontal hemispheric activation asymmetry found at the electrophysiological level, suggesting an enhanced sensitivity to more risky choices (Gray, 1981; Yu & Dayan, 2005).

A recent line of research investigated gambling tendency in a group of individuals with high-BAS scores and found that, in comparison with low-BAS, the high-BAS group showed an increased tendency to opt in favour of the immediate reward (losing strategy) instead of the long-term option (winning strategy), and members of this group were more impaired in metacognitive monitoring of their strategies and showed an increased left hemisphere activation when they responded to losing choices. A "reward bias" effect was hypothesized to act for high BAS, based on a left hemisphere hyperactivation (Balconi et al., 2015, 2014c; Finocchiaro & Balconi, 2015, 2017).

An earlier EEG study by Goldstein and Carlton (1988) studied lateralization of EEG activity in eight pathological gamblers and eight normal controls, matched for age and socio-economic status. The authors hypothesized that GD is associated with compulsiveness, and therefore expected difficulty switching between behaviours in GD. Therefore, they investigated switching between hemispheric activities, by employing tasks that typically involve left or right hemispheric activity. In the GD group, no significant shifts in right or left hemispheric activation existed, while in normal controls, these shifts were present. Furthermore, it took the GD group significantly longer to activate either left or right hemisphere. This last finding could have influenced the lack of lateralization differences, since less data with lateralized activation in the GD group was available. A possible explanation of the results is that the ability to shift brain activation on task demands is decreased in GD. This implies that an inflexibility in brain activity could lie at the base of GD, leading to perseveration and persistence in gambling activities, despite the negative consequences.

While an imbalance between prefrontal structures and the mesolimbic reward system has been related to addictive behaviour, whether their dysfunction in GD is reflected in the interaction between them and their lateralization remains unclear. Koehler and colleagues (2015) strive to address this question using functional connectivity resting-state fMRI in individuals with GD and controls. GD patients demonstrated increased connectivity from the right middle frontal gyrus to the right striatum as compared to controls, which was also positively correlated with non-planning aspect of impulsiveness, smoking and craving scores in the GD group. Moreover, GD patients demonstrated decreased connectivity from the right middle frontal gyrus to other prefrontal areas as compared to controls. The right ventral striatum demonstrated increased connectivity to the right superior and middle frontal gyrus and left cerebellum in GD patients as compared to controls.

The seed regions used by this study for the functional connectivity analysis were lateralized to the right hemisphere because of a previous voxel-based morphometry study (Koehler et al., 2015) showing a significant difference in local grey matter volume centred in right PFC and right striatum between GD patients versus matched controls. The right lateralization is consistent with previous evidence showing that the prefrontal EFs, such as inhibitory control, are mainly situated in the right

hemisphere (Aron et al., 2004; Simmonds et al., 2008). Moreover, the involvement of right PFC has also been shown for self-regulation (Cohen & Lieberman, 2010; Knoch & Fehr, 2007). With respect to the reward system, imaging studies on GD reported right lateralized changes during reward processing: alterations only in right ventral striatum have been found in response to gambling stimuli (van Holst et al., 2012a) as well as during the processing of monetary reward (Reuter et al., 2005). However, this study is not without limitations since it involved mainly male subjects and considered specific targeted seed regions.

Given these premises, it is possible to state that further clinical EEG studies are needed to determine the presence and direction of the cortical imbalance in groups of GD patients.

4.6 To Summarize: Gambling Between Specificity and Uniqueness

The present chapter highlights the actual solely behavioural addiction included in the DSM-V under the non-substance related disorder, which is GD. What mainly distinguish GD from SUD is the absence of substance intake that is replaced by a repetitive and pathological behaviour. Indeed, in GD, there are no physical signs of pharmacological withdrawal, as frequently reported in SUD; however, irritability, anxiety, and sadness can be described when the gambling activity is interrupted voluntarily.

Before, several behavioural and neural parallels were previously traced between GD and SUD, and those include neural responsiveness in specific brain areas (such as frontocortical circuits and reward system structures), loss of control over the behaviour, tolerance aspects, withdrawal, repeated ineffective attempts to avoid or stop playing, and impairment of normal functioning (American Psychiatric Association, 2013).

Regarding the cognitive functioning, it is interestingly noticed that these disorders share the progressive loss of control in terms of amount of time dedicated to obtaining the substance or to be engaged in the repetitive behaviour. Progressively, all individual's activities revolve around the gambling behaviour, and he/she displays impaired cognitive control in cutting down or regulating the gambling activities. Reduced levels of self-control, indicating possible deficit in the inhibitory control brain networks, and higher degree of reward-seeking behaviour were found to characterize GD. Interestingly, the so-called "myopia for the future", the lack of metacognition and the possible impairment in interoceptive processes has been described in GD by discussing theories and models, behavioural study, and electrophysiological research.

To conclude, despite the relevance of EFs in GD, research in this field is still scarce and findings are not always consistent. Study limitations stress the need for further research utilizing comprehensive cognitive batteries, but also neuroscientific

methods (such as EEG and specific ERP analysis) on representative, unbiased, ecological samples of individuals with GD. Within this framework, we strongly believe the study of EF deficits deserve further attention and are extremely important in GD, because EFs integrity may have implications for the capacity of individuals with GD to seek a cure, to benefit from psychosocial treatments, and to avoid relapses.

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