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Review article

The Interaction of Person-Affect-Cognition-Execution (I-PACE) model for addictive behaviors: Update, generalization to addictive behaviors beyond internet-use disorders, and specification of the process character of addictive behaviors

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ABSTRACT

We propose an updated version of the Interaction of Person-Affect-Cognition-Execution (I-PACE) model, which we argue to be valid for several types of addictive behaviors, such as gambling, gaming, buying-shopping, and compulsive sexual behavior disorders. Based on recent empirical findings and theoretical considerations, we argue that addictive behaviors develop as a consequence of the interactions between predisposing variables, affective and cognitive responses to specific stimuli, and executive functions, such as inhibitory control and decision-making. In the process of addictive behaviors, the associations between cue-reactivity/craving and diminished inhibitory control contribute to the development of habitual behaviors. An imbalance between structures of fronto-striatal circuits, particularly between ventral striatum, amygdala, and dorsolateral prefrontal areas, may be particularly relevant to early stages and the dorsal striatum to later stages of addictive processes. The I-PACE model may provide a theoretical foundation for future studies on addictive behaviors and clinical practice. Future studies should investigate common and unique mechanisms involved in addictive, obsessive-compulsive-related, impulse-control, and substance-use disorders.

1. Introduction

The Interaction of Person-Affect-Cognition-Execution (I-PACE) model of specific internet-use disorders was published more than two years ago (Brand et al., 2016b). One aim was to describe the psychological and neurobiological processes underlying the development and maintenance of an addictive use of specific internet applications, such as those used when gaming, gambling, viewing pornography, buying-shopping, and social-networking. Since the publication of the I-PACE model, it has been cited relatively frequently by researchers globally not only for gaming disorder (e.g., Deleuze et al., 2017; Dieter et al., 2017; Dong et al., 2019; Kaess et al., 2017; Lee et al., 2018a, b; Li et al., 2018; Paulus et al., 2018; Sariyska et al., 2017), but also for gambling disorder (e.g., Ioannidis et al., 2019b; Starcke et al., 2018), compulsive sexual behavior disorder including problematic pornography use (e.g., Carnes and Love, 2017; Strahler et al., 2018; Wéry et al., 2018), buying-shopping disorder (e.g., Lam and Lam, 2017; Vogel et al., 2018), excessive use of communication applications (e.g., Dempsey et al., 2019;

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Elhai et al., 2018; Kircaburun and Griffiths, 2018; Montag et al., 2018; Rothen et al., 2018), unspecified internet-use disorder (e.g., Carbonell et al., 2018; Emelin et al., 2017; Ioannidis et al., 2019a; Lachmann et al., 2018; Vargas et al., 2019; Zhou et al., 2018b), and for other addictive behaviors including substance-use disorders (Zhou et al., 2018a). The eleventh edition of the International Classification of Diseases (ICD-11), as recently being released (World-Health-Organization, 2019), focuses on the disorder itself (e.g. gambling disorder) without referring to the medium of the disorder, for example gaming disorder instead of internet-gaming disorder in the fifth edition of the Diagnostic and Statistical Manual (DSM-5) (APA, 2013). In the ICD-11, the environment of the behavior may subsequently be specified as predominantly offline or predominantly online for gambling and gaming disorders. Consequently, a model explaining the underlying processes of the problematic behavior should be valid for both online and offline environment and for a combination of offline and online behaviors as well. We continue proposing that the behavior itself is the core element to consider and the environment (online versus offline) may be typically secondary but may contribute importantly to the expression of specific addictive behaviors and common variance across these behaviors (Baggio et al., 2018). We suggest an updated version of the I-PACE model, which we hypothesize will be valid not only for specific internet-use disorders, but also for other types of addictive behaviors. This updated I-PACE model concentrates on the individual psychological and neurobiological mechanisms of addictive behaviors. Media-specific aspects and other environmental factors related to the behavior that likely accelerate or decrease the development of an addictive behavior could then be defined and described for specific versions of the model. Fig. 1 summarizes the proposed differentiation between medium/ environmental aspects, individuals' reactions, and behavioral and neurobiological factors involved in addictive behaviors.

In addition, we aim at specifying more explicitly the process character of the model by differentiating two sub-models, one for the mechanisms involved in the early stages and one for the mechanisms involved in the later stages of the addiction process. We do not repeat the detailed discussion of all the components included in the I-PACE model (cf. Brand et al., 2016b). Instead, we mainly concentrate on the most recent articles, in particular meta-analyses and systematic reviews that inspired the I-PACE update.

2. The updated I-PACE model of addictive behaviors

The revision of the I-PACE model includes three main steps. First, we focus on predisposing variables, which have been considered as being involved in different types of addictive behaviors (gambling disorders, gaming disorder, and others) and differentiate them from more behavior-specific predisposing variables. Second, we define more precisely the inner circle of the addiction process in the I-PACE model with respect to recent findings. Third, we differentiate between early and later stages of the process in order to illustrate explicitly potentially different roles of moderating and mediating variables depending upon the stage of addiction. The revised I-PACE model of addictive behaviors is shown in Fig. 2. Fig. 2A shows the interactions between variables that are considered particularly important in early stages of addictive behaviors. Fig. 2B illustrates the interactions of variables in later stages of addictive processes.

2.1. The P-component of the I-PACE model

The P-component represents a person's core characteristics likely involved in the addiction process as predisposing variables (see discussion in Brand et al., 2016b). The general predisposing variables (left side in the upper box of the model) may contribute importantly to all types of addictive behaviors (e.g., gambling disorder, gaming disorder, buying-shopping disorder, pornography-viewing disorder/hypersexual behavior). The list of these potentially predisposing variables is not exhaustive. It only summarizes variables for which relatively broad evidence exists, including data from meta-analyses, even though the evidence may differ in strength for the different types of addictive behaviors. Data suggest a significant genetic contribution to gambling disorder (Lobo, 2016; Potenza, 2017, 2018; Xuan et al., 2017) and unspecified internet-use disorder (Hahn et al., 2017). Furthermore, negative early childhood experiences have been reported as vulnerability factors for gambling disorder (Roberts et al., 2017) and gaming disorder (Schneider et al., 2017), findings consistent with recent theoretical considerations of the role of attachment in addictive behaviors (Alvarez-Monjaras et al., 2018). Psychopathological correlates, in particular depression and social anxiety, have been reported repeatedly for gambling (Dowling et al., 2017), gaming (Männikkö et al., 2017), unspecified internet-use (Ho et al., 2014), and buying-shopping (Müller et al., 2019) disorders, and other behavioral addictions (Starcevic and Khazaal, 2017). Temperamental features, such as high impulsivity, have also been associated with gambling (Dowling et al., 2017), gaming (Gervasi et al., 2017; Kuss et al., 2018; Ryu et al., 2018), and unspecified internet-use (Kayiş et al., 2016) disorders, as has dysfunctional coping styles with gaming disorder (Schneider et al., 2018). In the I-PACE model, we use general terms (e.g., psychopathology, temperamental features including, for example, impulsivity) that may be specified further with respect to specific addictive behaviors. The behavior-specific predisposing variables (right side of the upper box in the model, Fig. 2A and B) are considered characteristic for the different specific addictive behaviors. For example, individuals with higher novelty-seeking may be more likely to develop gambling disorder (Del Pino-Gutiérrez et al., 2017). Individuals with higher aggressiveness and narcissistic personality traits may be more prone to develop gaming disorder (Gervasi et al., 2017). Individuals with high trait sexual motivation may be more likely to develop hypersexual behavior or pornography-use disorder (Stark et al., 2017), and individuals with high materialistic values may be particularly prone to developing buying-shopping disorder (Claes et al., 2016; Müller et al., 2014).

2.2. The inner circle: the affect (A-), cognition (C-), and execution (E-) components of the I-PACE model

One main idea of the inner circle of the I-PACE model is that the development of a problematic and addictive behavior occurs only in interactions between individuals' predisposing variables and certain aspects that specific situations deliver. The interactions result in experiences of gratification and compensation that are associated with specific behaviors. In the early stages (Fig. 2A), individuals may perceive external (e.g., confrontation with behavior-related stimuli) or internal triggers (e.g., negative or very positive moods) in specific situations. The perceptions may result in affective and cognitive responses, such as increased attention to these stimuli and urges to

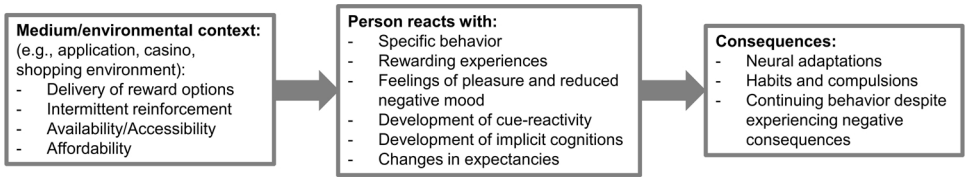

Fig. 1. Differentiating environmental aspects, individual reactions of the person, and consequences of repeated specific behaviors over time. The revised I-PACE model concentrates on the person's reactions and consequences involved in developing addictive behaviors.

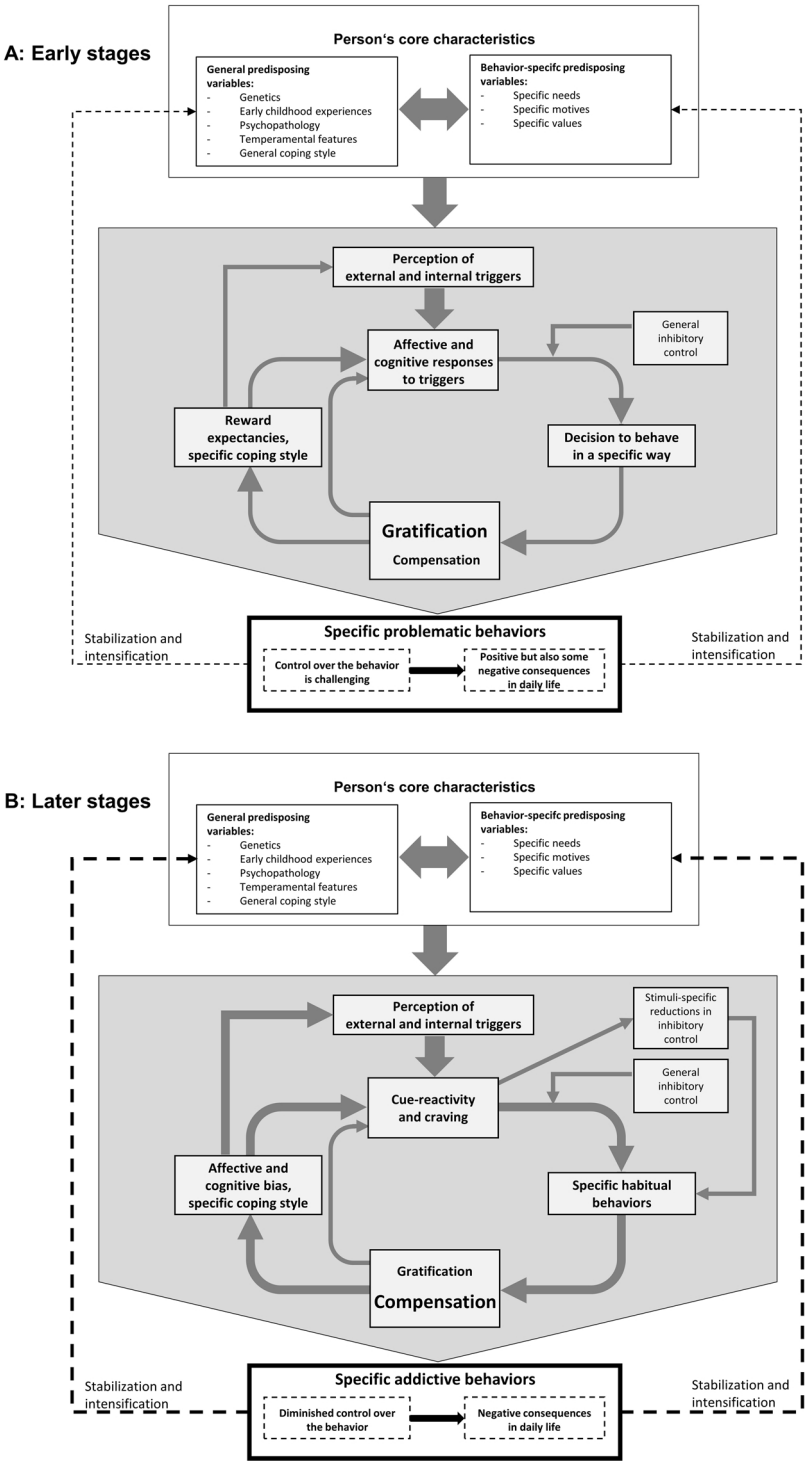

Fig. 2. The revised I-PACE model for addictive behaviors. Figure A shows early stages of the development of addictive behaviors. Figure B illustrates later stages of the process and factors contributing to the maintenance of addictive behaviors. Bolder arrows indicate stronger connections/accelerated mechanisms.

behave in specific ways; e.g. urges to play online games or view pornography (Starcke et al., 2018).

The affective and cognitive responses lead to decisions to behave in specific ways. The decision to engage in a specific behavior may be guided by two interactive systems: an impulsive/ reactive system, which is mainly based on associative learning (classical and operant conditioning), and a reflective/ deliberative system, which is mainly linked to reasoning and executive functions (Kahneman, 2003; Schiebener and Brand, 2015; Strack and Deutsch, 2004). In individuals with addictions, behavior is considered to depend increasingly on impulsive/ reactive neural systems, including limbic structures (Noël et al., 2006). Prefrontal-cortex-related inhibitory control over urges and desires may decrease during the addiction process (Bechara, 2005; Volkow and Morales, 2015). Combining these theoretical perspectives, we propose that relationships between affective and cognitive responses to external or internal triggers and decisions to engage in specific behaviors are moderated by the level of general inhibitory control (in contrast to mood-specific or stimulus-specific inhibitory control) and self-regulation/ self-directedness (Hahn et al., 2017), at least in the early stages of addictive behaviors. The meta-analysis by Meng et al.

(2015) illustrates that prefrontal dysfunctions are associated with gaming disorder, indicating the potential conflict between reward-anticipation and self-regulation systems, including conflicts involved in the delaying of gratification (Volkow and Baler, 2015). Regarding a general inhibitory control, Yao et al. (2017) report functional and structural brain alterations in gaming disorder that are linked to reductions in executive functioning. Specific behaviors (e.g. playing an online game, gambling in a casino, buying items) may lead to feelings of gratification or relief from negative moods (Laier and Brand, 2017). These experiences subsequently change the subjective reward expectancies that are associated with the specific behaviors. They may also modify the individual coping style. For example, if individuals learn that playing online games is effective in generating good feelings or avoiding negative emotional states, they may generalize the expectancy that playing online games is helpful for dealing with emotions in daily life (Kuss et al., 2018; Laier et al., 2018). The changes of expectancies and coping styles may increase the likelihood of responding with urge feelings or feelings of desire in subsequent situations when being confronted with external or internal triggers. This interaction of craving experiences and expectancies has been demonstrated by individuals with higher symptom severity of an addictive use of internet-communication services (Wegmann et al., 2018b). Over time, these associations between affective and cognitive responses, decisions to behave in specific ways, experiences of gratification and compensation and behavior-specific expectancies may become stronger. Consequently, control over the behaviors by general inhibitory mechanisms may become more difficult, and decisions to behave in specific ways may be guided more dominantly by impulsive/reactive responses to triggers. Mechanisms proposed to be involved in later stages of addictive behaviors are summarized in Fig. 2B.

In later stages of the addiction process, although the shift may be gradual, the aforementioned associations may become increasingly strong, resulting in habitual behaviors that may feel automatic in certain situations. Cue-reactivity and craving may evolve from affective and cognitive responses over time as a consequence of conditioning processes (Starcke et al., 2018). Previous research highlights the important role of sensitivities towards addiction-related stimuli and activations in neural reward systems involving the ventral and dorsal striatum and other limbic structures in addictive behaviors (Fauth-Bühler and Mann, 2017; Fauth-Bühler et al., 2017; Luijten et al., 2017; Palaus et al., 2017). Subjective expectancies may evolve into affective and cognitive biases, which may include biased or seemingly automatic attention to the respective behavior-related stimuli and triggers (Jeromin et al., 2016). We propose that compensatory effects become stronger over gratifying effects in later stages of the addiction process (cf. Brand et al., 2016b). In addition to the moderating effects of general inhibitory control on relationships between cue-reactivity/craving and the habitual behaviors, we propose that stimuli-specific inhibitory control may act as a mediator in the later stages of addictive processes (Everitt and Robbins, 2016). Several researchers have emphasized impairments in inhibitory control and executive functions in gambling disorder (Ioannidis et al., 2019b; van Timmeren et al., 2018), gaming disorder (Argyriou et al., 2017; Kuss et al., 2018; Yao et al., 2017), and unspecified internet-use disorders (Ioannidis et al., 2019a). We propose, however, that although the general inhibitory control may also decrease during addictive processes, the development of diminished specific stimulus-related inhibitory control is crucially involved in habitual behaviors in later stages of addictive behaviors. We propose that if cue-reactivity and craving have been developed as responses to external or internal triggers, this may lead to reductions in controlling desire when being confronted with addictive stimuli, which may then increase the likelihood of behaving habitually (Piazza and Deroche-Gamonet, 2013).

3. Neurobiological mechanisms

3.1. Neuroscientific theories of addiction integrated in the inner circle of the I-PACE model

Several neuroscientific theories and models explaining addictive behaviors have been integrated in the theoretical framework of the inner circle of the I-PACE model (Brand et al., 2016b). Direct links can be seen to the *Impaired Response Inhibition and Salience Attribution (I-RISA)* model (Goldstein and Volkow, 2011), *Incentive-Sensitization* (Robinson and Berridge, 2008), *Reward Deficiency Syndrome* (Blum et al., 1996) models and theories, and in dual-process approaches of addiction (Bechara, 2005; Everitt and Robbins, 2005, 2016) and ideas of imbalance between goal-directed behaviors and habits (Robbins et al., 2019). We also refer to aspects of more specific theoretical models integrating neuroscientific considerations of gambling disorder (Blaszczynski and Nower, 2002; Goudriaan et al., 2004) and gaming disorder (Dong and Potenza, 2014; Wei et al., 2017). Combining these theories, we consider the progression of an imbalance between increasing incentive-oriented urges and desires on the one hand and decreasing situation-specific inhibitory control over these urges and desires on the other hand as important for the development and maintenance of addictive behaviors. Increasing incentive sensitization, as a result of conditioning processes (Berridge et al., 2009), may associate with attentional bias and cue-reactivity in later stages of addiction processes. Individuals with reward deficiencies may be particularly prone to developing incentive sensitization (Blum et al., 2012). Incentive salience may promote cue-reactivity and craving, which may contribute to engagement in addictive behaviors.

Reductions in executive functions have considered both as vulnerability factors and consequences of addictive behaviors including substance-use disorders (Volkow et al., 2012). In behavioral addictions, such as gambling and gaming disorders, one may argue that executive reductions constitute vulnerability factors and do not develop as a consequence of the addictive behavior, because no direct substance-related neurotoxic effects on the brain are involved. Consistent with this notion, we propose that a diminished level of general inhibitory control is a vulnerability factor for the addictive behavior and acts as a moderating variable of the relationship between affective responses to certain triggering stimuli (e.g., stress or negative moods) and decisions to engage in specific behaviors (see Fig. 2A). In addition, however, we argue that beyond this moderating effect of executive functions as a vulnerability factor of addictions, situation-specific inhibitory control (when being confronted with addiction-related stimuli) may decrease over time as a consequence of addictive behaviors, although – in contrast to substance-use disorders – no direct neurotoxic effects on the brain are involved in behavioral addictions. Reductions in stimuli-specific inhibitory control may develop based on cue-reactivity and craving and be accompanied by functional brain changes in addiction-related circuits (Ersche et al., 2012; Koob and Volkow, 2010; Volkow and Morales, 2015; Volkow et al., 2012). Thus, in later stages of addictive behaviors (Fig. 2B), stimuli-specific inhibitory control processes may be influenced by craving and urges related to encountering addiction-related stimuli, which may then make it more likely that an individual will behave habitually or seemingly automatically (Everitt and Robbins, 2005, 2013, 2016).

3.2. Neural correlates of the main processes within the inner circle of the I-PACE model

The aforementioned proposed imbalance between limbic/ reward-oriented brain circuits and prefrontal control in behavioral addictions has been reviewed relatively extensively for gambling disorder (Clark et al., 2013; Goudriaan et al., 2014; Potenza, 2013; van Holst et al., 2010) and gaming disorder (Kuss et al., 2018; Weinstein, 2017; Weinstein et al., 2017), including in meta-analyses (Meng et al., 2015).

Although less extensively, there also exist neuroimaging studies of compulsive sexual behaviors, including problematic pornography use (e.g., Brand et al., 2016a; Gola et al., 2017; Klucken et al., 2016; Schmidt et al., 2017; Voon et al., 2014), which have been examined in recent reviews (Kraus et al., 2016; Stark et al., 2018). Scientific studies of neural correlates of buying-shopping disorder are relatively scarce. There are, however, some studies from consumer psychology perspectives (e.g., Raab et al., 2011) and studies using electrophysiological measures to investigate neurobiological mechanisms of buying-shopping disorder (Trotzke et al., 2014) which have been reviewed recently (Kyrios et al., 2018; Trotzke et al., 2017). Although not yet recognized as a clinical condition, there are also recent publications on structural and functional neuroimaging findings of poorly controlled and problematic use of social network sites and other internet-communication applications (e.g., Dieter et al., 2017; He et al., 2017; Lemenager et al., 2016; Montag et al., 2017, 2018; Turel and Qahri-Saremi, 2016), which have been reviewed by Wegmann et al. (2018a).

There is significant variability across neuroimaging studies of addictive behaviors regarding the types of behavioral addictions, the techniques used (e.g., structural/functional magnetic resonance imaging [s/fMRI], positron emission tomography [PET]), the psychological constructs or processes of interest, the experimental tasks used to measure specific functions, the samples included (convenient samples with individuals showing different degrees of symptoms versus clinically diagnosed individuals or treatment-seeking patients), and the diagnostic procedures used. Nevertheless, when drawing conclusions from the studies, meta-analyses and reviews (see, for example, citations above), there is first evidence for hyperactive involvement of limbic structures, including the amygdala and the ventral striatum, and hypoactive prefrontal-striatal circuits involved in cognitive control over the behaviors. There are, however, some caveats, for example hypoactive engagement of reward circuitry during the anticipatory phase of monetary processing (Balodis and Potenza, 2015), with some researchers proposing differences relating to the processing of addictive cues (hyperactive reward responding) and non-addictive rewarding cues (relatively hypoactive reward responding) (Limbrick-Oldfield et al., 2013). The insula may be a mediator between the two systems (limbic and prefrontal-striatal), representing the somatic states linked to craving and the desire to behave specifically (see discussion in Namkung et al., 2017; Wei et al., 2017). The main structures that have been identified as potential brain correlates of addictive behaviors are summarized in Fig. 3.

In the recent meta-analysis of cue-reactivity-related brain activity in fMRI-studies with samples of patients with behavioral addictions compared to control subjects (Starcke et al., 2018), the dorsal striatum (caudate nucleus) was more active in individuals with addictions compared to those without and in individuals with addictions when contrasting the addiction-related condition with the neutral condition in the cue-reactivity tasks. The findings may reflect shifts from involvement of the ventral striatum in early stages of behavioral addictions when being confronted with addiction-related stimuli to involvement of the dorsal striatum in later stages of the disorder, when the behavior becomes more habitual (Everitt and Robbins, 2013, 2016; Zhou et al., 2019). The brain structures and circuits that likely underlie addictive behaviors and shifts from early to later stages of addiction processes are illustrated schematically in Fig. 3.

Widespread connections among fronto-striatal structures have been examined using resting-state fMRI investigations with healthy subjects and have been shown to be involved in behavioral flexibility (Morris et al., 2016). These circuits are also broadly consistent with functional networks involved in emotion regulation (Öner, 2018). Changes in connectivity between specific structures involved in fronto-striatal circuits (e.g., connectivity between amygdala and medial PFC) appear important in explaining emotion dysregulation in substance-use disorders (Koob, 2015; Wilcox et al., 2016). The connectivity of networks involved in cognitive control (fronto-parietal circuits and medial frontal

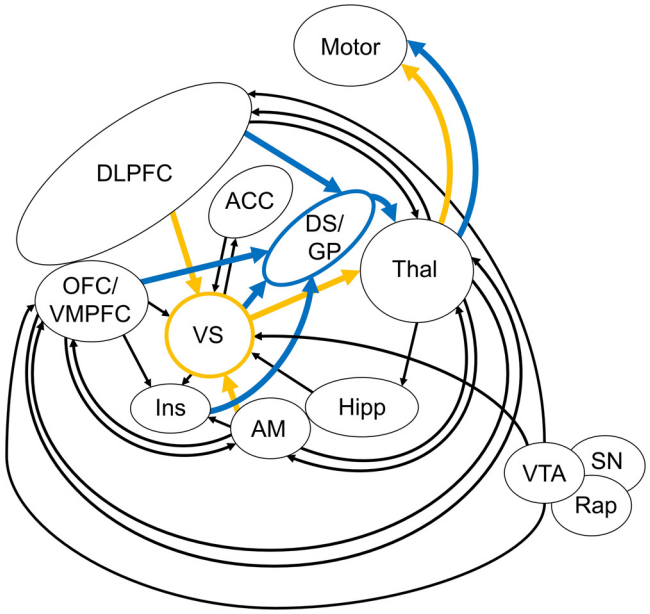

Fig. 3. Brain circuits potentially underlying addictive behaviors. Orange arrows represent main circuitry proposed to be involved in early stages of addictive processes. Blue arrows indicate the additional involvement of the dorsal striatum and related structures in later stages of addiction processes, when behaviors become more habitual. ACC = anterior cingulate cortex, AM = amygdala, DLPFC = dorsolateral prefrontal cortex, DS = dorsal striatum, GP = globus pallidus, Hipp = hippocampus, Ins = insula, Motor = motor cortex and associated regions for executing behaviors, OFC = orbitofrontal cortex, Rap = serotonergic raphé nuclei, SN = substantia nigra, Thal = thalamus, VMPFC = ventromedial prefrontal cortex, VS = ventral striatum, VTA = dopaminergic ventral tegmental area.

areas) and in reward processing (including subcortical and limbic structures) has also been shown to predict abstinence in cocaine-use disorder after treatment (Yip et al., 2019). A stronger separation of the two networks involved in executive control and in reward sensitivity has been proposed to underlie behavioral flexibility and decreased compulsivity, which may explain better therapeutic outcomes (Yip et al., 2019).

In summary, we propose that an imbalance in circuits underlying behavioral flexibility and emotion/urge regulation relate to main aspects of addictive behaviors. The pathways include dopaminergic projections from the ventral tegmental area and substantia nigra to prefrontal areas, the ventral striatum, and anterior cingulate gyrus as well as serotonergic projections from raphé nuclei to prefrontal areas (mainly orbitofrontal regions) (Everitt and Robbins, 2005; Volkow et al., 2012, 2013). The interconnections between striatal structures, thalamus, and prefrontal areas depend largely upon glutamate and gamma-aminobutyric acid (GABA) (Naaijen et al., 2015), and the neurochemical systems involved in fronto-striatal loops act in concerted and cross-regulatory fashions (Gleich et al., 2015). Neurochemical correlates of addictions have been discussed intensively elsewhere, and many studies emphasize the important role of dopamine in substance-use disorders (Herman and Roberto, 2015; Pascoli et al., 2018; Volkow et al., 2016). The findings concerning dopamine in behavioral addictions are, however, less robust (Potenza, 2018).

Although a meaningful number of studies on neural correlates of behavioral addictions have been published in recent years, there remain limitations that should be mentioned. First, most studies have been published on gambling disorder and gaming disorder (see comments above). Less evidence exists for other behavioral addictions for example compulsive sexual behaviors including problematic pornography use, buying-shopping disorder, and other potential phenomena that are not yet recognized as clinical conditions, such as problematic use of social-

network sites. In particular, studies that systematically investigate neural correlates of specific psychological functions (e.g., craving, inhibitory control) across specific types of behavioral addictions are missing. Studies investigating stages of addiction processes or symptom severity as predictors or moderating variables of neural activity and potential structural brain abnormalities are important to better understand mechanisms underlying the progression of addictive behaviors. Consistent with this, longitudinal studies on brain correlates of addictive behaviors that test specific hypotheses are missing. Investigating the potential shift from activity of the ventral to the dorsal striatum as response to addiction-related cues across types of behavioral addictions and across different stages of addiction, using both cross-sectional and longitudinal designs, would help to understand better the nature of addictive behaviors. Such studies are necessary to disentangle potential shifts from craving to compulsion and from expecting gratification to expecting relief from negative states when encountering addiction-related stimuli in different stages of behavioral addictions, which in turn should help in optimizing treatments. Studies comparing different types of addictive behaviors and different stages of addiction processes including prospective longitudinal studies could also investigate hypothesized involvement of reductions in inhibitory control as a vulnerability factor and/ or as a consequence of the addictive behaviors, and one that may mediate links between affective responses and habitual/ compulsive behaviors (see discussion in Everitt and Robbins, 2016).

4. Conclusion and future directions

The updated I-PACE model is a theoretical approach for describing the process of addictive behaviors by combining psychological and neuroscientific theories of substance-use disorders and behavioral addictions. We consider disorders due to addictive behaviors being the consequence of interactions between core characteristics of a person and several moderating and mediating variables, which may be dynamic and develop over time as a consequence of engagement in specific behaviors. We propose that the I-PACE model of addictive behaviors may be useful for psychological and neuroscientific research because it permits formation and testing of clear hypotheses regarding interaction effects of specific variables in explaining variance in symptom severity of behavioral addictions. The model may also inspire clinical practice (cf. King et al., 2017; Potenza, 2017) by defining and investigating possible mediating variables that may represent important targets for treatment (e.g., expectancies, affective and cognitive responses to triggers). The updated I-PACE model also offers the possibility of deriving hypotheses on the stages of addictive processes (both during progression and recovery), for example by arguing that reductions of specific inhibitory-control accelerate in later stages in the progression of addiction processes. It is important to note, however, that we consider theoretical models as being dynamic. The validity of specific hypotheses, combined in a theoretical framework, should be evaluated empirically, and theoretical models should be updated by considering recent scientific findings from different perspectives.

It is important to keep in mind that the theoretical model proposed is based on different degrees of scientific evidence with respect to specific addictive behaviors. As mentioned in the previous sections, the involvement of specific psychological mechanisms and neurobiological processes is relatively well studied in gambling disorder and gaming disorder, and less intensively investigated in other types of behaviors that potentially may become addictive (for example pornography use, buying-shopping, and social-networking). Furthermore, for some aspects and mechanisms proposed in the updated I-PACE model, different levels of evidence exist. For executive functions and inhibitory control, a relatively large number of studies has used experimental paradigms and has examined particular aspects of executive functioning in different types of addictive behaviors. On the other hand, for cue-reactivity and craving in specific addictive behaviors, some studies applied a correlational design, which makes interpretations of causality and the time of development of cue-reactivity and craving in the addiction process difficult to define (Zilberman et al., 2019). Considering these limitations it is important to emphasize that the model proposed is a theoretical model that summarizes the current state-of-the-art of behavioral addiction research and aims at inspiring theory-based future studies.

Another issue that is important to consider is that personality and temperament features are rather vague predictors for specific addictive behaviors, given that these variables are involved in many psychopathologies and often explain only a mild-to-moderate proportion of symptoms across different disorders (Zilberman et al., 2018).

We would also like to comment on the current debate with respect to classifying buying-shopping disorder and pornography-use disorder as impulse-control disorders or as behavioral addictions. Problematic pornography use may be considered as one facet of compulsive sexual behavior disorder within the category of impulse-control disorders in the ICD-11. Buying-shopping disorder has been proposed as an example for other specified impulse-control disorders in the ICD-11 (World-Health-Organization, 2019). Many researchers, however, argue that both types of disorders would be better classified as addictive behaviors (Potenza et al., 2018).

One challenge for future research and theory building involves identifying and disentangling potential commonalities and differences between disorders due to addictive behaviors and other mental disorders, such as obsessive-compulsive-related and impulse-control disorders, which may relate to behavioral addictions on psychological and neurobiological levels (Chamberlain et al., 2016; Fineberg et al., 2013, 2018; Robbins et al., 2019). For example, inhibitory control and reward processing have also been proposed to be important in obsessive-compulsive-related and impulse-control disorders, such as discussed in skin-picking disorder and trichotillomania, which have also been related to functioning of fronto-striatal brain circuits (Chamberlain et al., 2008). Dysfunctions of fronto-striatal loops may, however, be involved in multiple other mental health disorders as well (Mitelman, 2019). Nonetheless, the fact that fronto-striatal loops are involved in different mental disorders does not necessarily mean that the psychological processes related to the clinical phenotypes of disorders are the same. First, fronto-striatal loops are differently defined and analyzed across studies. Future studies should examine more closely specific contributions of structures currently broadly defined within fronto-striatal loops to certain specific psychological processes underlying specific problematic behaviors. Second, the general involvement of inhibitory control and reward processing does not mean that the psychological processes are comparable across disorders, although there may be some overlap of impulsivity/compulsivity and addictive behaviors (e.g., Chamberlain et al., 2018). It is important to define more precisely the factors underlying the temporal progression of motivations for people to engage excessively in specific behaviors. For example, in addictive behaviors, it may be that core motivations to engage in gaming or gambling, at least at early stages, involve reward anticipation. In later stages, avoidance of negative feelings is likely additionally involved. In obsessive-compulsive-related disorders, it may be that the core drive at early stages involves avoidance of negative feelings or anxiety. Later, the behavior itself may potentially be experienced as rewarding because it may help relieve stress. In other words, the general involvement of specific neurocognitive functions may not entirely explain a disorder. The same idea potentially applies to neural mechanisms. It may be the case that in disorders due to addictive behaviors, the ventral striatum contributes importantly at early stages of the disorder, with respect to cue-reactivity and craving. In later stages, the dorsal striatum may become more involved and relate to habitual and compulsive aspects of addictive disorders. In contrast, the dorsal striatum is likely involved in obsessive-compulsive-related disorders and impulse-control disorders, such as trichotillomania, from early stages (Isobe et al., 2018; van den Heuvel et al., 2016).

In future studies, it seems important to investigate processes and interactions of different neurocognitive functions in different types of addictive behaviors in order to achieve a better understanding of the underlying natures of behavioral phenomena. The I-PACE model may be used for defining and clarifying specific hypotheses in researching these phenomena. It is important to examine hypothesized processes in addictive behaviors and compare them with other mental disorders, such as obsessive-compulsive-related and impulse-control disorders to understand whether the underlying processes involved are different or similar. In this process, data generated should help clarify the extent to which different terms may be currently employed for describing similar mechanisms across disorders. In this manner, the updated version of the I-PACE model provides a theoretical framework that should help address major questions relating to addictive, obsessive-compulsive, impulse-control and other disorders, including those relating to internet use, which may become increasingly relevant over time given changes in the digital technology environment.

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