Structure and Mechanism of Addictive Impulsivity Based on Drive-Control Interaction

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Abstract

Substance-dependent individuals exhibit pronounced impulsive characteristics, manifested as persistent substance use that is difficult to discontinue. The emergence of this behavior, in addition to deficits in inhibitory control, is importantly driven by motivational forces originating from multiple dimensions. Weak inhibitory capacity cannot withstand the influence of these driving forces, resulting in a state of imbalance that gives rise to compulsive or habitual drug-use behavioral tendencies under uncontrollable craving. These driving forces originate from various sources, including reward effects, S-R (stimulus-response) type cue reactivity formed through conditioning, and personality traits such as sensation seeking; the control components, conversely, encompass response inhibition and executive control functions such as delay discounting.

Full Text

Structure and Mechanism of Addictive Impulsivity Based on the Interaction Between Drive and Control

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Abstract: Substance addicts exhibit significant impulsivity, manifesting as persistent substance use and difficulty achieving abstinence. Beyond deficits in inhibition and executive control, driving forces from multiple psychological dimensions constitute a critical cause of impulsive substance use. These drives

originate from diverse sources, including reward effects, conditioned stimulus-response (S-R) cue reactivity, and personality traits such as sensation seeking. When inhibitory capacity is insufficient to resist these drives, an imbalance emerges, producing compulsive or habitual substance use tendencies under uncontrollable craving—behaviors that epitomize addictive impulsivity.

Keywords: addictive behavior, impulsivity, drive force, inhibition

Impulsivity is defined as unplanned, hasty responses to internal and external stimuli, where individuals fail to consider negative consequences for themselves or others (Koob & Volkow, 2010). This phenomenon appears across numerous clinical conditions and exacerbates dysfunctional behaviors, with addiction serving as a paradigmatic example. Impulsivity correlates with the maintenance and relapse of various substance addictions (Long et al., 2020), functions as an endophenotype that predicts addiction, and represents a shared behavioral characteristic among different addict populations (Dalley & Ersche, 2019).

Impulsivity is typically divided into three major types. Trait impulsivity is a pre-existing personality dimension related to self-regulation, usually assessed through self-report measures. Waiting impulsivity reflects discounting of delayed rewards and high-risk decision-making. Stopping impulsivity refers to an individual' s inability to control motor responses and effectively terminate initiated behaviors in a timely manner—also known as response inhibition. This represents a top-down control dysfunction reflecting the capacity to inhibit dominant responses (MacKillop et al., 2016). These three types are considered three latent structures of impulsivity that are mutually independent yet each correlates robustly with substance abuse. However, this classification involves considerable overlap, as both motor and waiting impulsivity (both behavioral manifestations) may also be embedded within impulsive personality.

Impulsive personality traits are primarily measured using the BIS-11 Impulsiveness Scale (Barratt Impulsiveness Scale-11) and the UPPS Impulsive Behavior Scale. The BIS-11 assesses three characteristics: attentional bias, non-planning, and motor impulsivity, with the latter reflecting a heritable trait of low response inhibition (Morein-Zamir & Robbins, 2015). This motor impulsivity demonstrates high correlation with response inhibition measured through laboratory tasks such as Go/No-go and Stop Signal Task (SST) (Smith et al., 2014), suggesting they reflect the same underlying psychological construct. Numerous studies demonstrate that pre-addiction levels of motor impulsivity predict cocaine use (Belin et al., 2008; Dalley et al., 2007), nicotine use, and cocaine reinstatement following punishment (Diergaarde et al., 2008).

Among addicts' personality traits, sensation seeking predisposes individuals to experimental and recreational drug use. Subsequently, low inhibitory control makes self-regulation difficult, gradually fostering goal-directed behavior that evolves into habitual and compulsive use under drug-related cues, thereby exhibiting strong impulsive features. Following withdrawal, withdrawal symptoms and basic (non-cue-induced) craving gradually diminish with prolonged absti-

nence, yet the intensity of conditioned craving (behavioral responses, emotions, and stress induced by drug-related cues—i.e., habitual drug use behavior and craving) increases over time (Bedi et al., 2011). This manifests as uncontrollable compulsive drug use behavior, precipitating relapse and creating a vicious withdrawal-relapse cycle that accentuates impulsive characteristics.

Consequently, weak inhibitory and control capacities play crucial roles throughout the trajectory from initial drug use to addiction and repeated relapse. Moreover, continuous drug use may further impair these capacities, generating progressively stronger impulsivity levels that persist throughout the addiction course, representing a key psychological hallmark of addictive behavior (Zeng et al., 2015).

Nevertheless, impulsivity possesses multidimensional and complex properties (Vassileva & Conrod, 2019), functioning as both a "response" and an "outcome." It may manifest as a behavior or as a trait. The single factor of low control cannot directly produce impulsive behavior. While high impulsive behavior and features are important expressions of poor inhibitory control, impulsive traits are not entirely equivalent to low control, and impulsive behavior is not merely a consequence of low control. Rather, it emerges when low inhibitory control fails to restrain one or more driving forces. For addicts, this results in persistent drug use and addiction, followed by repeated withdrawal and relapse. What are these uncontrollable forces? This remains incompletely understood. Current research predominantly emphasizes impaired inhibitory control in impulsive behavior while rarely identifying what the response inhibition system cannot control. Impulsive features are equated with "disinhibitory" characteristics—that is, top-down control mechanism dysfunction. Yet impulsive behavior reflects an individual' s inability to inhibit (reward-)driven responses that are inappropriate for current demands (Aron et al., 2007). Therefore, impulsivity should comprise at least two components: inhibitory control and drive response, with imbalance between them producing impulsivity. This is corroborated by numerous imaging studies on the neural basis of impulsivity in neurocognitive tasks: behaviors during different addiction stages are regulated by distinct brain circuits involving key regions including the orbitofrontal cortex-dorsal striatum, basolateral amygdala, craving-related hippocampus, insula, and cingulate cortex, dorsolateral prefrontal cortex, and inferior frontal gyrus related to impaired inhibitory control (Koob & Volkow, 2010). Research on cocaine addicts also reveals that the prefrontal-striatal-amygdala network negatively correlates with inhibitory control task performance (Wang et al., 2018). Furthermore, studies demonstrate that brain networks responsible for drive and those responsible for inhibitory control in addicts show abnormal changes in opposite directions (Zeng, et al., 2018).

Thus, regarding addictive behavior, impulsivity manifests as persistent drug use resulting from addicts' inability to control certain forces. These forces constitute the drivers that shape impulsive drug use behavior. When inhibitory control cannot withstand these drives, impulsive drug use responses emerge. What

factors (drives) trigger such impulsive responses (drug use behavior)? How do these drives develop? What is their relationship with addicts' inhibitory characteristics? Only by clarifying these questions can we better understand addictive impulsivity and identify effective interventions. Accordingly, this article examines the structural composition and origins of addictive impulsivity from the perspective of drive and inhibitory factors, providing a theoretical foundation for understanding impulsive drug use behavior and developing more targeted interventions.

1. The Structure of Addictive Impulsivity

As previously noted, impulsivity is not a unitary psychological construct. Multiple theories offer different perspectives: the "three-factor theory" proposes that impulsivity comprises three independent dimensions—behavioral disinhibition, impulsive decision-making, and inability to focus attention (de Wit, 2009). However, these dimensions describe different types of impulsive features rather than structural components, with each type manifesting as distinct impulsive behaviors.

The iRISA (Impaired Response Inhibition and Salience Attribution) model posits that impaired response inhibition and salient incentive brain regions underlie addictive impulsivity: response inhibition represents control, while drug salience constitutes a drive. Yet salience is not an ultimate explanation—what causes this salience, and in what form does it manifest? Moreover, the sources of drive and control in addictive impulsivity clearly extend beyond this. Evidence indicates that reward, habitual behavior, addiction memory, and executive function brain regions all contribute to addiction (Zilverstand et al., 2018), serving control and drive functions respectively. Consequently, the iRISA model cannot fully capture the drive and control factors of addictive impulsivity.

The two-factor model proposes that reward drive (continuously increasing approach impulse) and reckless impulse (continuously decreasing inhibitory control) constitute the two major structures of impulsivity. Their interaction prevents individuals from inhibiting reward-driven impulses, generating impulsive behavior (Verges et al., 2019).

Both iRISA and two-factor models acknowledge that impulsivity comprises multiple elements and that inhibitory response plays a role, viewing addicts' declining inhibitory control as a regulatory factor in impulsive drug use. However, from the perspective of what drives impulsive behavior, the iRISA model emphasizes attentional salience function, yet salience itself is not a "stimulus" and lacks driving force; it can only facilitate or mediate. The factors that trigger attentional salience are the true drives, but the model does not specify what these are. The two-factor model identifies reward effect as an important drive component but neglects habitual drug use tendencies under drug-related cues formed through conditioning after addiction. This "S-R" type of impulsive behavior may exert far greater influence on drug use during addiction and post-

withdrawal relapse than reward effects (Everitt & Robbins, 2016; Zeng et al., 2018).

Furthermore, different forces trigger impulsive behavior from initial drug use through addiction. Drug-related cues (paraphernalia, environments, and people associated with use) serve as important stimulus sources that promote habitual drug use behavior formation through conditioning (Ito & Doya, 2015). Various negative emotions in daily life, stress, addicts' own negative urgency, and sensation-seeking personality traits all trigger uncontrollable drug use behavior, causing use or post-withdrawal relapse (Emmanuel & Lina, 2018). Thus, reward effect, life events, negative emotions, and stress may all be important drive factors in addictive impulsivity, promoting S-R habitual drug use behavior in stimulus form. Although the mechanism remains uncertain, reward effect and drug-related cues—including negative emotions and stress—are clearly important stimulus sources in habitual drug use that readily trigger uncontrollable S-R habitual drug use behavior, namely addictive impulsive behavior.

In summary, addictive impulsivity features should originate from two forces: drive and control, with imbalance between them producing impulsive behavior. Drive force includes at least reward effect and "stimulus-response"force triggered by conditioning. Sensation seeking and negative urgency in addicts' personality traits may also constitute drives. Among stimulus sources (drives), reward effect and drug-related cue responses are factors determining addictive impulsivity features, while inhibitory control regulates drive. When drive is strong and inhibitory control is weak, impulsive drug use behavior occurs.

2.1 Response Inhibition Features Related to Impulsive Personality Traits

In addictive behavior, the most salient manifestation of impulsivity is addicts' impulsive personality characteristics. Research demonstrates that self-reported impulsivity features show stable, reliable correlations with drug use (Argyriou et al., 2018). Substance addicts exhibit higher BIS trait impulsivity than healthy non-using controls, primarily manifested as sensation seeking, attentional impulsivity (bias), non-planning, low inhibitory control, and negative urgency (Zeng et al., 2013). However, sensation seeking relates more to substance use than to addiction itself (Pattij & De Vries, 2013), though it may serve as a drive for addictive impulsivity. Low inhibitory control manifests as motor impulsivity—difficulty canceling initiated motor responses—commonly measured using Go/No-go and Stop Signal Tasks. Substance addicts demonstrate poorer stopping impulsivity on Stop Signal Tasks, reflecting impaired response inhibition (Belin, et al. 2008), which is precisely the expression of motor impulsivity. In neuropsychology and cognitive neuroscience, this impulsivity is termed "disinhibition" or response dyscontrol. Strong inhibition can suppress automated, situationally inappropriate responses. This impulsivity depends on connectivity mechanisms between the dorsolateral striatum (DLS) and prefrontal cortex (PFC) (Dalley & Ersche, 2019). Other research indicates that the right inferior

frontal gyrus (IFG) and anterior cingulate cortex (ACC) regulate successful stop inhibition performance, while more pronounced dorsolateral prefrontal cortex (DLPFC) activation during successful inhibition (Go/No-go task) in early adolescents predicts fewer later problem behaviors (Martz et al., 2018).

Cocaine addicts exhibit trait impulsivity before drug use that is familially heritable and has specific neural underpinnings: primarily structural and functional brain abnormalities related to fronto-striatal pathway structural anomalies (Dalley & Ersche, 2019; Ersche et al., 2013). Additionally, reduced white matter density in the right IFG and increased gray matter volume in the putamen—structural and functional features of brain regions involved in drive and control—demonstrate that drug abuse behavior and manifested impulsivity relate to prefrontal cortex-associated control functions (Everitt & Robbins, 2016).

Nevertheless, although action impulsivity stemming from inhibitory control deficits and its neural basis serve as important susceptibility factors promoting substance use, these brain structure associations also appear in the general population without substance use (Deserno et al., 2015). Research shows that drug abusers and their first-degree siblings exhibit similar trait impulsivity on the BIS and display similar brain activation and gray matter volume changes (Ersche et al., 2012; Morein-Zamir & Robbins, 2015). Activation of the lateral orbitofrontal cortex during successful response inhibition correlates with self-reported alcohol, nicotine, and illicit drug use, with siblings showing similar high activation in comparable brain regions (Morein-Zamir & Robbins, 2015). Prospective studies on human infancy also indicate that behavioral impulsivity and accompanying brain function exist at least partially before drug use, constituting a risk factor for addiction.

In summary, the similar behavioral and neural characteristics between addicts and their family members, and their differences from control groups, reflect pre-existing low inhibitory control traits that precede drug use. These personality traits, termed impulsive personality traits, include at least high sensation seeking and low response inhibition. Grounded in abnormal physiological mechanisms, they render addicts congenitally susceptible to drug use with vulnerable inhibitory control capabilities.

2.2 Cognitive Control Deficits in Delay Discounting and Their Neural Mechanisms

Another form of impulsive behavior, termed selective impulsivity, significantly predicts persistent cocaine and nicotine seeking and use (Pattij & De Vries, 2013). In the short term, this impulsivity manifests as inability to inhibit initiation of inappropriate responses based on incorrect temporal or probability predictions (Dalley & Ersche, 2019); in the long term, it appears as inability to tolerate delayed rewards (Castellanos & Tannock, 2002; Noreika et al., 2013), reflecting discounting of delayed rewards and high-risk decision-making (Hobkirk et al., 2019). Individuals with attention-deficit/hyperactivity disorder (ADHD)

and drug addicts cannot tolerate delayed rewards (Bickel et al., 2014; Weafer et al. 2014), and impulsive forms also include extreme intolerance of delayed rewards (Dalley & Robbins, 2017).

In substance use contexts, individuals with lower control capabilities are abnormally sensitive to immediate satisfaction and rewarding stimuli, making them more vulnerable to substance use problems (Martel et al., 2009). Human experiments measure this using the 4-Choice Serial Reaction Time Task (4CSRTT) (Voon et al., 2014), which requires withholding responses until a clear signal appears. Addicts typically show remarkably similar deficits: premature responses and slower SSRTs (Stop Signal Reaction Times) (Ersche et al., 2013).

This cognitive control dysfunction over goal-directed behavior is regulated by bilateral frontoparietal cortex, including dlPFC, vlPFC, and posterior parietal lobe (Laird et al., 2011). Rodent lesion studies and human functional imaging also indicate that nucleus accumbens (NAC), basolateral amygdala (BLA), hippocampus, insula, lateral prefrontal cortex (PFC), posterior cingulate cortex (PCC), parietal cortex, and orbitofrontal cortex (OFC) are involved in delay discounting impulsivity (Dalley & Ersche, 2019). This demonstrates not only the neural mechanisms of cognitive control but also that waiting impulsivity includes drive forces determined by functions of nucleus accumbens, amygdala, etc. The early "goal-directed" drug use behavior produced by addicts under reward drive is precisely a manifestation of this cognitive control failure, and post-addiction compulsive behavior is also influenced by this dysregulated cognitive control, showing strong impulsivity (王鹏飞 et al., 2019).

However, it remains uncertain whether this waiting impulsivity tendency toward risky decision-making is inherent in personality traits or neural basis or is shaped by addictive drugs. Ersche et al. (2013) found that stimulant addicts and their non-addicted siblings both show increased impulsivity and compulsive personality traits, along with expansion in limbic-striatal regions in neural basis, suggesting that this ventral striatum-prefrontal cortex-based waiting impulsivity may also relate to innate personality traits. Thus, both response inhibition and cognitive control functions that regulate impulsivity may involve genetic factors and may further deteriorate during drug use. For addicts, it is clear that such inhibition and control cannot regulate drug use tendencies caused by drive forces, thereby forming impulsive drug use throughout both early goal-directed behavior and later habitual, compulsive drug use processes.

3. Structural Factor Analysis of Drive Forces in Addictive Impulsive Behavior

As discussed, addictive impulsivity features and behaviors originate from at least two forces: drive and control, with imbalance between them producing impulsivity. Beyond reward effects from addictive substances, drive forces for addictive impulsivity also derive from "S-R" responses triggered by conditioning and addicts' own sensation seeking, negative urgency, etc. These psychological

phenomena possess driving force, manifesting significant impulsivity and triggering drug use behavior when addicts have low inhibitory control.

3.1 "Reward" Effect and Addiction Memory

In early drug use, substances activate relevant brain regions to trigger reward effects that sustain use. Reward effects also regulate memory-related brain regions, enhancing drug-related memory and potentially increasing drug use probability (Goodman & Packard, 2016; White et al., 2013). This purposeful behavior is driven by rewarding outcomes. Drug users' low inhibition cannot control this reward force, leading to persistent reward-oriented drug use that develops into early "goal-directed" drug use behavior in addiction (Koob & Volkow, 2010), manifesting as waiting impulsivity.

These waiting impulsive features depend on functional integration of the ventral striatum (Dalley & Ersche, 2019). The mesolimbic dopamine system is the hub of the reward circuit (Haber & Knutson, 2010). The mechanism integrating the nucleus accumbens core and peripheral limbic subregions in the ventral striatum, such as the limbic cortical system, regulates "reward" effects (Dalley & Robbins, 2017). This region is involved in evaluating drug rewards, intensifying urgency to use until individuals become completely preoccupied with drug use thoughts, especially during withdrawal states (Koob & Volkow, 2016), driving relapse. Research comparing reward and executive control network resting-state functional connectivity between cocaine users and non-users and its relationship with impulsive decision-making found that imbalance between reward and executive control circuits causes use impulsivity (Hobkirk et al., 2019). Additionally, strong inverse coupling between drug users' reward network and left cognitive control network is highly correlated with risk-taking tendency (Andersen, 2019).

Reward effects can drive drug use in early addiction development. However, research shows that abnormal ventral striatum activation related to reward anticipation cannot significantly predict fewer later problem behaviors in adolescent addiction (Martz et al., 2018), indicating that besides reward effects, other factors drive drug use behavior and impulsive features. This factor may relate to conditioning formed through long-term drug use.

3.2 "Stimulus-Response" Association Under Conditioning

During long-term drug use, drug effects become associated with drug-related cues, forming conditioned reflexive behavior—habitual drug use responses and craving under related cues—through conditioning. This transforms reward-controlled goal-directed behavior into S-R habitual drug use behavior and uncontrollable compulsive drug use behavior under craving, becoming important features of addiction. Due to its reflexive, automatic, and unconscious characteristics, the S-R response becomes a crucial drive force for addictive impulsive behavior that addicts' vulnerable response inhibition cannot control (Zeng et al., 2018; 曾红 et al., 2015), thereby developing into addictive behavior

or post-withdrawal relapse.

The neural basis of this "S-R" behavioral response is abnormal connectivity between the dorsal striatum (DS) and related brain regions (Milton & Everitt, 2012). Imaging studies on alcohol addiction show that severe alcohol addicts display higher related cue activation in the dorsal striatum region, while dorsolateral striatum activity intensity in response to related cues positively correlates with drug craving (Wang et al., 2018). Additionally, the prefrontal-dorsolateral striatum-sensorimotor loop composed of the putamen and supplementary motor area (SMA) is significantly activated (Jahanshahi et al., 2015). Under related cues, signals from motor areas project to the putamen and feedback to the frontal lobe via the substantia nigra (SN) region, triggering behavioral responses (Ito & Doya, 2015). The combined action of these three components leads to direct production of habitual drug use behavior under related cues. Therefore, the anatomical connection between the dorsolateral striatum and parietal motor cortex constitutes the neural basis of S-R habitual behavior (de Wit & Phillips, 2012).

Since S-R is the drive for addictive impulsivity, the S in the S-R behavioral structure is the factor forming the drive. Under conditioning, everything associated with drug use and drug effects may become conditioned stimuli for reflexive behavior, triggering conditioned drug use responses. These people, environments, tools associated with drug use, and emotions and stress during substance use become important stimulus sources for addicts' continuous drug use. During long-term drug use, they gradually become drug-related cues; once presented, they stimulate addicts to engage in habitual drug use behavior (王鹏飞 et al., 2019), showing impulsive features.

3.3 Related Factors as Conditioned Stimuli: Negative Emotions and Stress

Stress is a major source of allostatic load, causing long-term, progressive brain changes and triggering drug use tendencies (Emmanuel & Lina, 2018). This is inseparable from stress' s role in neural function. Stress is regulated by corticotropin-releasing factor and other stress hormones, strengthening amygdala function, leading to negative emotional states and enhanced craving (Hiser & Koenigs, 2018), while also weakening hippocampus and prefrontal cortex function, reducing executive control and increasing relapse risk (Andersen, 2019; Ruisoto & Contador, 2019).

Addicts face numerous life events before and after drug use, including unemployment, interpersonal conflicts, family problems, discrimination, etc. These stresses exist chronically in their lives, and they lack appropriate coping methods (曾红 et al., 2015), making them easily become related cues and important factors driving drug use.

Additionally, urgency caused by stress related to positive and negative emotions is a significant feature of addictive impulsive behavior (Ruisoto & Contador,

2019). Research shows that among adolescents, negative urgency is the psychological characteristic most closely related to problematic drug use (Chester et al., 2016). These features mainly manifest as susceptibility to addictive drugs, lowering the threshold for initial drug use. Individuals with these characteristics are more likely to experimentally and recreationally use addictive substances than those without them (Verdejo-Garcia et al., 2008). Once started, low inhibitory control features make self-control impossible, ultimately leading to addiction.

3.4 Personality Traits of Sensation Seeking and Negative Urgency

Impulsive personality traits, beyond inhibitory control deficits, include motivational characteristics such as sensation and novelty seeking. These personality traits increase susceptibility to addictive drugs (Cheng et al., 2015; Everitt & Robbins, 2013). Susceptibility promotes and facilitates initial use of addictive substances and further intensifies with increased use duration. Self-report studies using BIS-11, UPPS-P, and other related questionnaires show that sensation seeking, attentional bias, and other features have stable, reliable correlations with drug use (Argyriou et al., 2018). Additionally, urgency caused by stress related to positive and negative emotions is also a significant feature of addictive impulsive behavior (Ruisoto & Contador, 2019).

4. Interaction Between Inhibitory Control and Drive

In summary, addicts show low levels in both inhibitory control and cognitive control, while showing abnormally high levels in reward, stress, and "S-R" responses. From a behavioral perspective, reward effect, "S-R" response, sensation seeking, and negative urgency may all trigger impulsive drug use; they are drive forces for addictive impulsivity. However, whether these drive forces can directly trigger impulsive drug use depends on users' inhibitory and control features. Imbalance between the two forces affects impulsive drug use. Addicts show low levels in response inhibition and executive control, while showing abnormally high levels in reward effect and "S-R" response due to drug effects and associations—that is, powerful drive forces. Addicts' inhibitory control capabilities cannot regulate this drive, causing imbalance between the two and resulting in uncontrollable impulsive drug use behavior. Thus, impulsivity is formed by the joint action of these driving and controlling factors.

If we compare addiction and relapse to flood disasters, the drive forces from reward effect, conditioning, and stress are the massive, destructive floodwater that is the main cause of flooding; inhibitory control is the dam that can regulate flood disasters. However, if this dam is not strong enough, smaller water flow may not cause disaster, but once the river surges and force increases, flooding will inevitably occur, causing disaster.

Of course, there is also the issue of proportion or weighting of drive forces such as reward effect, life events, and negative emotions versus control forces. However, different individuals have different innate temperament characteris-

tics, addiction severity, and living environments. Therefore, the roles of various factors described in this article in forming addictive impulsivity also differ in magnitude. Currently, there are no weight studies on these factors. However, numerous studies find that addicts' living environment and stress faced after withdrawal are important factors for relapse; but some case reports also indicate relapse is driven by craving for drugs. Therefore, the proportional role of drive and inhibitory forces in causing impulsive behavior should be quite personalized. Perhaps future research can understand this through self-report methods, though self-report results may be potentially affected by social desirability. Neuroimaging results can also serve as an important method for weighting the roles of these different forces, awaiting further in-depth exploration. More effective methods may be comprehensive assessments, including self-assessment, behavioral, and imaging measurements.

From a neural basis perspective, regardless of impulsivity type, it is not the product of structural or functional abnormalities in a single brain region but rather the result of imbalance between drive and inhibitory control circuits, or abnormal functional connectivity (imbalance) between brain regions responsible for drive and control. Imbalance between reward and executive control circuits affects impulsive drug use (Hobkirk et al., 2019). Research shows that addicts display abnormal functional connectivity between specific executive control-related cortices and reward functions (Guan et al., 2015); cocaine abusers show abnormal resting-state brain network coupling in reward-executive control brain regions, related to impulsive decision-making.

The neural mechanism of inhibitory control related to action impulsivity is closely related to the neural circuit from ventrolateral prefrontal cortex (including inferior frontal gyrus IFG)/anterior cingulate cortex to dorsal striatum (caudate nucleus, putamen) (vl-PFC/ACC–Caudate Nucleus & Putamen) (Robbins et al., 2012). Both types of impulsivity involve brain regions related to drive and inhibitory control respectively. These data show that the neural mechanism of impulsivity is not the function of a single brain region but the result of abnormal functional connectivity between brain regions related to drive and inhibitory control psychological phenomena.

Our research shows that heroin addicts display stronger resting-state functional connectivity in the left putamen-precentral gyrus circuit, while showing weaker functional connectivity in the putamen-inferior frontal gyrus (Zeng, et al., 2018), indicating that their corresponding psychological function–habitual (drug use) behavior tendency–is enhanced, while the ability to inhibit this habitual behavior is weakened. This precisely reflects that imbalance between drive force (habitual behavior) and inhibitory control can trigger action impulsivity, manifested as uncontrollable drug use behavior under related cues, causing addiction or relapse after withdrawal.

Conclusion: Addiction Intervention Methods Based on Drive-Control Balance

Reward and S-R responses formed by conditioning become sources of drive for addictive impulsivity, while response inhibition and executive control are control forces. When drive force is sufficiently powerful and inhibitory control is weak, their joint action easily forms impulsive behavior, causing the real-world phenomenon of continuous drug use and relapse. Theoretically, avoiding impulsive drug use behavior can be achieved by reducing drive force or increasing inhibitory control capabilities, reaching balance between the two. However, most current evidence finds that inhibitory control function basically remains unchanged after short-term withdrawal (杨玲 et al., 2020), with no reports on long-term withdrawal yet. Moreover, impulsive personality traits, a psychological phenomenon with genetic nature, are difficult to change in the short term. This means that for addicts, inhibitory control as a factor regulating impulsivity is difficult to fundamentally change, even with long-term withdrawal. Therefore, an important method to help them reduce impulsivity and impulsive behavior should be reducing drive force—that is, reducing reward effect, eliminating S-R associations, or reducing the appearance of corresponding stimuli, which may help addicts reduce drive force and thus reduce their impulsivity for drug use.

If drive force can gradually decrease with treatment, intervention, or prolonged withdrawal time, even if inhibitory control remains at a low level (or partially recovers from drug-induced damage), drive force and inhibitory control can reach balance, avoiding impulsive drug use behavior. That is, as withdrawal time increases and treatment influences, the drive for addictive impulsive behavior weakens or control strengthens, making it possible for addicts to maintain abstinence and prevent relapse even with difficult-to-change impulsive personality traits. Using the flood analogy again, preventing flood disasters requires either reducing floodwater or strengthening dams. However, addicts' dams (inhibitory control) are partly genetic traits (Morein-Zamir & Robbins, 2015), plus long-term drug effects, making them difficult to recover in a short time. Therefore, reducing addicts' impulsivity also needs to consider reducing floodwater—that is, reducing drive force. Combining control enhancement with drive reduction may help addicts reduce impulsive behavior even when control cannot or can only minimally improve. This will have key significance for addiction treatment and relapse prevention and is an operational and applicable method.

Neurocognitive and behavioral combined intervention models have been evaluated and proven to be effective clinical intervention methods (Vassileva & Conrod, 2019). Approach bias modification training based on action cognition theory uses the principle of "action extinction action," targeting habitual drug use behavior triggered by related cues—an important manifestation of addictive impulsivity—to conduct extinction training. By pushing away related cues, it extinguishes existing associations and establishes new "S-R" responses, thereby reducing or extinguishing impulsive responses under related cues during addiction and reducing drug use behavior. This method' s target is precisely the

important drive of impulsivity: "S-R" association. This method has successfully helped alcohol addicts reduce approach bias effect, reduce related brain region activation, and decrease craving and alcohol use behavior (Eberl et al., 2013), and has also achieved positive results in heroin addicts (叶浩生 et al., 2017).

Additionally, Contingency Management (CM) is an incentive-based psychotherapy approach that rewards participants' specific positive behavior changes and is the most effective treatment method for substance addiction (Griffith et al., 2000). Using CM to try to replace drug reward effects with new rewards can reduce or substitute drug reward drive, thereby reducing addicts' impulsive behavior and achieving abstinence maintenance goals. These methods have key significance for addiction treatment and relapse prevention and are operational and applicable methods that can be targeted and combined with other methods for addiction treatment. Of course, their efficacy requires further clinical empirical research.

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