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REVIEW

Differential Contribution of Drug Classes to Impulsive Behaviors in Patients Diagnosed with Substance Use Disorder

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Abstract

Purpose of review This paper synthesizes recent evidence on the differential contribution of drug classes to impulsive behaviors in patients with substance use disorder. It focuses on delineating which domains of impulsivity vary by substance class, which impulsivity dimensions predict treatment outcomes, and the principal neurobiological findings.

Recent findings Recent studies report elevated impulsivity across most substance use disorders with substance-specific patterns. Stimulants, particularly methamphetamine, produce the most pervasive deficits in inhibitory control, planning, and decision-making. Alcohol and opioids are associated with broad decision-making impairments and elevated trait impulsivity that often persist during substitution treatments. By contrast, cannabis shows the weakest and most inconsistent effects. Impulsive decision-making consistently predicts relapse. Neuroimaging implicates frontostriatal circuits, the insula, and the cingulate cortex, with substance-specific differences in regional volume, receptor availability, and network connectivity. Genetic evidence is limited and heterogeneous.

Summary Impulsivity is a transdiagnostic marker with substance-specific profiles that influence both relapse risk and treatment response. Longitudinal, comparative, and network-focused studies employing consensus measures are needed to clarify causality and inform personalized interventions.

Keywords Action impulsivity · Decision making impulsivity · Delay Discounting · Impulsive Choice · Substance use · Trait impulsivity

Introduction

The term impulsivity is widely used in psychology to describe "the inability to stop oneself from engaging in behaviors with negative consequences, a preference for immediate over delayed gratification, a tendency to engage in risky behaviors, and a desire to seek novel sensations" [1]. This construct involves the frontostriatal circuitry, leading to dysregulation of top-down cognitive control [2]. Impulsivity is closely linked to most substance use disorders (SUDs), functioning both as a vulnerability factor and as a potential consequence of chronic substance use [3, 4].

The study of impulsivity is inherently complex, as it represents a multidimensional construct that manifests in various forms [5, 6]. Consequently, a variety of theoretical approaches have been proposed, encompassing both personality-related and neuropsychological factors. Among the most widely used models are Barratt's theory, which distinguishes three subcomponents—attentional, motor, and non-planning impulsivity [7]—and the Five-Factor Model of Personality, which identifies five facets: lack of premeditation, lack of perseverance, sensation seeking, negative urgency, and positive urgency [8]. Despite the validity of these models, subsequent studies have noted a considerable overlap among their components [9]. A complementary line of research has examined impulsivity from a neurocognitive perspective, using laboratory-based paradigms. From this standpoint, impulsivity is typically divided into at least two processes: action impulsivity (Rapid-Response Impulsivity, RRI), deficits in response inhibition and decision-making

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impulsivity or choice impulsivity (a preference for immediate rewards) [9–11]. Three methodological and conceptual distinctions are essential for interpreting the impulsivity literature: (a) trait versus state impulsivity, where trait impulsivity refers to relatively stable personality dispositions and state impulsivity reflects transient fluctuations associated with context, intoxication, or withdrawal; (b) self-report versus behavioral measures, whereby self-report instruments (e.g., BIS-11, UPPS-P) index perceived dispositional tendencies, whereas behavioral tasks (e.g., Stop Signal Task, Go/No-Go, delay discounting) capture performance-based control and choice processes; and (c) action impulsivity versus choice/decision-making impulsivity, with the former indexing failures of response initiation or inhibition (response action initiation [RAI] vs. stopping of an ongoing action [SOA]) and the latter reflecting preferences for immediate or risky outcomes (e.g., delay and probabilistic discounting, Iowa Gambling Task, Balloon Analogue Risk Task) [12–14]. To enhance conceptual clarity in the present review, we adopt an operational taxonomy distinguishing three domains: (1) trait impulsivity, assessed via self-report measures such as the BIS-11 and UPPS-P; (2) action impulsivity, or rapid response impulsivity (RRI), subdivided into RAI ("refraining from action initiation") and SOA ("stopping an ongoing action"); and (3) decision-making impulsivity, encompassing delay discounting, probabilistic discounting, and risk- or feedback-based tasks [12–14]. Risky decision-making is considered a subtype of impulsivity, as it shares core reward devaluation processes and demonstrates sufficient empirical and neurobiological overlap to warrant its inclusion within the broader impulsivity construct [14–16].

Other authors, drawing on integrative frameworks such as the Research Domain Criteria (RDoC) [17], have emphasized the relevance of the cognitive control domain—which regulates cognitive and emotional systems—and the positive valence domain, which modulates reward valuation processes in addiction [18]. Consistent with this integrative approach, the Addictions Neuroclinical Assessment (ANA) framework has been proposed as a neuroscience-informed heuristic model designed to address the clinical heterogeneity and etiological complexity of substance use disorders (SUDs) [19, 20]. This approach is grounded in the premise that dysfunctions in impulsivity and compulsivity constitute core functional components of SUDs. The model links three principal neurofunctional domains to distinct phases of the addiction cycle: incentive salience, associated with the binge/intoxication stage; negative emotionality, characteristic of the withdrawal stage; and executive function, related to the preoccupation/anticipation stage [21]. Within the ANA framework, impulsivity plays a central and multidimensional role. Evidence from deep phenotyping studies suggests that multiple dimensions of impulsivity converge within the broader construct of executive dysfunction [20, 22]. Moreover, impulsivity is consistently associated with substance-related motivation and the internalization of negative affect, positioning it as a key integrative factor across neurofunctional domains [20].

Studies examining the relationship between impulsivity and drug use have yielded consistent results across both behavioral and self-report measures. For instance, Stroop task performance has revealed significant differences between alcohol [23, 24] and healthy controls. Likewise, higher scores have been reported on the Barratt Impulsiveness Scale (BIS-11) among cocaine users [25] and on the Impulsive Behavior Scale (UPPS) [8] among individuals with alcohol use disorder (AUD) [26]. These results are thought to reflect neuropsychological alterations induced by chronic substance use, particularly within the executive control network, as a consequence of striatal D2 receptor loss or direct prefrontal cortex damage [27]. However, alternative interpretations have been proposed—most notably that trait impulsivity may constitute a predisposing vulnerability to substance use [5].

Although addiction is generally associated with elevated impulsivity, distinct patterns emerge depending on the substance class. For instance, stimulants such as cocaine or methamphetamine are primarily linked to deficits in action impulsivity [4, 10]. In contrast, evidence connecting cannabis dependence with heightened impulsivity remains limited and inconsistent [28, 29]. Individuals with alcohol use disorder typically display high levels of trait impulsivity, which correlate with various behaviors linked to alcohol consumption [30]. Alcohol use has also been linked to increased cognitive impulsivity—including deficits in planning, cognitive inhibition, and decision-making—while action inhibition (RAI/SOA) appears to remain relatively intact [29, 30]. However, several meta-analyses have reported that the association between laboratory-based measures of impulsivity and alcohol use shows small effect sizes, except for the Iowa Gambling Task (IGT) and the Stroop Color–Word Test [29, 31]. Regarding opioids, these substances have been associated with decision-making impairments [32]. However, some authors argue that current evidence remains insufficient to draw firm conclusions about the role of impulsivity in opioid addiction [29].

One of the principal limitations in the literature examining the differential effects of individual substances on impulsivity is the extremely high prevalence of polysubstance use, which represents the clinical norm in approximately 70–95% of patients [33]. This substantially complicates efforts to identify substance-specific effects, as pharmacokinetic interactions create a unique biological context and can produce additive or synergistic structural brain damage

[34], resulting in neurobiological alterations that cannot be attributed to a single substance. A similar challenge arises from psychiatric comorbidity, as individuals with substance use disorders exhibit rates of co-occurring psychiatric conditions that are 10 to 20 times higher than those observed in the general population [33]. This overlap further constrains interpretability, given that several psychiatric disorders independently influence impulsivity, thereby hindering the isolation of substance-specific effects [35, 36]. Despite these methodological challenges, the inclusion of samples characterized by polysubstance use and psychiatric comorbidity is unavoidable. Excluding these features would yield models that fail to capture the true complexity and consumption patterns of real-world clinical populations, ultimately limiting the ecological validity of addiction research [33, 37].

While numerous studies have consistently demonstrated a link between impulsivity and substance use, the present review synthesizes research from the past five years, focusing on the differential contribution of major drug classes—including depressants, stimulants, opioids, cannabinoids, and hallucinogens—to impulsive behaviors in individuals diagnosed with substance use disorder. To provide a concise overview of the methodological rigor and strength of the evidence across the reviewed studies, we included three supplementary tables examining study design and methodological limitations (Table 1), the primary constructs assessed by the most commonly used tasks and scales (Table 2), and patterns of psychiatric comorbidity and polysubstance use (Table 3).

Differential Effect of Impulsivity Across Substances

Recent literature on impulsivity in SUDs highlights a complex interplay of effects. Impulsivity consistently emerges as a robust transdiagnostic vulnerability shared across substance classes, while distinct substance-specific profiles have also been reported, particularly within the decision-making domain. However, findings remain inconclusive for cannabis, and to date, no studies have been identified that directly examine this relationship in users of hallucinogens.

Strong transdiagnostic effects have been consistently reported for trait impulsivity, with patients across substance classes exhibiting significantly higher scores than healthy control groups [38–41]. Elevated scores on self-report measures (e.g., BIS-11, UPPS-P) have been documented across a wide range of substance-using populations, including methamphetamine and polysubstance users [39, 42], cocaine/crack users [40, 43], amphetamine users [44], nicotine users [45–47], heroin and GHB users [41], heroin users undergoing methadone maintenance treatment [48, 49], tramadol users [50], and individuals with alcohol use disorder [51]. In the broader literature, trait impulsivity has been conceptualized as both a vulnerability phenotype and a behavioral endophenotype in SUDs, suggesting the presence of a shared underlying susceptibility across addictive disorders [52].

This characterization may reflect the fact that trait impulsivity exhibits structural neurobiological correlates across multiple diagnoses. Specifically, elevated self-reported impulsivity has been associated with alterations in prefrontal cortical circuits across diverse clinical populations, suggesting a common disruption of neural mechanisms underlying attention regulation and cognitive control [43, 53]. Together, these findings provide strong support for characterizing trait impulsivity as a transdiagnostic construct.

Delay discounting has also been identified as a robust transdiagnostic process across psychiatric disorders [54], with compelling evidence supporting its role as an endophenotype of SUDs more broadly [52, 55]. In this regard, the reviewed studies indicate that individuals with SUDs consistently exhibit significantly steeper delay discounting than healthy controls in cocaine use disorder (CUD) [56, 57], opioid use disorder (OUD) [58–60], and alcohol use disorder (AUD) [61]. Findings are less consistent for nicotine use, with mixed results reported across studies [45, 54, 62–64]. Alterations in this domain have been linked to reduced activation within executive control networks [54, 57].

Furthermore, impairments in risk-based decision-making, as assessed by tasks such as the Iowa Gambling Task (IGT) and the Cambridge Gambling Task (CGT), have been consistently observed among individuals with alcohol use disorder [65–69], opioid use disorders [47, 50, 59, 65, 70, 71], cocaine use disorder [72, 73], and methamphetamine/amphetamine use [74–77] compared with healthy controls. However, it remains unclear whether risk-based decision-making per se constitutes a truly transdiagnostic component of addictive disorders. For instance, a theoretical framework on decision-making [78] classifies these tasks (IGT, CGT, BART) as "generic" and "multidetermined" measures, given that performance is influenced by multiple underlying subprocesses, including inhibitory control, cognitive flexibility, and reward sensitivity. Accordingly, these authors argue that a more informative approach is to focus on the assessment of these specific cognitive processes as the genuine transdiagnostic features underlying addiction-related decision-making deficits [78, 79].

Although these transdiagnostic characteristics are robust and supported by multiple studies consistently reporting elevated impulsivity across substances, we also identified differentiated profiles associated with specific substances, particularly in decision-making and trait impulsivity. Nevertheless, the strength of these conclusions is inherently

limited, and any findings suggesting substance-specific effects should be interpreted with caution. Polysubstance use is highly prevalent in the reviewed studies, and most investigations are unable to isolate the effects of a single substance or adequately control for psychiatric comorbidity, which may partially mediate the observed associations. This methodological reality is essential for interpreting the results presented in the following sections. To facilitate readability and avoid redundancy, the remainder of this manuscript will not repeatedly specify when individual findings lack methodological robustness due to polysubstance use or psychiatric comorbidity, and this limitation should be considered implicit throughout.

Evidence of substance-specificity has emerged within the impulsive choice/decision-making domain when examined using advanced methodologies such as computational modeling and cross-commodity tasks [61, 80]. When delay discounting rates were compared across substance classes—including alcohol, cannabis, opioids, and stimulants—using cross-commodity paradigms in individuals undergoing treatment for SUDs [61], distinct substance-specific patterns emerged. Specifically, alcohol users exhibited the lowest discounting rates when the drug reward was immediate, whereas opioid users showed the lowest discounting when the drug reward was delayed. These findings should be interpreted with caution, as they are based on a single study and are subject to several methodological limitations. In a related line of evidence, drug-related cues have been shown to increase preferences for immediate rewards and to reduce the subjective value of future rewards in individuals with heroin addiction [60]. If replicated, these findings would underscore the complex and context-dependent nature of the relationship between substance type and decision-making impulsivity.

In risk-based decision-making tasks, observed deficits appear to be driven by distinct underlying mechanisms depending on the substance involved. In opioid use disorder (OUD), a greater tolerance for ambiguity or unknown risk—but not for known risk—has been associated with a higher likelihood of relapse during the first four weeks following treatment initiation [80]. In contrast, individuals with cocaine use disorder (CUD) have been shown to exhibit impaired integration of gain, loss, and risk information, with reduced sensitivity to gains playing a particularly prominent role in maladaptive risky decision-making [73]. In methamphetamine dependence, several studies have linked impulsive decision-making to reduced dopamine D2 receptor availability [76] and to a diminished tendency to avoid losses [75]. Additionally, these individuals demonstrate poor responsiveness to contingency management interventions [77] and reduced sensitivity to negative feedback during decision-making tasks [74].

Although deficits in action impulsivity are observed across all substance use disorders (SUDs), their magnitude and expression vary substantially across substances. Stimulant users (methamphetamine or cocaine), as well as polysubstance users including methamphetamine, exhibit particularly pronounced impairments in impulsive action [39,81], especially in relation to motor perseveration. In the case of cocaine use disorder, one study reported that the severity of these deficits was associated with longer duration of addiction and may diminish with abstinence [82]; however, the behavioral effect size was small, and the finding derives from a single study. In opioid use disorder, individuals receiving buprenorphine/naloxone treatment continue to show persistent impairments that do not differ significantly from those observed in active users [83]. These deficits may persist even following long-term methadone maintenance, particularly among individuals with a history of relapse [83]. In addition, drug-related cues influence task performance in this domain, with short-term abstinent heroin users committing more errors than those with longer periods of abstinence [84]. Tobacco smokers display a distinct pattern of action impulsivity, modulated by craving intensity or exposure to smoking-related cues [85]. No significant differences have been reported between tobacco and cannabis users, although both groups tend to perform worse than healthy controls [46]. However, these conclusions regarding tobacco use are based on single studies and therefore require further empirical confirmation. With respect to cannabis use alone, evidence suggests a relatively milder impairment in response inhibition. Although some studies indicate that motor impulsivity (BIS-11) represents the most elevated trait impulsivity subtype in cannabis use disorder [46,86], one lacked a control group [86], and the other included a very small cannabis-using sample [46], limiting the strength of these conclusions.

These substance-related differences show limited robustness, particularly with respect to the precise quantification of action impulsivity deficits in substances such as cannabis [46,86,87], due to inconsistent findings across studies. Notably, in cannabis use disorder, impairments appear to be primarily confined to the action impulsivity domain, with comparatively less evidence for alterations in other impulsivity-related domains.

Substance-specificity in trait impulsivity appears to manifest primarily in terms of magnitude and impulsivity profile. Addiction to stimulants, such as methamphetamine and amphetamine, has consistently been associated with the highest levels of trait impulsivity [38,42,88,89], even when compared with dependence on other substances, including cannabinoids, alcohol, benzodiazepines, or polysubstance use not involving methamphetamine [39]. This conclusion is further supported by the fact that several of these

studies relied on monosubstance-using samples [38,88] and moderately sized cohorts [39,90], although they are limited by gender bias, with samples that were almost exclusively male. Notably, this heightened impulsivity appears to be pervasive and persistent, remaining elevated even after long-term abstinence, which supports its characterization as a stable trait marker in stimulant-dependent populations [80]. Evidence for other substance-related differences is comparatively moderate. Cigarette smoking has been uniquely associated with higher trait impulsivity compared with cannabis use alone [46]; however, as this finding is based on a single study, the evidence remains insufficient and warrants replication. In opioid users, trait impulsivity tends to fall within an intermediate range, with impulsivity subtypes differing from those observed in stimulant dependence. In this population, vulnerability appears strongly linked to emotion-based impulsivity, as evidenced by elevated positive and negative urgency, as demonstrated in a monosubstance use study [91]. Supporting this pattern, a study using samples without other SUD diagnoses or psychiatric comorbidity found that both active heroin users and those recently abstinent exhibited higher trait impulsivity than controls, with no significant differences between the two groups [70]. With respect to alcohol use disorder, overall trait impulsivity appears comparable to that observed in opioid dependence [65,92] and lower than that reported in cocaine use disorder [81]. Within this group, motor impulsivity and non-planning impulsivity, as assessed by the BIS-11, are particularly elevated [51].

The available findings—although largely derived from individual studies and therefore requiring cautious interpretation—suggest that impulsivity may vary across substances as a function of personal and familial history. One study identified a strong association between impulsivity and a history of sexual abuse among cocaine and polysubstance users, whereas this relationship was absent in alcohol users [81]. In contrast, a family history of alcohol abuse has been identified as a clinical marker of elevated trait impulsivity in individuals with AUD [93], based on a study with an adequately sized sample ($n = 197$) and robust statistical control of relevant covariates.

In conclusion, impulsivity in SUDs demonstrates a strong transdiagnostic foundation, characterized by generalized impairments in action impulsivity and steeper delay discounting, both of which are associated with structural alterations in frontostriatal circuitry that appear to be shared across substances [43, 53]. At the same time, substance-specific differences are evident, particularly within the decision-making domain, where reward preferences vary as a function of drug type [61, 80], and in the markedly elevated magnitude of trait impulsivity observed in stimulant users [39].

Impulsivity and Treatment Outcomes

The reviewed literature indicates that different facets of impulsivity exert distinct effects on treatment outcomes, with these effects varying according to the substance of abuse. Although available evidence is largely limited, often relying on single studies and samples with insufficient control for polysubstance use or psychiatric comorbidity, these findings are broadly consistent with conclusions drawn in the previous literature. Among these, decision making impulsivity has consistently emerged as the strongest predictor of relapse, most notably among alcohol users, but also in stimulant and, to a lesser extent, opioid users.

In individuals with AUD, a high risk preference measured by the Balloon Analog Risk Task (BART) [68] or the Risk-Based Decision-Making Task [94] predicts relapse within the first months post-treatment. Similarly, poor performance on the IGT has been shown to predict relapse in smokers [95], although this finding is based on a single study and therefore provides limited evidential strength. Additionally, reduced evidence accumulation in the Probabilistic Reward Task [32] and inconsistent intertemporal choices in an Intertemporal Choice Task (ICT) using Bayesian modeling of discounting and consistency [47] have also been associated with relapse in two studies with single-substance users that excluded patients with neurological disorders, mood disorders, and psychotic symptoms.

Among methamphetamine users, maladaptive decision-making on the IGT has been associated with poorer abstinence outcomes [77] and weaker responses to contingency management during treatment. In this context, individuals who continued using tended to favor larger but less frequent rewards, whereas those who maintained abstinence preferred smaller, more frequent rewards with fewer losses [77]. However, these conclusions are not very consistent because the study relied on very small samples and involved polysubstance use of alcohol and cannabis. Furthermore, a study involving cocaine users found no relationship between IGT performance and treatment outcomes [96]. Regarding heroin use, a study employing the Risky Decision-Making Task with computational modeling of ambiguity and known-risk tolerance found that greater tolerance for ambiguity or unknown risk—but not for known risk—was associated with a higher likelihood of relapse during the first four weeks following treatment initiation [80]. Importantly, associations reported in this area [77, 80, 96] are derived from studies characterized by systematic polysubstance use (particularly alcohol and cannabis) and, in the case of cocaine use disorder, frequent comorbidity with attention-deficit/hyperactivity disorder (ADHD). These limitations warrant cautious interpretation. Nevertheless, converging evidence suggests that the effectiveness of interventions

for substance use disorders is enhanced when treatments are tailored to the patient's specific impulsivity profile [97]. Among impulsivity domains, decision-making impulsivity appears to be one of the most modifiable, likely reflecting its stronger state-dependent component [98, 99]. In this context, evidence suggests that patients presenting with impairments in risk- or feedback-based impulsivity may particularly benefit from interventions such as Goal Management Training (GMT) [100] or Contingency Management (CM) [97], both of which have demonstrated consistent efficacy in reducing impulsive choice behavior.

Evidence regarding the relationship between action impulsivity and treatment outcomes is relatively limited, although it appears to be associated with relapse in both alcohol and stimulant use. Patients with alcohol use disorder (AUD) who relapsed showed poorer inhibitory control on the Emotional Go/No-Go task [94], whereas better inhibitory performance on the Stop-Signal Task (SST) was associated with longer abstinence periods among cocaine users [82]. Notably, whereas the study conducted in alcohol users did not specify whether participants engaged in other substance use or presented psychiatric comorbidities and relied on a small sample size ($n=41$), the study involving cocaine users implemented rigorous methodological controls for these variables and included a larger sample ($n=115$), albeit with a small behavioral effect size. More broadly, the literature indicates that action impulsivity is a domain amenable to modification, and that interventions targeting motor inhibition and behavioral regulation, such as Inhibitory Control Training and Cognitive Remediation Programs, show the greatest efficacy in reducing substance use among patients with elevated levels of action impulsivity [6, 101]. The relationship between delay discounting and treatment outcomes in stimulant users varies considerably across specific substances. Lower decision-making impulsivity in delay discounting measures—such as the Monetary Choice Questionnaire (MCQ) [102]—has been associated with higher smoking cessation rates in a study with strong longitudinal evidence, appropriate control of multiple covariates, and an adequate sample size. A similar association was reported in another study using an fMRI-based delay discounting task [54]. However, other investigations have failed to replicate this relationship, with one study reporting no significant association [64] and a further study in cocaine users likewise finding no relationship between delay discounting and treatment outcomes [96]. Importantly, many of these studies do not adequately report psychiatric comorbidity or rely on samples characterized by polysubstance use, particularly involving nicotine and alcohol, which limits the specificity and interpretability of the findings. Despite this heterogeneity, delay discounting appears to be a modifiable impulsivity domain in individuals with SUDs [100]. The literature indicates that interventions based on Episodic Future Thinking reliably reduce temporal discounting and decrease the subjective valuation of substances [103]. In addition, Contingency Management interventions have been shown to reduce delay discounting and to improve abstinence outcomes [97].

Analysis of trait impulsivity indicates that high scores predict relapse in alcohol use, whereas evidence for stimulant users remains inconsistent. Two studies found that BIS-11 scores predicted early relapse in AUD [94, 99], however, both studies included samples with psychiatric comorbidity and use of other substances, which could confound the association. Another study, with a large sample (n=304), identified an indirect relationship between trait impulsivity and alcohol relapse, showing that higher rash impulsivity (measured by the Dysfunctional Impulsivity Scale) increased craving vulnerability, which in turn elevated relapse risk [104]. Findings concerning trait impulsivity and tobacco use are contradictory. One study with single-substance users and comorbidity control reported that both positive and negative urgency on the UPPS-P predicted lower cessation success and smaller reductions in smoking [105]. In contrast, other studies found no significant associations between relapse and scores on the same scale [96, 54], the BIS-11 [94], the Eysenck Impulsivity Questionnaire, or the Sensation Seeking Scale [96]. Regarding cocaine use, one study in monosubstance users with rigorous control for severe comorbid conditions concluded that trait impulsivity, as measured by the BIS, was associated with poorer treatment outcomes among White adults, but not among Black adults [106]. However, this evidence is derived from a single study and therefore has limited generalizability. Trait impulsivity has been consistently described as the most stable component of the impulsivity construct, reflecting a deeply rooted behavioral predisposition [5,107], and is consequently difficult to modify. Nonetheless, several "third-wave" interventions, including Dialectical Behavior Therapy (DBT) skills training, mindfulness-based interventions, and Acceptance and Commitment Therapy (ACT), appear to be promising options for individuals with SUDs and high trait impulsivity, given their impact on the emotional and neurocognitive processes underlying impulsive behavior [108,109].

Although a small number of studies have examined the relationship between impulsivity and quality of life in patients undergoing treatment for substance use disorders (SUDs), the conclusions drawn thus far rely on single-study evidence and should be considered preliminary. In a study involving individuals with AUD, trait impulsivity measured by the BIS-15 was associated with improvements in health-related quality of life and well-being during the early weeks of detoxification—possibly exerting an even stronger influence than consumption patterns themselves

[110]. Similarly, trait impulsivity assessed using the BIS-11 has been shown to predict all components of quality of life—physical, psychological, social, and environmental—among methamphetamine users, even after controlling for other clinical variables [49]. Moreover, the high prevalence of psychiatric comorbidity in the alcohol study and poly-substance use in the methamphetamine study may have distorted the observed associations, thereby further limiting interpretability.

In opioid users, research has examined whether impulsivity persists after prolonged abstinence or treatment, with findings suggesting that such deficits tend to endure over time. Accordingly, while opioid substitution therapy and related treatments contribute to clinical stabilization, harm reduction, and facilitation of abstinence, this does not necessarily imply a normalization of all state-dependent impulsivity domains. For instance, a study comparing active opioid users with patients undergoing methadone maintenance treatment found that, despite clinical improvement, deficits in specific impulsivity-related processes persisted during treatment [84]. Similar results were reported for active users and those treated with buprenorphine/naloxone, as decision-making impairments and action impulsivity — measured with the Cambridge Gambling Task (CGT) and Stop-Signal Task (SST)—persisted in the treatment group [70]. Another study employing a battery of tasks, including the Iowa Gambling Task (IGT), CGT, Monetary Choice Questionnaire (MCQ), Go/No-Go Task, and SST, found that the duration of abstinence was minimally related to impulsive decision-making and unrelated to other forms of impulsivity [58]. Importantly, the conclusions of these latter studies rest on a more solid empirical foundation, as they were based on monosubstance-using samples and applied strict exclusion criteria for psychiatric comorbidity. Consequently, it would be advisable to integrate targeted interventions addressing state impulsivity in opioid-dependent patients in order to reduce relapse risk. This recommendation is likely also applicable to alcohol and stimulant use disorders, where decision-making impairments have likewise been shown to persist during abstinence [29, 111].

Genetic and Neurobiological Studies

Available evidence on the biological basis of impulsivity in SUDs points to a shared neurogenetic substrate, albeit with substance-specific manifestations. However, few studies have directly examined the genetic correlates of impulsivity in SUDs. One recent investigation identified the OPRM1 SNP rs495491 as being significantly associated with trait impulsivity — specifically the motor subfactor of the BIS-11— suggesting that genes related to opioid receptors, such as OPRM1and OPRK1, are implicated in impulsivity [112]. In contrast, other studies reported no significant associations between impulsivity domains and IL-10 (−819 C/T), TNFA (−308G/A), or ENOS (−786T/C) polymorphisms among cocaine and crack users [40], nor between impulsivity and the TAAR1 rs8192620 genotype in methamphetamine and heroin users [113]. The limited focus on genetic factors and the heterogeneity of findings stand in contrast to earlier literature emphasizing a genetic contribution to impulsivity in addiction [5, 114]. The available genetic findings derive from isolated investigations and should therefore be interpreted with caution, as single-study evidence limits the robustness of proposed gene–behavior associations.

In contrast to the limited genetic literature, neuroimaging studies provide a broader and more diverse body of evidence, although most research has primarily focused on stimulants, alcohol, and opioids. Overall, the most consistent neural alterations have been identified in the prefrontal cortex, insula, anterior cingulate cortex, nucleus accumbens, and striatal regions (including the caudate and putamen) [Tobacco: 62, 54; Cocaine: 73, 114, 115; Methamphetamine: 42, 74, 76, 88, 116; Alcohol: 94, 117, 118; Tramadol: 50; Heroin: 119 ; and Opioids in general: 120]. There is a single electrophysiological study, so its generalizability is limited. This study reports that patients using gamma-hydroxybutyrate (GHB), heroin, and methadone exhibited reduced P300 amplitude and increased latency in the central sites and frontal, temporal, and parietal lobes compared with controls—findings associated with elevated impulsivity [41]. These regions largely overlap with those described in earlier literature [e.g., 120].

Previous research has emphasized the difficulty of distinguishing neuroimaging biomarkers by substance type based solely on specific brain regions, suggesting instead that broader functional and structural connectivity patterns may provide a more accurate characterization [121]. Overall, however, the results converge on a pattern of alterations across multiple key neural networks, supporting theoretical models that conceptualize SUDs as brain-based conditions characterized by large-scale circuit dysfunction. In particular, a frontostriatal imbalance appears to underlie disruptions in cognitive control, decision-making, and response inhibition [122–124].

The executive control network (ECN), which is implicated in “cold” executive control and response inhibition, shows consistent functional and structural alterations across multiple SUDs. Reduced gray matter volume has been reported in prefrontal regions—including the ventrolateral and dorsolateral prefrontal cortex and the anterior cingulate cortex (ACC)—in individuals with nicotine dependence [125], tramadol use disorder (with additional reductions in frontal lobe volume) [50], and methamphetamine dependence [88].

Comparable alterations have been observed in the salience network (SN), which regulates attentional allocation to salient stimuli and interoceptive awareness. In a study of individuals with alcohol use disorder (AUD), elevated glutamate levels in the ACC were associated with higher levels of trait impulsivity [118]. Additionally, reduced gray matter volume of the insula has been reported in tobacco users [125] and amphetamine users [42]. The SN, ECN, and reward network (RN) converge anatomically in the ACC, a region that exhibits glucose hypometabolism in heroin users [48] and whose activity has been shown to correlate with impulsivity in tobacco use [125]. In the neuroimaging literature published over the past five years, alterations in the default mode network (DMN) have been reported primarily in stimulant users, such as tobacco [54] and cocaine users [126], which limits broader inferences regarding the role of the DMN across SUDs more generally. In contrast, the reward network (RN)—whose dysregulation is thought to enhance the valuation of drug-related rewards while diminishing sensitivity to natural rewards—has been consistently implicated in AUD [117] and heroin dependence [119]. In the latter, increased nucleus accumbens volume has been associated with higher trait impulsivity. Moreover, in individuals with methamphetamine dependence, reduced striatal dopamine D2 receptor availability has been linked to impairments in risky decision-making [76].

However, substance-specific patterns have also been observed, which may help explain differences in impulsivity profiles across drug types. It is important to emphasize that conclusions attempting to attribute substance-specific neurobiological mechanisms to a single drug in isolation are methodologically less robust, given the high prevalence of polysubstance use among participants in the reviewed studies. Recent findings in stimulant use disorders nonetheless support the hypothesis that impulsivity arises from an imbalance among large-scale neural networks, mediated in part by dopaminergic alterations [, 127, 128]. In line with this view, a study involving CUD patients found greater temporal discounting compared with controls, alongside altered effective connectivity among the executive control network, salience network, and default mode network, as measured by fMRI [114]. Similarly, methamphetamine use has been linked to disrupted brain connectivity [89], which is linked to higher impulsivity. In nicotine users, early onset has been associated with riskier decision-making (riskier choices; steeper delay discounting), and reduced gray matter volume in the insula (IC), as well as in the anterior cingulate cortex (ACC), ventrolateral prefrontal cortex (VLPFC), caudate, and putamen, compared with controls [62, 125]. Another study implicated the dorsolateral prefrontal cortex (DLPFC), medial frontal gyrus, anterior insula, posterior cingulate, caudate, and putamen in delay discounting tasks among smokers [54], although it should be noted that participants might have consumed alcohol and cannabis. However, an fMRI study with strict single-substance use and exclusion of comorbidity [64] reported no significant differences between smokers and non-smokers in ventral striatum, ACC, or DLPFC activity during delay discounting tasks.

The executive central network is the system most consistently reported as altered across studies on stimulant use, however, all studies except one [74] include samples with use of other substances. Studies involving methamphetamine users have likewise reported reduced gray matter volume among the most impulsive patients, particularly in the prefrontal cortex [24]. Furthermore, during drug cue-reactivity tasks, methamphetamine users have shown reduced activation in the medial superior frontal gyrus, inferior frontal gyrus, precentral gyrus, and parietal lobe, reflecting deficits in action impulsivity (response initiation and stopping) [113]. A recent event-related potential study in MUD [74] found a hyporeactive neurophysiological pattern in the ACC, characterized by reduced error-related negativity and feedback-related negativity during negative feedback, compared with controls (given that this is a single study with a small sample ($n = 18$) of justice-involved users, these conclusions should be interpreted with caution). Research on CUD has shown that gray matter volume in the lateral prefrontal cortex correlates with trait impulsivity, while medial prefrontal cortex volume correlates with delay discounting, and posterior parietal cortex volume correlates with both [115]. Altered connectivity within the ECN (right–left) is associated with greater delay discounting in cocaine patients, reflecting impaired cognitive control [57]. Additionally, in individuals with CUD, activity in the bilateral superior temporal gyrus has been found to correlate positively with stop-signal reaction time (SSRT), indicating poorer action impulsivity (stopping; SOA) [82]. Stimulants appear to be the substances that exert the greatest detrimental impact on the central executive network. For example, in cocaine users, reduced gray matter volume has been detected in the right inferior frontal cortex compared with individuals with OUD, potentially underlying the greater action impulsivity typically seen in stimulant users [129].

Additional SN alterations have also been reported in stimulant use disorders, with reduced amplitudes in the anterior cingulate cortex and related regions observed in event-related potential (ERP) components (ERN, FRN) in patients with methamphetamine use disorder (MUD) who did not present polysubstance use or psychiatric comorbidity [74]. Taken together, these findings suggest reduced sensitivity to negative feedback and a propensity toward riskier decision-making, processes that are plausibly linked to an increased attribution of salience to drug-related stimuli. Regarding the

reward network, methamphetamine-related findings include lower D2 receptor availability in the striatum and globus pallidus (RN) and hyperactivation of the amygdala and the nucleus accumbens, which results in impaired behavioral updating in response to feedback and, consequently, maladaptive decision-making [76]. In the same substance group, hyperactivation of the RN (amygdala, insula, and nucleus accumbens) has also been reported during action impulsivity (SOA) tasks [42]. Turning to another network, it has been observed that D2 receptors mediated the relationship between default mode network (DMN) suppression and Stroop performance in healthy controls, whereas D3 receptors mediated this relationship in patients [126]. This pattern suggests a potential compensatory mechanism, in which increased D3 receptor activity may partially offset D2 receptor deficits in individuals with CUD. However, all findings concerning dopamine D2 and D3 receptor alterations should be interpreted with caution, given the presence of polysubstance use in several samples [42, 76, 127] and the inclusion of participants with uncontrolled affective and anxiety disorders in some studies [42].

With respect to alcohol use disorder, neurobiological evidence similarly supports its conceptualization as a disorder of large-scale circuit dysfunction, characterized by frontostriatal imbalance. Most studies in this area, however, relied on samples with polysubstance use and provided limited control for psychiatric comorbidity. Several studies have reported alterations in ECN. Alterations in gray matter have been described — notably in the supplementary motor area — and white matter deterioration in the corpus callosum and association tracts, together with shorter deliberation times compared with controls [69]. Additionally, transcranial magnetic stimulation (TMS) studies have revealed altered cortical inhibition, specifically impaired suppression of motor-evoked potentials, and differences in trait impulsivity among controls, heavy drinkers, and patients with severe AUD [26]. Regarding the SN, changes in oxyhemoglobin concentration within the right frontotemporal cortex have been observed when comparing abstinent and relapsed alcohol users during an emotional Go/No-Go task. Individuals who relapsed showed lower activation in these regions relative to abstinent participants, suggesting deficits in emotional regulation circuits involved in action impulsivity (emotionally modulated RAI/SOA) [94]. With respect to the reward network, functional connectivity analyses using fMRI in individuals with AUD have revealed differences relative to controls, indicating alterations in reward-related neural networks [51, 130]. A relationship between nucleus accumbens volume and trait impulsivity has also been reported [117]. Notably, this study applied rigorous exclusion criteria for Axis I psychiatric disorders, although tobacco smokers were not excluded, which may have influenced the findings. In addition, the SN appears to be affected during emotional inhibition tasks, showing reduced activation that limits the detection of behaviorally relevant stimuli [94]. Moreover, elevated glutamate levels in the thalamus and dorsal anterior cingulate cortex (ACC) have been linked to higher levels of trait impulsivity [118]. Collectively, these alterations suggest a neural profile characterized by reduced executive control and increased influence of pathological reward-related and self-referential processes.

In opioid use disorders, a broadly shared pattern of ECN dysfunction has been reported, although with substantial variability depending on the clinical subtype. In patients undergoing methadone maintenance treatment, glucose hypometabolism in the prefrontal cortex, ACC, striatum, and thalamus has been associated with deficits in probabilistic decision-making [48]. In contrast, individuals with OUD who presented social impairment and a history of polysubstance use during the previous year showed increased activation of dorsolateral and ventrolateral prefrontal regions during impulse control tasks [120]. This pattern has been interpreted as a compensatory mechanism in response to underlying deficits in cognitive control. Reduced frontal lobe volume has been reported in tramadol users, correlating with higher impulsivity levels [50], however, this study relied on a very small sample ($n=12$) that also involved polysubstance use, including cannabis and other opioids. In opioid users, an fMRI study found that poorer cognitive control was associated with lower State Engagement Variability among patients receiving methadone treatment [131]. In heroin users, findings indicate greater nucleus accumbens (NA) volume and enhanced connectivity in the bilateral superior frontal gyrus (SFG), both of which correlate with action impulsivity [49]. It should be noted that conclusions drawn from these opioid studies are based on single, isolated investigations, which limits the strength of the evidence and constrains their clinical and translational applicability.

Overall, the findings indicate that SUDs share a common neurobiological architecture that helps explain the diverse impulsivity profiles observed across substances. These results reinforce the conceptualization of addiction as a disorder of interdependent neural networks, in which circuit-level dysfunction underlies loss of control and compulsive behavior.

Conclusions and Future Directions

The scarcity of comparative studies and the methodological variability across existing research make it difficult to clearly delineate the role of specific impulsivity domains

for each type of substance, further obscured by pervasive polysubstance patterns and unreported psychiatric comorbidities. Among the various drugs examined, cannabis appears to exert the least detrimental effects, particularly in decision-making, where users show a greater ability to delay gratification and process feedback from past behaviors—impairments that are markedly more severe with other substances. Despite these advances, the findings should be interpreted with caution, as studies on cannabis use are frequently confounded by concurrent nicotine and alcohol consumption, which complicates the attribution of substance-specific effects. In contrast, stimulants—especially methamphetamine—are strongly associated with pronounced impulsivity, likely resulting from severe disruptions in dopaminergic and prefrontal functioning , with methamphetamine-related impairments often persisting long-term, even during abstinence. . These substances are also linked to deficits in planning and sustaining goal-directed behavior, which in turn hinder both abstinence maintenance and treatment adherence. These conclusions regarding stimulants and cannabis are consistent with evidence reported in prior literature [28, 29, 87, 131].

Opioids tend to be associated with impairments that persist over extended periods, both during abstinence and substitution therapy, likely reflecting long-lasting neurobiological alterations or neurotoxic effects on the nervous system [119]. Future research should aim to strengthen the evidence in this area to confirm these conclusions with greater robustness and to clarify the mechanisms underlying their persistence. Alcohol use is similarly characterized by deficits overlapping with those observed in stimulant and opioid users, not only in decision-making [29, 87] or trait impulsivity [10], as suggested by earlier studies. These deficits are more pronounced among individuals with an earlier onset of consumption and appear to be linked to family-related factors.

The consistent evidence of elevated impulsivity across all SUDs supports the hypothesis that such behavior may predate addiction, acting as a vulnerability factor for both substance use and dependence [9]. This interpretation is consistent with dual models of addiction, such as the Impaired Response Inhibition and Salience Attribution (iRISA) framework and triadic models of addiction [128]. However, because nearly all studies reviewed employed cross-sectional designs, it remains unclear whether the observed patterns reflect pre-existing impulsivity traits or are a consequence of prolonged substance use and the associated neuroadaptive changes.

Differences in impulsivity have clear clinical implications for the treatment of addiction, underscoring the need for clinicians to understand the specific deficits their patients present—whether related to action impulsivity (RAI/SOA), decision-making, delay discounting, or perseverance. A more precise characterization of the impulsivity profiles associated with each substance would help guide clinical decision-making, improve diagnostic accuracy, and enable a more accurate assessment of the factors that predict treatment success or failure.

Finally, several aspects must be considered when interpreting the results of this review. First, there is a marked imbalance in the representation of substances studied, with a clear predominance of research on alcohol, stimulants, and opioids, whereas no studies were identified involving hallucinogens, likely because classical psychedelics are generally nonaddictive [132]. A strong gender bias is also evident — except in studies involving cannabis — likely reflecting the greater proportion of men in treatment settings, which limits the generalizability of findings. An age bias is likewise apparent: studies involving stimulant and cannabis users tend to include younger participants, whereas older individuals are more commonly represented among alcohol users. Moreover, methodological heterogeneity complicates comparisons across studies, as different instruments are used to assess impulsivity. Because polysubstance use is the norm rather than the exception among individuals with SUDs [34], most of the reviewed studies included participants who used substances in addition to the primary drug of interest—most commonly alcohol, cannabis, and tobacco. This substantially complicates the interpretation of findings by generating unique and unpredictable biological interactions, exacerbating cognitive deficits, and hindering the isolation of substance-specific neurocognitive effects. Nevertheless, the conclusions remain pertinent within the ANA and RDoC frameworks, which prioritize transdiagnostic pathophysiological dimensions and shared genetic vulnerabilities over strict diagnostic categories. A further limitation is that several conclusions rely on isolated studies or small sample sizes. Accordingly, future research should seek to replicate and extend these findings in larger, well-characterized samples to strengthen their consistency, robustness, and clinical applicability. Finally, the predominance of cross-sectional designs precludes establishing firm conclusions about the causal relationship between impulsivity and addiction.

In conclusion, the evidence reviewed in this work suggests that different substance classes contribute in subtly distinct ways to impulsive behaviors and to the moderating factors—including age of onset and duration of abstinence—that influence these patterns. However, studies directly comparing different drug types remain scarce. Future research should address this gap by designing studies that systematically compare impulsivity across various forms of SUD and examine neural network connectivity using consensus-based assessment tools, such as those aligned with the framework

proposed by Yücel et al. [18] within the Research Domain Criteria (RDoC) approach. Given the degree of conceptual and methodological heterogeneity observed in the reviewed literature, subsequent investigations would particularly benefit from being grounded in the Addictions Neuroclinical Assessment (ANA) framework [17, 19]. This approach enables a shift from symptom-based classifications toward a model centered on pathophysiology and deep phenotyping, thereby facilitating the investigation of the etiological heterogeneity of SUDs and the development of precision medicine strategies [18, 20, 22]. In this context, the application of computational modeling to decision-making tasks allows for the dissociation of latent cognitive processes that are not directly accessible through traditional behavioral measures—such as learning rate, loss aversion, and reward sensitivity [28, 133]—and may more effectively discriminate between individuals with different SUD profiles [6, 134]. In parallel, Ecological Momentary Assessment (EMA) has shown promise in capturing the dynamic nature of addiction [135, 136] and may be particularly suitable for investigating state impulsivity with enhanced ecological validity. EMA approaches are already producing valuable insights, particularly for examining the relationship between momentary fluctuations during treatment and relapse risk [137, 138]. Finally, studies focusing on gene–environment interactions, the integration of polygenic risk scores, and imaging genetics are yielding promising findings for identifying vulnerability markers associated with addiction and impulsivity [139, 140].

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  ○ This study aims to compare attentional and inhibitory control functions among individuals with different substance use disorders and healthy controls, using the Attentional Network Task and the Stop Signal Task.

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Data Availability No datasets were generated or analysed during the current study.

Declarations

Competing interests The authors declare no competing interests.

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