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REVIEW

Impulsivity in Alcohol Use Disorder: A Multidimensional Perspective

Müge Bozkurt[1], **Cüneyt Evren**[2]

[1]Department of Psychiatry, Istanbul University Faculty of Medicine, İstanbul, Türkiye
[2]Liman Psychiatry and Psychotherapy Center, İstanbul, Türkiye

ORCID iDs of the authors: M.B. 000-0001-8300-0943, C.E. 0000-0002-4431-3514.

Main Points

- Impulsivity plays a crucial role in the development, maintenance, and escalation of drinking behavior, ultimately leading to alcohol use disorder (AUD).
- Impulsivity may either serve as an underlying vulnerability for AUD or be a consequence of chronic alcohol use.
- Repeated cycles of heavy drinking, withdrawal, and relapse induce structural changes in brain regions related to emotional regulation and higher cognitive functions, which impair self-control and increase impulsive behaviors.
- Impulsivity may serve as an endophenotype for AUD.
- Targeting impulsivity in treatment and prevention, through interventions like cognitive-behavioral therapy and mindfulness-based approaches, shows promise in reducing impulsivity-related drinking behaviors and improving long-term outcomes.

Abstract

Impulsivity is a key construct in the development, maintenance, and escalation of drinking behavior, ultimately leading to alcohol use disorder. Because impulsivity is a multidimensional construct and has many ways of measurement, it is difficult to reach a clear consideration of its association with alcohol. Assessment of impulsive personality traits, impulsive choice, and impulsive action simultaneously in studies is recommended. On the other hand, impulsivity seems to be both a determinant and a consequence of alcohol use disorder. Short- and long-term effects of alcohol are also associated with increased impulsivity, further enhancing the risk for continued alcohol consumption and thus maintaining a cycle of use. Impulsivity is also considered an endophenotype, a heritable trait that can predict the likelihood of developing alcohol use disorder. Impulsivity can be a target for interventions, including cognitive-behavioral therapy and mindfulness-based approaches, to improve impulse control and reduce relapse rates. A comprehensive understanding of its role in alcohol use disorder seems to be important for the development of effective treatment and prevention strategies.

Keywords: Addiction, alcohol, impulsivity

Corresponding Author:
Müge Bozkurt
E-mail:
mugeulku@gmail.com

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Introduction

Impulsivity is defined as a predisposition for rapid, unplanned actions, without considering potential negative consequences (Moeller et al., 2001). As an individual ages and matures, the inhibitory mechanisms that allow them to control their impulses and desires, and act appropriately in the moment, improve. Lack of this development is characterized by novelty seeking, lack of planning, reward dependence, and pleasure seeking, which are indicators of psychiatric disorders like alcohol use disorder (AUD) (de Wit, 2009).

Current theories suggest that impaired impulse control underlies substance-seeking behavior, and together with individual differences in other neurobiological domains such as compulsivity and emotion regulation, plays a significant role in understanding the heterogeneous nature of

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addiction (Vassileva & Conrod, 2019). Impulsivity plays a crucial role in the development, maintenance, and escalation of drinking behavior, ultimately leading to AUD (Sliedrecht et al., 2021). Impulsivity appears to be both a determinant and a consequence of AUD. As a determinant, trait impulsivity is linked to the initiation of alcohol use, continued use, and the inability to quit. Additionally, state impulsivity, or situational impulsive behaviors, also increases the risk of alcohol use. Momentary fluctuations in decision-making or inhibition can pose a significant risk for individuals attempting to abstain from alcohol (de Wit, 2009).

On a neurobiological level, deficits in response inhibition, often due to dysfunctions in the frontal cortex and striatum, contribute to impulsive behaviors, with abnormal frontostriatal connectivity potentially linking impulsivity and addiction (Galandra et al., 2018). Neurochemical imbalances, particularly in dopamine and serotonin signaling, also promote impulsive actions (Sliedrecht et al., 2021). There has been growing support for incorporating impulsivity measures into standard neuropsychological assessments for addiction research and treatment, similar to the MATRICS model used in schizophrenia studies (Yücel et al., 2019). However, the multifaceted nature of impulsivity, the challenges in defining and assessing it, and difficulties in distinguishing between cause and effect have made it hard to fully understand its role in alcohol abuse. Impulsivity may either serve as an underlying vulnerability for AUD or be a consequence of chronic alcohol use (Sliedrecht et al., 2021).

Multifaceted Structure of Impulsivity

Impulsivity is often viewed as a personality trait, referred to as trait impulsivity, which is commonly measured by self-report scales like the Barratt Impulsiveness Scale (BIS) or the Urgency-Premeditation-Preseverance-Sensation Seeking-Positive Urgency (UPPS-P) impulsive behavior scale. Other approaches emphasize neurobehavioral assessments that are more sensitive to momentary changes in impulse control, influenced by environmental contexts and the individual's current state. These situational impulsivity assessments involve performance-based tasks related to short-term versus long-term rewards or the inhibition of prepotent responses (Herman & Duka, 2019). While some argue that performance tasks may reflect underlying trait impulsivity, they are susceptible to variations within the same individual. Therefore, it is suggested that self-report scales reveal general long-term risk, whereas performance tests capture more immediate, situational risks. However, it should also be noted that self-report scales depend on whether people evaluate themselves accurately and whether they have insight. The combination of both assessments offers the most comprehensive evaluation of impulsivity, especially when identifying individuals at risk (Cyders & Coskunpinar, 2011).

From a neurobiological perspective, impulsivity is characterized by a failure to suppress reward-related responses through top-down control mechanisms, leading to a reduction in cognitive or inhibitory control (disinhibition). Inhibitory control involves executive functions, such as working memory and cognitive flexibility, which regulate goal-directed behavior. Thus, impulsivity can be divided into two key dimensions: motor impulsivity, which refers to acting without sufficient thought, and choice impulsivity, characterized by a preference for immediate rewards over future, larger rewards (Verdejo-Garcia & Albein-Urios, 2021). Behavioral tasks like the stop-signal task (SST) and go/no-go (GNG) tasks are used to assess motor impulsivity, while delay discounting tasks evaluate decision-making impairments, where individuals opt for smaller, immediate rewards rather than larger, delayed ones. Although the dimensions of impulsivity are closely related, they appear to represent distinct features (Kozak et al., 2019). On the other hand, some authors conceptualize impulsivity into three categories: 1) reflection impulsivity (a tendency to make fast decisions without adequately accumulating and evaluating information); 2) motor impulsivity (the inappropriate execution of motor actions, defined as the inability to stop a pre-potent motor response, i.e., no longer appropriate, or the inability to wait for an appropriate signal to act); and 3) temporal impulsivity (a difficulty in delaying gratification) (Herman & Duka, 2019).

Barratt's model of impulsivity categorizes impulsivity into motor, attentional, and non-planning dimensions, while the UPPS-P model includes urgency, lack of perseverance, lack of premeditation, and sensation-seeking (Kozak et al., 2019; Verdejo-Garcia & Albein-Urios, 2021). Recent studies suggest that sensation-seeking may represent a distinct aspect of impulsivity (Mitchell & Potenza, 2014). Although all dimensions of impulsivity appear to be close, they are thought to represent separate characteristics.

Impulsivity as a Vulnerability Factor

Research consistently demonstrates a strong link between impulsivity, sensation seeking, and addiction (de Wit, 2009). Impulsive behaviors often contribute to the early stages of recreational substance or alcohol use (Verdejo-García et al., 2008). Repeated alcohol use, along with failed attempts to quit, can be attributed to insufficient inhibitory control over the immediate reinforcing effects of alcohol. High sensation seekers may be drawn to alcohol use due to its novelty and stimulating qualities, while individuals with high positive urgency may use substances to prolong positive affect (Verdejo-García et al., 2008). These tendencies often lead to an underestimation of alcohol's negative consequences and an exaggeration of its positive effects (Stautz & Cooper, 2013). Additionally, impulsive decision-making is characterized by choosing the immediate euphoria from alcohol use over long-term social, personal, educational, and economic success (de Wit & Richards, 2004).

It has been found that all five dimensions of impulsivity, as assessed by the UPPS model, are predictors of alcohol use frequency and alcohol-related problems (Jones et al., 2021). Individuals with higher levels of negative urgency may be particularly vulnerable to shifts in emotional states and may drink alcohol as a maladaptive strategy for mood regulation. Furthermore, the increased craving for alcohol following exposure, driven by negative urgency, may perpetuate the cycle of alcohol-seeking behavior (VanderVeen et al., 2016). A study conducted with adolescents found that deficits in planning were associated with alcohol-related problems and illegal substance use (Martinez-Loredo et al., 2018). It is not a coincidence that the initiation of substance experimentation typically occurs during adolescence, a period when the prefrontal cortex (PFC) and inhibitory mechanisms are not yet fully developed (Chambers et al., 2003). Impulsivity is also hypothesized to mediate the increased risk of substance use

disorder (SUD) and AUD in individuals with childhood disorders such as attention deficit hyperactivity disorder (ADHD), conduct disorder, or oppositional defiant disorder (Ortal et al., 2015).

Studies have demonstrated that the relationship between impulsivity and AUD is neither entirely linear nor simple. For instance, social motivations such as positive attributions about alcohol use, viewing drinking as exciting, or believing that alcohol facilitates socialization influence the relationship between impulsivity and alcohol consumption (Verdejo-Garcia & Albein-Urios, 2021). In addition, negative emotions or stressors also play a role in this relationship. For example, cumulative stress combined with high impulsivity can predict problematic alcohol use among adolescents (Fox et al., 2010). These findings indicate that, alongside impulsivity, factors related to alcohol use—such as peer relationships, stressors, and expectations surrounding substance use—are also significant determinants of AUD (Verdejo-Garcia & Albein-Urios, 2021).

Impulsive Decision-Making and Alcohol Use Disorder

Decision-making processes involve the interaction between prefrontal cognitive circuits and subcortical affective circuits (Bickel et al., 2012; Hoffman et al., 2008). The affective system, which includes the ventral striatum, amygdala, and anterior insular cortex, is responsible for evaluating attributed value, such as internal motivation and reward, and is particularly activated by immediate rewards (Bickel et al., 2012). In contrast, the cognitive circuit, comprising the anterior cingulate and dorsolateral PFC, evaluates consequences and options, becoming active when an individual opts for a delayed reward (Hoffman et al., 2008). In healthy individuals, the interaction between these systems leads to effective decision-making. However, in individuals with SUD, the cognitive system is hypoactive while the affective system is hyperactive, resulting in impulsive choices (Hoffman et al., 2008).

Impulsive decision-making can be described as a preference for immediate rewards regardless of future consequences. This involves heightened sensitivity to immediate rewards and insensitivity to delayed outcomes (de Wit, 2009). In the context of alcohol use, this manifests as increased sensitivity to the rewarding effects of alcohol, such as instant euphoria or relief from withdrawal, while being indifferent to long-term negative consequences, such as loss of employment, family, or friendships (Poulton & Hester, 2020). Impulsive decision-making is typically measured using delay discounting tasks, which assess how much an individual prefers immediate rewards over larger, delayed ones.

Delay discounting rates have been found to be associated with weekly alcohol consumption, with higher rates observed among heavy-drinking adolescents compared to non-heavy drinkers (Field et al., 2007). Studies on adolescents have shown that individuals with higher delay discounting are more likely to experiment with alcohol, cigarettes, cannabis, and other illicit substances at a younger age (Kollins, 2003). A study on adolescents also found that delay discounting was a significant predictor of alcohol involvement after 6 months (Fernie et al., 2010). Similarly, a recent investigation involving 177 individuals in recovery from addiction revealed that a parental history of substance use was associated with higher levels of delay discounting (Athamneh et al., 2017). Notably, participants with both parents suffering from addiction exhibited significantly greater discounting compared to those with one or no addicted parent. Collectively, these findings suggest that impulsive decision-making is a critical vulnerability factor in the development of addiction and thus, delay discounting could be considered a behavioral marker for addiction (Poulton & Hester, 2020).

Impaired Inhibitory Control

Impaired inhibitory control refers to the inability to suppress a prepotent response or an established behavioral pattern. It describes the tendency to act without thinking, giving in to impulses rather than overcoming them. This is typically assessed using behavioral tasks such as GNG or SSTs. These tasks require participants to respond to frequent stimuli while inhibiting their response to less frequent, contrasting stimuli (Poulton & Hester, 2020).

Neuroimaging studies have identified a common neural circuit involved in "stopping impulsivity," which includes the right inferior and middle frontal gyri, anterior cingulate cortex, pre-supplementary motor area, right inferior parietal lobe, and left middle temporal cortex (Rubia et al., 2001). Interestingly, the GNG task primarily activates the left hemisphere, whereas the SST shows greater activation in the right hemisphere (D'Alberto et al., 2017; Rubia et al., 2001). In contrast, the circuitry underlying "waiting impulsivity" has been extensively studied in animals and is distinct from that of "stopping impulsivity." Waiting impulsivity is believed to depend on top-down interactions between prefrontal regions and subcortical structures, including the hippocampus, amygdala, and nucleus accumbens (Dalley et al., 2011). Recent human studies also suggest that increased waiting impulsivity is linked to reduced functional connectivity between subcortical regions, such as the subthalamic nucleus and ventral striatum, and prefrontal areas, including the subgenual cingulate (Morris et al., 2016).

Clinical studies have shown that individuals with addiction exhibit greater impairment in inhibitory control compared to healthy controls (Bickel et al., 2012). However, unlike impulsive decision-making, inhibitory control as assessed by GNG tasks may not effectively distinguish between those with risky substance/alcohol use and those with addiction (Poulton & Hester, 2020). This might be because the tasks are often neutral and lack immediate rewards or punishments, making them less sensitive in detecting response inhibition deficits (Fernie et al., 2010). It has been suggested that adding rewards to such tasks may better reveal underlying impulsivity (Poulton & Hester, 2020).

Longitudinal studies have demonstrated that deficits in inhibitory control can predict the risk of developing addiction. For instance, in children aged 12 – 14, prefrontal activity during GNG tasks predicted heavy alcohol use 4 years later (Norman et al., 2011). Similarly, prefrontal activation in adolescents during GNG tasks predicted substance and alcohol use 18 months later. A study of 498 children found that early adolescent response inhibition was a predictor of illicit substance use and alcohol-related problems, independent of family risk, ADHD, and conduct disorder (Nigg et al., 2006). In this respect, inhibitory control seems to be an important factor for the evaluation of at-risk groups.

Motor impulsivity, as measured by the SST, has been identified as a significant predictor of developing alcohol dependence among heavy drinkers over a 4-year follow-up period (Rubio et al., 2008). Similarly, research with adolescents has demonstrated that performance on the SST could predict alcohol use over a 6-month follow-up (Fernie et al., 2013). Specifically, adolescents who eventually develop binge drinking exhibit greater activity in the right middle, medial, and precentral gyri, as well as in the left postcentral and middle frontal gyri, when failing to inhibit a motor response compared to those who do not develop binge drinking (Whelan et al., 2014). Interestingly, animal studies have provided additional insights into the link between impulsivity and alcohol-related behaviors. For example, an inbred strain of alcohol-preferring rats exhibited poorer response inhibition on the SST compared to outbred rats, suggesting that heightened motor impulsivity may be a predisposing factor for alcohol-related problems (Beckwith & Czachowski, 2016). Studies on children of alcoholic parents have shown that poor motor inhibitory control on the SST may serve as a vulnerability factor for future problem drinking (Nigg et al., 2004). However, more recent research reported no significant difference in SST performance between individuals with and without a family history of alcohol abuse (Sanchez-Roige et al., 2016).

Alcohol Leading to Impulsivity

Alcohol use can increase impulsive behaviors either through its direct, acute effects or through long-term structural and functional neurobiological changes (de Wit, 2009). Both the acute and chronic effects of alcohol impair inhibition and decision-making, increasing the likelihood of engaging in risky behaviors such as unprotected sex or driving under the influence of alcohol. The direct effects of alcohol on decision-making can lead to unplanned and continued alcohol use or even escalation. After chronic alcohol use, impairments in inhibitory capacity become evident, which contributes to the persistence of substance use (de Wit, 2009).

Acute alcohol intoxication has been shown to elevate motor impulsivity, particularly by impairing the ability to inhibit actions and wait for appropriate cues (Herman & Duka, 2019). However, the effects of alcohol on decision-making remain inconclusive, with studies reporting mixed results—some showing a decrease, others no change, and some an increase—indicating a need for further exploration in this domain (Herman & Duka, 2019).

Beyond the immediate alterations in brain activity caused by alcohol, animal studies have demonstrated that elevated blood alcohol levels can directly cause brain damage (Crews et al., 2004). Such alcohol-induced changes in brain structure may exacerbate impulsivity, increase alcohol consumption, and contribute to neurodegeneration, worsening the severity of alcohol use disorders (Crews et al., 2004). Human studies further suggest structural abnormalities in both gray and white matter in brain regions involved in inhibitory control and emotional regulation in young binge and heavy drinkers, compared to their low-drinking peers (Cservenka & Brumback, 2017; Sousa et al., 2017; Wilson et al., 2015). Some research has reported increased gray matter volumes in binge drinkers, possibly indicating compensatory mechanisms (Sousa et al., 2017), while other studies have found cortical volume reductions in binge drinkers (Wilson et al., 2015). This suggests that the neurotoxic effects of heavy alcohol use may lead to neural reorganization, regardless of pre-existing vulnerabilities, and may heighten the risk of developing AUD (Cservenka & Brumback, 2017).

Impulsivity as an Endophenotype

Endophenotypes lead between genes and diseases along developmental pathways, and because they are less complex than the disease phenotypes, they are more amenable to genetic analysis (McCloskey et al., 2009). For a trait to be considered an endophenotype, it must be more prevalent in the patient population compared to the general population, be able to distinguish the psychiatric disorder from other disorders, be heritable, and be present during both exacerbation and remission phases of the illness (Gottesman & Gould, 2003; McCloskey et al., 2009). Some brain abnormalities may form part of a dysfunctional neural network that could contribute to the risk of developing AUD. These disruptions in brain circuitry may play a role in both reward deficiency and impaired response inhibition, which are predictive of impulsive behaviors. Such impairments are believed to reflect the inherited vulnerability toward AUD in high-risk individuals (Seigneurie et al., 2013).

Findings indicate that individuals with a family history of alcohol-related issues exhibit elevated impulsivity related to response initiation and inhibition, even if they do not have a personal history of alcohol or drug use disorders (Acheson et al., 2011). On the other hand, heightened impulsivity linked to sensitivity to consequences may be tied to additional risk factors, such as a more extensive family history of AUD, or it may develop as a result of prolonged heavy alcohol or drug use (Acheson et al., 2011). Young adults with a family history of AUD demonstrated distinct impulsive behavior compared to those without a family history (Sanchez-Roige et al., 2016). They exhibited higher levels of waiting impulsivity but showed lower impulsivity during decision-making. Additionally, under the influence of alcohol (0.8 g/kg), they struggled to stop an initiated response. Crucially, these effects were independent of their personal drinking history. Increased waiting impulsivity could serve as a premorbid marker for heavy drinking, potentially modifiable by acute alcohol intake, and might represent an endophenotype linked to the risk of developing AUD, particularly in the offspring of alcoholics (Sanchez-Roige et al., 2016). Studies show that especially some sub-dimensions of impulsivity may indeed represent an endophenotype for AUD. However, more studies are needed in this area for determining risk groups and planning prevention programs.

Role of Impulsivity in Relapse

Alcohol use disorder is a mental disorder that involves multiple cycles of treatment, abstinence, and relapse. Because repeated cycles of relapse have been associated with more severe withdrawal syndrome and increased psychiatric symptoms, it is important to identify relapse determinants. Impulsivity is one of the leading causes of relapse, yet it needs to be explored further (Sliedrecht et al., 2021). Research has found that the severity of AUD is linked to impaired behavioral control, which can increase the risk of relapse (Sliedrecht et al., 2021). Relapse in alcohol consumption when self-control is compromised, may happen due to: 1) a diminished valuation of delayed consequences (impulsive choice), 2) an inability to suppress dominant responses

(impulsive action), or 3) a combination of both mechanisms (Reyes-Huerta et al., 2018). The way patients frame drinking situations, the timing within a drinking episode, and alcohol-related cues may contribute to relapse. Framing drinking as a choice between immediate consumption and long-term benefits (delay discounting) or as a GNG decision (inhibition failure) can impact drinking behavior (Reyes-Huerta et al., 2018). While evidence shows that higher delay discounting predicts lapses after abstinence, acute alcohol consumption primarily affects behavioral inhibition, leading to a higher likelihood of relapse, especially in binge drinkers (Reyes-Huerta et al., 2018). Additionally, alcohol-related cues can weaken inhibitory control and increase the perceived value of immediate rewards, further contributing to relapse (Reyes-Huerta et al., 2018). In some AUD patients, impulsivity could be connected to a heightened sensitivity to alcohol's rewarding effects (Westman et al., 2017). Deficits in impulse control might negatively affect treatment adherence, indirectly leading to relapse. Impulsivity may also serve as a mediator for factors such as stress or mood, which impact craving and relapse (Coates et al., 2020). A recent review reported that self-reported impulsivity, as measured by the BIS-11, along with impulsive decision-making and motor impulsivity, are associated with relapse in AUD (Sliedrecht et al., 2021). However, due to the heterogeneity of the studies, a meta-analysis was not feasible. The authors emphasized the need for future research to use well-defined and more semantically consistent sub-aspects of impulsivity and clearer relapse definitions in AUD. They suggested that the use of standardized constructs for impulsivity and relapse could help bridge key concepts in alcohol research and facilitate translational studies (Sliedrecht et al., 2021). The interaction of impulsive choice and impulsive action across different contexts highlights the need for future research to explore how these mechanisms influence relapse after treatment.

Impulsivity as a Target for Prevention and Treatment

Psychosocial interventions are expected to be beneficial in managing impulsivity during AUD treatment. For example, contingency-based interventions may help reduce delay discounting during periods of abstinence, while cognitive-behavioral therapy (CBT) can assist in managing positive and negative urgency in high-risk situations (relapse prevention) (Witkiewitz et al., 2013). Additionally, mindfulness-based approaches may help reduce negative urgency by lowering stress and rebalancing frontostriatal connectivity (Witkiewitz et al., 2013). Response inhibition and working memory-targeted interventions have been reported to prevent impulsive drinking in problematic drinkers, especially those with low scores on these functions (Houben et al., 2011).

The primary goal of AUD treatment is generally accepted as abstinence. Factors that reduce the discount rate, such as precommitment mechanisms, can help prolong abstinence by promoting a preference for delayed rewards (Reyes-Huerta et al., 2018). Framing choices in sequences or using calendar dates for delayed rewards may also reduce discount rates, encouraging more consistent decision-making (Reyes-Huerta et al., 2018). However, if reducing alcohol consumption per drinking session is considered a treatment outcome, therapeutic strategies should focus on preventing inhibition failures. While most treatments aim to help patients abstain from drinking, they often do not address regulating the amount of alcohol consumed (Reyes-Huerta et al., 2018). Future research should focus on improving response inhibition in the presence of alcohol cues. Although we still have limited data in this area, the treatment target for alcohol use disorder may provide guidance on which sub-dimension of impulsivity should be addressed.

Impulsivity is also a target of prevention programs. A CBT framework has been used to help at-risk youth understand how individual differences in impulsivity and response inhibition affect their behavioral and emotional control as well as decision-making. The cognitive-behavioral interventions in this approach aim to help impulsive adolescents become better "stoppers" by identifying high-risk situations and recognizing internal cues that precede impulsive actions (Castellanos-Ryan et al., 2016). Additionally, cognitive strategies are provided to support the cognitive processes required for self-control (e.g., mindfulness, self-talk, goal orientation). When this program is applied to at-risk youth, studies have reported delayed alcohol use onset (Conrod et al., 2013), reduced conduct problems (O'Leary-Barrett et al., 2013), and long-term behavioral changes resulting from decreased self-reported impulsivity (O'Leary-Barrett et al., 2016).

Prevention programs also target sensation seeking by using a range of cognitive strategies focused on managing reward sensitivity and reward-driven behaviors. This intervention has shown significant effects on reducing excessive alcohol consumption (Conrod, 2016), but no significant effects on behavioral problems, aggressive behaviors, or impulsivity (Castellanos-Ryan et al., 2016). Further research is needed to determine whether these interventions produce changes in neurocognitive measures of impulsivity and if they mediate long-term behavioral changes.

Another area that should be focused on in the prevention of AUD is the psychopathologies characterized by high levels of impulsivity such as ADHD, conduct disorder, or oppositional defiant disorder. Early detection and treatment of these psychopathologies, with a focus on impulsivity, can reduce future alcohol and substance abuse risk. Additionally, identifying impulsive traits even in the absence of a formal diagnosis, and addressing them through early interventions is important for prevention (Kozak et al., 2019).

Conclusion

Binge drinking and AUD are linked to heightened impulsivity, both as a stable personality trait and as behavioral impulsivity. While elevated impulsivity increases the likelihood of frequent alcohol consumption, acute intoxication further impairs inhibitory control, potentially leading to more intense drinking episodes. Repeated cycles of heavy drinking, withdrawal, and relapse induce structural changes in brain regions related to emotional regulation and higher cognitive functions. These changes weaken self-control and increase impulsive behaviors, ultimately reinforcing the cycle of alcohol consumption. A comprehensive understanding of impulsivity's role in AUD is critical for clarifying the disease's development, formulating effective treatment plans, and guiding prevention efforts. Exciting data suggest that identifying and intervening with high-risk groups can reduce impulsivity-related issues and alcohol-related problems. However, the multidimensional nature of impulsivity adds complexity to these assessments. To address this challenge, it is recommended that

future studies take a multidimensional approach to impulsivity, considering its various subcomponents for a more nuanced analysis.

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