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REVIEW

Contextualizing Impulsivity in Alcohol Use Disorders: Interactions with Social Cognition and Interoception

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Abstract

Purpose of review We capitalize on recent developments in the neuropsychology of alcohol use disorders to propose an expanded conceptualization of impulsivity. We principally underline the added value of an integrative perspective, encompassing its interactions with external and internal contexts.

Recent findings We review the emerging literature suggesting that impulsivity is not an isolated phenomenon, and that meaningful relations with social cognition and interoception processes exist. Acknowledging these relations holds promise for improving impulsivity conceptualization and measurement, as well as for providing treatment venues in alcohol use disorders.

Summary The current approach of conceptualizing and studying impulsivity in alcohol use disorders does not fully consider the context in which impulsive behaviors occur. Recent evolutions in alcohol research support the inclusion of social environment and inner bodily states in studies measuring alcohol-related impulsivity.

Keywords Impulsivity · Alcohol use disorders · Social cognition · Interoception · Emotions

Introduction

Impulsivity broadly refers to the tendency to act in an unplanned, premature, or inappropriate manner, which often results in undesirable outcomes [1]. It is considered a core vulnerability factor and defining feature in a wide range of psychopathological states [2], including addictive disorders [3, 4]. In alcohol research, the focus of the present paper, impulsivity has long been involved in the emergence of problematic consumption patterns [5, 6], as well as in the development [7, 8] and persistence of severe alcohol use disorders [9].

Despite its proposed role in alcohol use disorders (AUD), several key issues and core theoretical limits surrounding impulsivity have been identified. First, classical perspectives conceptualized impulsivity as a unitary process combining subdimensions in an umbrella construct [10], but more recent proposals have suggested that impulsivity should rather be considered a constellation of distinct processes, each independently influencing psychopathological states [11, 12]. Second, and relatedly, impulsivity measurement is heterogeneous, in part due to an abundance of tools tapping subdimensions that only partly overlap [11, 13, 14]. This led to a lack of gold-standard evaluation tools and inconsistent findings, preventing the emergence of a coherent framework [15]. These issues may partially explain the very limited efficacy of current impulsivity interventions in addictive disorders [16–18].

For the purpose of this paper, we do not take a strong position regarding the optimal number of impulsivity subdimensions, as the underlying structure of impulsivity remains a matter of debate [19]. We instead adopt a pragmatic approach by capitalizing on a parsimonious two-factor model of impulsivity [20] distinguishing reward sensitivity/seeking and rash impulsivity (i.e., acting without forethought due to reduced inhibition). Each component accounts for unique variance in addictive outcomes, and this model compares favorably with more complex

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multifactorial models in predicting substance use [21–23]. Importantly, this distinction maps closely onto dual-process models of addiction, which conceptualize problematic alcohol use as arising from the interaction between a hyperactive reflexive system driving reward approach toward alcohol cues and a weakened reflective system responsible for inhibitory control [24, 25]. These dual processes reflect general mechanisms underlying impulsive behavior, extending beyond addiction per se [20]. Accordingly, this two-factor formulation is consistent with recent consensual definitions of impulsivity, which describe it as both (1) a predisposition toward rapid reactions to internal or external stimuli without regard for negative consequences and (2) a tendency to act with less forethought than do most individuals of equal ability and knowledge [26].

Building upon this two-factor model, we go one step further by highlighting another, comparatively neglected, but critical issue: impulsivity in AUD has been approached as an isolated, purely cognitive/behavioral phenomenon, with its role confined to that of a direct determinant of alcohol use. We propose that such a focus is outdated, and that it should make way for a more contextualized perspective on impulsivity in AUD. Specifically, we will introduce the idea that impulsivity research in AUD should be integrated within a broader neuropsychological framework, especially alongside recent findings in social cognition and interoception. These two domains are increasingly considered essential to a comprehensive understanding of AUD. By outlining interrelations between these constructs and the two impulsivity subdimensions relevant to AUD (i.e., reward sensitivity and reduced inhibition), we will emphasize the potential for a better, more ecological and comprehensive understanding of impulsivity itself, its situational/dispositional modulators (i.e., internal/interoceptive and external/social context), and its broad impact on AUD.

The Decontextualized Approach of Impulsivity in AUD

In AUD, the vast majority of studies have put aside the influence of context on impulsivity. They have mostly relied on self-reports, notably based on the Barrat [27, 28] or UPPS [29] frameworks, or on neuropsychological tasks (e.g., flanker, go-no go or stop signal paradigms, Iowa gambling task) used in isolation, and investigated their direct links with alcohol use outcomes. Such an approach assumes a relative stability of impulsivity in AUD, and does not model the influence of the real-life environmental settings in which it unfolds [10, 12, 30]. It is also incompatible with initial conceptualizations of impulsivity [31] that already underlined its tight relationship with sociability and physiological variables. We hence argue that there is an urgent need to integrate impulsivity-related processes in their broader interpersonal and interoceptive context in AUD, and therefore to investigate its interrelatedness with alterations in social and interoceptive processes.

Importantly, recent lines of research go in this direction by pointing to the need to consider the environment in the relation between impulsivity and AUD. Several studies have evidenced moment-to-moment variations of impulsivity, such variations influencing urges to drink and alcohol consumption [32–34]. Impulsivity and its impact on alcohol use thus appear sensitive to contextual variations, but the specific factors driving such variations remain underexplored. The few studies directly investigating this question have focused on alcohol-related cues and stress. On the one hand, studies have shown a significant relationship between self-reported impulsivity and alcohol-related attentional, memory, or approach biases [35], implying that alcohol cues may exacerbate impulsive tendencies, and particularly reward sensitivity [36]. On the other hand, studies measuring chronic stress or eliciting acute stress (notably using social stress induction) have demonstrated that stable or transient modulations of the emotional and arousal states interact with impulsivity to promote alcohol use [37–39], here again showing that the impact of impulsivity on AUD cannot be fully understood through research isolating it from its broader environment. While highlighting the importance of a contextualized approach to impulsivity in AUD, these studies are restricted to alcohol-related cues or stress variables, hence ignoring the wide variety of contextual factors at stake [40]. To overcome this issue, we describe below how two recent research evolutions in the alcohol field, respectively focusing on the role played by social cognition and interoception, can be leveraged to integrate the outer (i.e., social signals) and inner (i.e., body signals) contexts of impulsivity in AUD.

Social Processes in AUD and their Links with Impulsivity

The neuropsychological exploration of AUD has long focused on "cold"/neurocognitive processes with a large body of literature having identified alterations in perceptual, attentional, memory and executive abilities [41–43]. In the past two decades, this literature has extended to the social domain by studying social cognition and social decision making. Social cognition refers to the psychological processes allowing individuals to detect and interpret social signals produced by others [44, 45], and encompasses four subcomponents: emotion recognition, theory of mind, attributional biases, and social perception/knowledge. Social decision making refers to the choices people implement in interpersonal contexts and that have effects on others (e.g., how they solve moral dilemma involving different consequences for different individuals). Together, these skills support healthy social functioning and the development of a social support network, an essential protective factor for mental health. Recent studies have shown that AUD relates to marked alterations in all four social cognition components [46], as well as to biased social decision-making [47], which may foster isolation, reduce social support, and reinforce alcohol use as a coping strategy as well as relapse [48, 49], in line with what has been described in other addictive states [50]. These findings have fueled the growing movement to consider severe AUD as also being an interpersonal disease with marked difficulties to behave adaptively in social situations. A next important step is to integrate social processing alterations in the dominant models of AUD and to explore their connections with the key variables involved in this disorder, including impulsivity. Initial insights supporting connections with impulsivity came from studies indicating that impulsivity and antisocial personality (strongly related to social cognition and decision-making deficits [51]) jointly contribute to promoting addictive disorders [52–54]. Capitalizing on these preliminary results, we propose that, beyond their separated bidirectional relations with AUD, impulsivity and social processes interact and reinforce each other, further increasing their role in AUD.

On the one hand, impairments in impulsivity may promote social cognition alterations. In particular, reduced inhibition (i.e., rash impulsivity) can impair the ability to inhibit prepotent, readily accessible, or spontaneous social interpretations, reducing the accuracy of interpersonal judgments and fostering biased appraisals in social situations. This is notably consistent with models proposing that cognitive control is key to reappraising the hostile attributional biases [55] often observed in AUD [56, 57]. Evidence outside the AUD literature supports an effect of impulsivity on social cognition. For instance, emotion recognition and theory of mind performance are associated with cognitive control ability or self-reported impulsivity [58–60], and altered by concurrent tasks taxing executive resources or the stimulation of brain regions involved in cognitive control [61–63]. Impulsivity might further (independently and in conjunction with social cognition alterations, which impact the initial interpretation of social situations) shape social decision making by promoting rapid, emotionally-driven responses at the expense of carefully reasoned ones [64, 65]. This can lead to unfavorable social choices, particularly in situations requiring a balance between immediate and long-term outcomes. Clarifying whether, and to what extent, impulsivity processes underlie social processing difficulties in AUD has implications for conceptualizing the role of impulsivity in the disorder.

Moreover, impulsivity is primarily thought to proximally predict dysregulated alcohol use, both by increasing the attraction towards alcohol-related stimuli (i.e., increased reward sensitivity) and by lowering the ability to control consumption behaviors (i.e., reduced inhibition). Through its effects on social cognition and decision-making, it may, however, also contribute more distally to the disorder by deteriorating interpersonal functioning, a critical factor in the emergence and maintenance of AUD. Clinically speaking, these insights may guide more informative assessment (e.g., systematic querying of social relationships when impulsivity is evidenced) and shed light on impulsive processes as potential intervention levers for attenuating social cognitive deficits, decision-making impairments and interpersonal problems in the disorder. At the experimental level, the effect of impulsivity on social cognition in AUD could first be investigated through large studies comprehensively testing cross-sectional associations within the target population, and determining if the two dimensions of impulsivity statistically account for differences in social cognitive ability and biased decision-making. Based on these results, studies may experimentally investigate whether reducing relevant impulsivity subdimensions (e.g., through targeted training or brain stimulation) attenuates candidate social processing alterations in AUD. The outcomes of this second line of studies would allow drawing causal conclusions regarding this relationship and identifying clinical interventions. We also suggest overcoming the current compartmentalized exploration of impulsivity and social cognition in AUD, in order to explore their interactions and their joint influence on key processes involved in this disorder, through a contextualized exploration of impulsive behaviors, as already initiated by studies using economic games [64].

On the other hand, social processing alterations may heighten impulsivity and, in particular, reward sensitivity. Misperceptions or negatively biased interpretations of social signals, as well as socially inappropriate decisions, can result in real or perceived interpersonal conflict, awkwardness, or rejection [66, 67]. The resulting negative socio-affective states represent known triggers of increased attention to alcohol cues (i.e., reward sensitivity), valuation of alcohol, and craving [68–70]. Hence, social processing alterations may drive or compound increases in sensitivity to alcohol cues in certain social situations, ultimately increasing the likelihood of impulsive alcohol consumption that would not occur in the absence of such social alterations. Examining these effects may offer conceptual insights into some determinants of social situational changes in impulsive processes in AUD, as well as clarify how impulsivity can mediate the relation between social processing difficulties and dysregulated alcohol use [71]. These insights would further provide a rationale for more socially contextualized investigations of impulsive processes in people with social processing impairments. This aligns with repeated failures to observe “trait-like” or stable attentional biases towards alcohol-related cues, a key marker of sensitivity to the reward value of alcohol [72, 73], and ensuing calls for their increased consideration as transitory, contextualized phenomena that may specifically operate in at-risk situations. From this perspective, assessment of such biases may be more relevant (i.e., predictive), and interventions more effective, when embedded in the settings in which they are more likely to emerge. A relevant starting point for testing the hypothesized effects would be to examine whether social processing alterations moderate the relationship between ambiguous or clearly negative social situations and indices of increased impulsivity (e.g., attentional biases). If findings reveal that such social situations only (or more strongly) increase impulsivity (i.e., generate higher reward sensitivity and/or reduced inhibition) in people with greater social cognitive or decision-making alterations, a second line of studies should determine whether addressing social processing alterations reduces those situational effects.

Interoception in AUD and its Links with Impulsivity

Interoception refers to the conscious and non-conscious processing of internal bodily states [74]. Interoceptive measures can be differentiated either by (1) measurement type, including self-report questionnaires, performance-based tasks, and physiological indices; (2) the bodily signal assessed, such as cardiac or respiratory cues; or (3) the dimension targeted, such as perception, attention, interpretation, or memory of internal bodily states [74]. Although influential conceptual frameworks have proposed broad dimensions of interoception, including interoceptive accuracy [75, 76], empirical evidence indicates that these dimensions are operationalized using heterogeneous measures that often show limited convergence [74]. Accordingly, we use the general term “interoception” when discussing theoretical hypotheses that do not specify a particular dimension, and refers to specific measures when discussing empirical findings. This is particularly important because findings on the role of interoception generally differ between and within measurement types.

To maintain homeostasis, the brain must continuously model the body’s physiological condition and adjust autonomic and voluntary actions accordingly [77]. In daily life, interoception supports fundamental adaptive behaviors (e.g., body temperature regulation, hunger, thirst) and complex psychological functions, including emotional experience, reward valuation, or decision-making [78, 79]. Altered interoception may promote maladaptive patterns of behavior, including addictive disorders [80]. Indeed, following seminal work showing that lesions of the insular cortex (the postulated hub of interoception) led to cessation of urge and smoking in smokers [81], interoception has been proposed to represent an important etiological factor for addictive disorders [80, 82, 83]. Behavioral and neuroimaging studies discussing the role of interoception in addictive disorders, and in particular AUD, have begun to accumulate [84–87]. Growing evidence suggests altered interoception in AUD, even though this dimension has long remained marginal in addiction research relative to the dominant focus on cognition and neural circuitry [88]. It has, for instance, been shown that individuals with AUD report unpleasant bodily sensations of craving [89]. It should be noted, however, that current conclusions regarding interoception and AUD are largely derived from brain lesion and neuroimaging studies. As such, these findings provide only indirect evidence for the role of interoception in AUD, because the implicated regions, including the insular cortex and anterior cingulate cortex, also support other processes (e.g., emotional awareness) and can be modulated by psychopathological states (e.g., anxiety). Moreover, the role of interoception in AUD remains largely unexplored in relation to impulsivity, despite strong theoretical expectations that altered interoception and heightened impulsivity interact to promote AUD [90].

In everyday life, the expression “gut feelings” reflects the reliance on bodily sensations to guide choices [91], a role emphasized by foundational decision-making theories [92]. Accordingly, alterations in interoception may contribute to maladaptive decision-making, including impulsive behaviors [93, 94], and a recent seminal paper proposed an interaction between interoception and impulsivity in addictive disorders [95]. Building on this view, we propose that interoception interacts with the two dimensions of impulsivity (reward sensitivity and rash impulsivity) in AUD, such that modifications in the perception of, attention to, interpretation of, or memory of bodily signals influence both dimensions. Because these dimensions mirror the dual-process model of addiction, this proposal aligns with the triadic model of addiction [82], which posits that the anterior insula integrates interoceptive signals into subjective urges or craving, amplifying impulsive habit systems while weakening prefrontal control by capturing cognitive resources required for inhibitory control. To our knowledge, however, behavioral studies directly examining interoception-impulsivity relationships remain scarce, and only one, finding no significant association, has been conducted in AUD [96]. The available evidence, mostly relying on non-clinical adult samples, does not support a robust general association between impulsivity and self-report or performance-based measures of interoception [93, 96–104]. Most effects are non-significant, and significant findings are inconsistent across impulsivity facets and interoception measures. This may reflect reliance on heartbeat detection tasks of limited validity and a narrow set of heterogenous self-report measures [105, 106]. Thus, despite strong theoretical expectations, direct evidence of the links between interoception and impulsivity in AUD is lacking, underscoring the need for studies using more valid performance-based and self-report measures.

Regarding rash impulsivity, the somatic marker hypothesis [92] posits that bodily cues bias behavior toward advantageous long-term outcomes by signaling the value or risk of available options; impulsive behavior may therefore arise when bodily warning signals are absent or poorly perceived [93, 99]. By contrast, within the triadic model, bodily signals of urges would capture attentional and cognitive resources, thereby transiently undermining inhibitory control [80, 82]. Although these perspectives may appear contradictory, together they suggest that interoception does not exert a uniform effect on inhibitory control. Consistent with this view, empirical studies show that synchronizing heartbeats with signals requesting to stop a behavior can improve motor inhibitory control [107, 108 but see 109]. As a multidimensional construct [110], interoception likely influences inhibitory control depending on the context and the type, valence, and intensity of bodily sensations. For instance, inhibitory control may follow an inverted U-shaped relationship with interoceptive signal intensity, such that weak sensations fail to engage control processes while strong sensations overwhelm them [80]. Finally, impaired inhibitory control in AUD may also arise from inaccurate prediction and reduced integration of future consequences (e.g., bodily harms) into current decisions, biasing behavior toward immediate rewards despite long-term costs [111].

Regarding reward sensitivity, interoception can be conceptualized as guiding reward approach based on a stimulus's ability to induce pleasant bodily sensations or alleviate unpleasant ones [83]. This effect may depend on prior physiological state, such that the same stimulus is experienced as pleasant when correcting a physiological imbalance but as unpleasant when homeostasis is present [112], or alternatively on discrepancies between expected and currently experienced bodily states, whereby interoceptive prediction errors motivate reward approach. These hypotheses align with positive and negative reinforcement frameworks of addiction, yet direct behavioral evidence in AUD remains limited. AUD individuals report more intense and unpleasant bodily sensations of craving than subclinical populations [89], suggesting that reward sensitivity may be driven by motivation to reduce these sensations. Beyond reinforcement accounts, optimal arousal theory proposes that impulsive behavior arises from low baseline physiological arousal, motivating stimulation-seeking [113–115], which may be independent from the valence of sensations. Relatedly, a low level of response to alcohol may sustain reward seeking to achieve desired states [116], whereas heightened bodily sensations during alcohol's rewarding effects may further promote impulsive behavior through increased reward sensitivity [117]. Recent empirical findings from subclinical samples suggest that individuals at risk for AUD may experience reduced bodily sensations during intoxication [118], while continuing to consume despite more intense hangover sensations [119]. Thus, reward sensitivity in AUD may reflect motivation to achieve desired stimulation alongside impaired integration of prior hangover-related bodily signals.

We recommend future studies to examine the interactions between interoception and impulsivity in AUD by going beyond traditional measures of interoception such as heartbeat detection tasks for validity and theoretical reasons. Instead, we need to move toward more robust paradigms, a broader range of bodily systems, and a fuller consideration of interoception's multiple dimensions [74]. For instance, one could probe the role of interoceptive phenomenology (i.e., the characteristics of subjective bodily sensations) in both impulsivity facets among AUD individuals. The bodily mapping paradigm (allowing individuals to report bodily sensations by coloring body regions) offers a particularly promising avenue to achieve this aim [95]. A large study [94] showed that participants with higher impulsivity reported more atypical body maps and reduced body sensations in the head across emotional states, potentially reflecting reduced top-down control during affective experience. Further research among AUD individuals is, however, needed. Moreover, the most famous bodily mapping tool (emBODY; [120]) only measures the intensity of bodily sensations, while their valence may be at least as important in predicting impulsivity, as mentioned earlier. We thus also recommend the use of a recently developed bodily mapping tool called InteroMap which includes both valence and intensity [121].

More broadly, one possible avenue for future research would be to build on recent findings in Attention-Deficit/Hyperactivity Disorder (ADHD; a disorder notably characterized by high impulsivity), which indicate reduced interoception in individuals diagnosed with ADHD or presenting ADHD symptoms [122]. However, because this literature generally focuses on ADHD diagnoses or symptom dimensions rather than on impulsivity as a distinct construct, it supports a broader role of interoception in self-regulation rather than demonstrating a direct and robust association with impulsivity specifically. Another promising perspective for extending the exploration of interoception/impulsivity links in AUD would be to apply emerging interoceptive indices of body-brain interactions (e.g., stomach-brain coupling [123]), notably in the framework of the novel paradigms arising from the rapidly expanding literature on the gut-brain axis in AUD [124, 125].

Finally, the few existing data linking interoception and impulsivity in AUD focused on the impact of the former on the latter and did not explore the reverse link: how impulsivity can modulate interoception. Indeed, impulsivity may lead individuals with AUD to have a reduced attention to their inner bodily states, as well as a lower ability to detect/distinguish them, facilitating the neglect of interoceptive signals, notably related to the alcohol effects on the body. Here again, these hypothetical links support the need to abandon the focus on the cognitive and isolated approach of impulsivity to understand how it interacts with the inner environment of the individual in real-life situations. This would generate a better understanding of how higher reward sensitivity and reduced inhibition in AUD act together with interoceptive abilities to promote dysregulated alcohol use.

Conclusion

The present paper centrally proposed that the exploration of impulsivity in AUD should evolve from a compartmentalized approach of cognitive impulsivity towards a contextualized perspective on impulsive behaviors, generating deep changes in this field at conceptual, experimental, and therapeutic levels. Capitalizing on a two-dimensional model of impulsivity, coherent with dual-process models by distinguishing reward sensitivity and rash impulsivity, we showed how interpersonal environment (social cognition / social decision-making) and internal context (bodily sensations / interoception) meaningfully interact with impulsivity to shape AUD, as summarized in Fig. 1. We argued that impulsivity should no longer be considered and measured

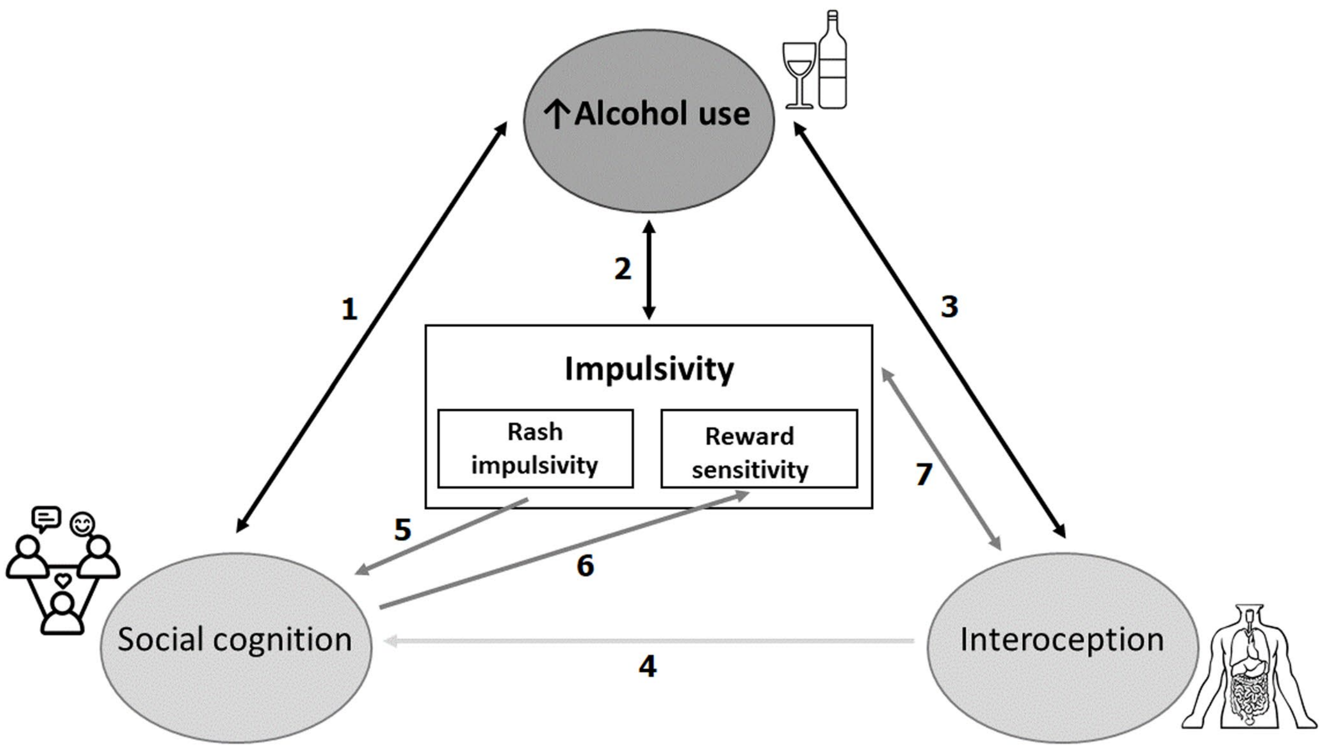

Fig. 1 Graphic representation of the proposed contextualized approach of impulsivity, considering its relations with social cognition and interoception. The arrows underline the postulated links between (1) *Social cognition and alcohol use*, as AUD is associated with marked alterations in all four social cognition subcomponents, which in turn reinforces alcohol use as a coping strategy to face unsatisfactory social interactions resulting from impaired social cognition; (2) *Impulsivity and alcohol use*, as impulsivity favors AUD by increasing the attraction towards alcohol-related stimuli (i.e., reward sensitivity) and by lowering the ability to control consumption behaviors (i.e., rash impulsivity). Conversely, the neurocognitive impact of AUD can increase impulsivity by further sensitizing the reflexive system and damaging the reflective one; (3) *Interoception and alcohol use*, as modified interoception is an important etiological factor for AUD, promoting urges and craving. Conversely, AUD can promote altered interoceptive abilities, notably through insular damage; (4) *Interoception and social cognition*, as biased or impaired processing of internal bodily states can reduce the ability to correctly perceive emotional and social cues useful in interpersonal contexts; (5) *Rash impulsivity and social cognition*, as reduced inhibition impairs the ability to inhibit prepotent social interpretations, fostering biased appraisals in social situations and hence reduced social cognition abilities; (6) *Reward sensitivity and social cognition*, as biased interpretations of social signals can result in interpersonal conflict, which is a known trigger of increased attention to alcohol cues and reward seeking; (7) *Interoception and impulsivity*, as rash impulsive behaviors may arise when bodily warning signals are poorly detected since bodily cues bias behavior toward advantageous long-term outcomes by signaling the value or risk of available options. Moreover, bodily signals of urges or craving capture attentional and cognitive resources, thereby undermining inhibitory control and favoring reward sensitivity. Conversely, impulsivity can lead individuals with AUD to have a reduced attention to their inner bodily states, as well as a lower ability to detect and distinguish them, facilitating the neglect of interoceptive signals

independently from the environment in which it takes place. We proposed research avenues to move the field forward by exploring how social abilities and interoception modulate impulsivity and its relation with AUD, to develop a socially and bodily anchored approach to impulsive behaviors.

This contextualized perspective integrating impulsive behaviors in their environment holds theoretical implications, by underlining that current models of impulsivity may need refinement to include environmental influences. We consider that social cognition and interoception should not be directly incorporated into the core concept of impulsivity (e.g., by creating concepts like social or interoceptive impulsivity) as adding new subdimensions would bring more noise than clarity into this already complex and debated research field. However, we propose, as illustrated in Fig. 1, that contextual influences should be integrated as key driving and moderating variables. We also provide experimental and methodological recommendations by suggesting that, beyond the diversity of measures (e.g., self-report questionnaires, neuropsychological tasks, neuroscience indexes) necessarily generating divergent results, future studies exploring impulsivity in AUD should increasingly do so in the context of interoceptive and social cognitive measures to better reflect the fact that real-life impulsive behaviors cannot be separated from their inner/outer environment. Finally, at the clinical level, the contextualized impulsivity approach might provide promising venues regarding interventions reducing impulsive behaviors in AUD. Indeed, the current treatment programs, focused on the cognitive component of impulsivity, have failed to significantly reduce impulsive tendencies in addictive disorders, and hence to significantly improve treatment outcomes and abstinence [16, 17, 126]. Integrating body signals (e.g., through mindfulness-based interventions [18]) and social cognition rehabilitation in these impulsivity-focused programs might increase their efficiency as well as their generalization to everyday life behaviors.

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  - An experimental study demonstrating a direct link between modified interoception (i.e., atypical bodily sensations related to emotions) and impulsivity.

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Declarations

Human and Animal Rights and Informed Consent N/A.

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