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The association between impulsivity, emotion regulation, and symptoms of alcohol use disorder

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Abstract

Emotion dysregulation and impulsivity are important factors influencing the development and course of alcohol dependence. However, few empirical studies investigate the association between different aspects of impulsivity (cognitive, attentional, behavioral), emotion regulation, and alcohol use disorder symptoms in the same model. The goal of this study was to assess the association between emotion regulation and different facets of impulsivity among patients with an alcohol use disorder and healthy controls. The sample was comprised of 273 individuals: 180 participants with an alcohol use disorder undergoing inpatient alcohol treatment and 93 healthy controls. Emotion regulation was assessed using the Schutte Self-Report Emotional Intelligence Test subscale. Impulsivity was assessed with Barratt's Impulsiveness Scale (BIS-11). Statistical models tested both the BIS-11 total score, as well as three secondary factors (non-planning, attentional, and motor impulsivity). Findings indicate that individuals with alcohol use disorder symptoms were characterized by poor emotion regulation and high levels of impulsivity in all analyzed domains. Moreover, path analytic models indicated that after accounting for demographic factors (i.e., biological sex, age, education) there was evidence for a significant indirect effect of alcohol use disorder symptomatology on non-planning and attentional impulsivity via emotion regulation. There was no association between emotion regulation and motor impulsivity. These findings indicate the importance of targeting emotion regulation skills as well as behavioral control when treating patients with alcohol use disorder.

Keywords

non-planning impulsivity; attentional impulsivity; motor impulsivity; emotion regulation; alcohol use disorder

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1. Introduction

Alcohol use is one of the most important factors contributing to the global health burden and costs to society (Rehm et al., 2009). In the European Union, where alcohol consumption is among the highest in the world, approximately 14% of all deaths in men and approximately 8% of all deaths in women between 15 and 64 years of age are due to alcohol (Rehm et al., 2012). These statistics support growing interest in developing successful treatment programs for alcohol use disorder (AUD). Despite consistent effort, the disorder is still characterized by persistent vulnerability to relapse even after specialized treatment (Moos & Moos 2006). Two potential factors that are significantly related to relapse in AUD, are impulsivity and emotion regulation. This paper examines differences between various aspects of impulsivity and emotion regulation across patients receiving treatment for AUD and healthy controls.

According to Moeller and colleagues (2001), impulsivity is “a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions” (p. 1784). More specifically, impulsivity is a multidimensional construct comprising attentional, behavioral, and cognitive components. The current literature makes a distinction between impulsive *action* (i.e., behavioral impulsivity — inability to stop initiated reaction) versus impulsive *decisions* (i.e., cognitive impulsivity — inability to weigh the consequences of one’s behavior and therefore to delay gratification; Arce & Santisteban 2006). Moreover, attentional impulsivity is defined as an impairment in the ability to maintain focus on a specific task (de Wit, 2009). Generally speaking, impulsivity is considered a trait-like characteristic, as well as a state-like condition, related to an individual’s current emotional state (Swann et al., 2004). A growing body of research has demonstrated that high levels of impulsivity may contribute to the development of alcohol use disorder (Dom et al., 2006; Finn et al., 2002; Lejuez et al., 2010) and result in poorer outcomes following its treatment (Loree et al., 2014). Although not as widely studied, there is convincing evidence that higher levels of impulsivity may also be a *consequence* of alcohol consumption (Dougherty et al., 2000). For example, several studies revealed that in non-problematic alcohol users, alcohol administration led to increased levels of impulsivity (Dougherty et al., 2000; Sanchez-Roige et al., 2016). In addition, prior research indicates frontal lobe dysfunction may lead to high levels of impulsivity observed among individuals with AUD (Wang et al., 2016). Moreover, high levels of impulsivity were associated with alcohol withdrawal, as well as number of alcohol detoxifications (Duka et al., 2003). In general, though fewer studies examine impulsivity as a consequence of AUD, there is strong support for it as well.

Similarly, emotional dysregulation is also viewed as a multi-faceted construct involving: (a) lack of awareness, understanding, and acceptance of emotions; (b) an inability to control behaviors when experiencing emotional distress; (c) a lack of access to adaptive strategies for modulating the duration and/or intensity of aversive emotional experiences; and (d) an unwillingness to experience emotional distress as part of pursuing meaningful activities in life (Gratz & Roemer, 2004). Impairment in emotion regulation is thought to be one of the major motives for alcohol drinking, as well as a core emotional disturbance among individuals with AUD (Petit et al., 2015). It was also shown that impairment in emotion regulation is associated with poorer outcomes among individuals with an AUD (Berking et

al., 2011; Kopera et al., 2014). Importantly, there is also evidence that poor emotion regulation may be a consequence of alcohol drinking. For example, Kober (2014) demonstrated that chronic alcohol use (through its negative impact on the prefrontal cortex – a key neural region for emotion regulation) impairs the ability to cope with aversive emotional states. Moreover, it was also shown that acute alcohol administration in non-AUD participants may increase emotional dysregulation (Watkins et al., 2015). Thus, there is evidence supporting the role of emotion dysregulation as both an antecedent and a consequence of problematic alcohol use.

Consequently, there is increasing interest in the impact of AUD symptomatology on potential deficits in both emotion regulation as well as impulsivity (Pedersen et al., 2016; VanderVeen et al., 2016; Watkins et al., 2015). Results from recent meta-analyses confirm that problematic use amongst older adolescents may be a consequence of a tendency to act rashly during periods of heightened positive or negative mood (Smith & Cyders 2016; Stautz & Cooper 2013). While the work of Riley and colleagues (2016) demonstrates that there is likely a bidirectional association between emotional aspects of impulsivity and problematic alcohol use. Namely, high urgency (emotional impulsivity) predicted subsequent drinking, but problematic drinking also predicted subsequent increases in urgency (Riley et al., 2016).

Taken together, there is a substantial body of evidence indicating that high impulsivity and poor emotion regulation are a result of problematic alcohol use and these factors may predict rates of relapse among individuals with AUD. Yet, few studies examine the impact of problematic alcohol use on impulsivity and emotion dysregulation in the same model. Moreover, there are surprisingly few studies addressing this issue in a clinical sample of individuals diagnosed with AUD. Understanding the relation between emotion regulation and impulsivity is of great interest among individuals diagnosed with AUD given that these two factors are established predictors of relapse to drinking. Although prior work has not focused on individuals with AUD, similar studies have been conducted among other clinical samples. Namely, prior work has indicated that poor emotion regulation may lead to increased impulsive behaviors among individuals with post-traumatic stress disorder, eating disorders, and borderline personality disorder (Messman-Moore et al., 2010; Weiss et al., 2012; Williams et al., 2006). Interestingly, all of these disorders are well-established risk factors for alcohol-related problems. In addition, there is very little data on associations between different aspects of impulsivity and emotional processes, highlighting the need for further work that would integrate emotional regulation with other domains of impulsivity (Smith & Cyders 2016). Specifically, direct associations between emotion regulation and impulsive decisions (which may be decisions to act, but also not to act, *cognitive impulsivity*), impulsive actions (motor impulsivity), as well as attentional impulsivity are yet to be thoroughly investigated. An empirical investigation of this sort as it pertains to AUD symptoms would be especially significant given that various dimensions of impulsivity may be related to different aspects of alcohol use problems: cognitive impulsivity has been associated with early onset of alcohol drinking problems, motor impulsivity – with poor alcohol treatment outcomes (Rubio et al., 2007), and attentional impulsivity - with severity of physical pain (which was shown to be an important predictor of relapse to drinking) in participants with AUD (Jakubczyk et al., 2016). Given that emotion regulation and impulsivity share common neurobiological underpinnings (e.g., activity of ventromedial

prefrontal cortex;), it is possible that in emotionally salient situations, significant resources are devoted to the processing of emotions with few resources remaining for executive functioning (e.g., behavior control, cognitive analysis, careful decision making; Brown et al., 2012). Therefore, poor emotion regulation may partially explain the link between AUD symptoms and higher impulsivity. To the best of our knowledge, prior work has not empirically tested this pathway.

Given the current knowledge gap regarding associations between impulsivity and emotion regulation among participants with AUD, the aim of the current study was to assess the association among symptoms of AUD, emotion regulation, and different facets of impulsivity (behavioral, cognitive, and attentional) in a clinical sample. We hypothesized that: 1) individuals with AUD would be characterized by greater impairments in emotion regulation and higher levels of non-planning, attentional, and motor impulsivity than healthy controls; 2) impairments in emotion regulation would be associated with higher levels of non-planning, attentional, and motor impulsivity; 3) there would be evidence for an indirect effect of AUD status on levels of non-planning, attentional, and motor impulsivity via impairments in emotion regulation.

2. Material and methods

2.1. Participants and procedures

This study was conducted in accordance with the ethical principles described in the Declaration of Helsinki in 1964 and received approval from the Bioethics Committee at the Medical University of Warsaw. Participation was confidential and voluntary; all participants were informed about the protocol of the study and gave written informed consent. The study population was comprised of two groups: individuals meeting criteria for AUD and healthy controls (HCs) not meeting criteria for AUD.

Participants over the age of 18 (n=180) meeting criteria for AUD were recruited among individuals entering an eight-week, abstinence-based, inpatient treatment program. In order to increase compliance in the therapeutic process, only individuals with stable mental conditions were enrolled in the treatment program. All participants presenting to the admission room of the treatment center, regardless of their age, biological sex, race or ethnicity, were invited to participate in the study during the admission. After obtaining informed consent, participants were asked to complete questionnaires during the first week after admission to the ward. Due to a significant overrepresentation of male individuals in Polish addiction treatment programs, the majority of the study group were males. More specifically, the study group comprised 148 men (83.2%) and 32 women (17.8%). Since non-White individuals did not present for treatment at this center during the time of the study, the study group included only White Europeans.

The diagnosis of AUD was based on the International Classification of Diseases and Related Health Problems 10th Revision (WHO, 1992) using the MINI International Neuropsychiatric Interview (Sheehan et al., 1998). Patients were not eligible for the treatment program if they met any of the following criteria: a history of psychosis, co-occurring psychiatric disorders requiring current medication (e.g., major depressive disorder,

psychosis, bipolar disorder), and the presence of acute alcohol withdrawal symptoms. Moreover, all individuals with clinically significant cognitive deficits (as established by less than 25 on the Mini-Mental State Examination; Folstein et al., 1975) were not eligible for admission to the treatment program. Lastly, individuals meeting criteria for another substance use disorder (with the exception of tobacco) were also not admitted to the treatment program and, therefore, not included in the present sample.

HCs (n=93; 87 [93.5%] men, 6 [5.4%] women) were selected from the administrative staff of the Medical University of Warsaw and the Nowowiejski Hospital (Warsaw, Poland). Flyers inviting study participants were posted across different university facilities. Taking into consideration the overrepresentation of males in the group of patients with AUD, the HC group was also designed to comprise a majority of men. The primary exclusion criterion for the HC group was presence of AUD symptoms or an indication of harmful alcohol use as assessed by the Alcohol Use Disorders Identification Test (AUDIT) (Babor & Higgins-Biddle 2000). Other exclusionary criteria included being 18 years of age or younger, intellectual disabilities, or current symptoms of severe comorbid mental illness (e.g., psychosis, bipolar disorder, major depressive disorder), consistent with the eligibility criteria of the overall treatment program.

2.2. Measures

Questions regarding basic sociodemographic characteristics (e.g., age, biological sex, education) were obtained using a self-report questionnaire prepared for the purpose of this study.

Mental disorders—Psychiatric comorbidity was assessed with the Polish version of the MINI International Neuropsychiatric Interview (Masiak & Przychoda, 1998).

Alcohol use problems—The severity of alcohol use problems was assessed using the Michigan Alcoholism Screening Test (MAST; Selzer, 1971), which is a self-report scale that contains 25 dichotomous (yes/no) items. A Polish version was used in this study (Habrata, 1988). In addition, for participants with AUD, a single question from the modified version of the Substance Abuse Outcomes Module (Smith et al., 1996) asking about the age at which individuals first experienced drinking problems throughout their life was utilized. The duration of problem drinking was defined as the difference in age at the baseline assessment and the age when problems related to drinking began. The Substance Abuse Outcomes Module also assessed alcohol use over the past 4 weeks prior to admission to the treatment center.

Impulsivity—The 30-item Barratt Impulsiveness Scale (BIS-11; Patton et al., 1995) was utilized as a subjective measure of three different dimensions of impulsivity derived from six basic first-order factors: attention (focusing on current tasks; e.g., “*I don’t pay attention*”), cognitive instability (intruding thoughts; e.g., “*I have racing thoughts*”), cognitive complexity (enjoying mental challenges; e.g., “*I like puzzles*”), self-control (planning and thinking deliberately; e.g., “*I plan trips well ahead of time*”), motor impulsiveness (acting quickly; e.g., “*I do things without thinking*”) and perseverance (stable lifestyle; e.g., “*I*

change jobs”). The three second-order factors are combinations of these basic factors: non-planning impulsivity (self-control and cognitive complexity), attentional impulsivity (attention factor and cognitive instability), and motor impulsivity (motor impulsivity and perseverance). The current study analyzed only the three *secondary* factors. Cronbach’s α s for the total BIS score was 0.84, and for the BIS-11 subscales: 0.76 for non-planning, 0.64 for attention, and 0.59 for motor.

Emotion regulation: was assessed with the optimism/mood regulation subscale score from the Polish version (Jaworowska et al., 2005) of the Schutte Self-Report Emotional Intelligence Test (SSEIT; Schutte et al., 1998). Sample items include: *“I have control over my emotions”*, *“When I experience a positive emotion, I know how to make it last”* (Petrides & Furnham, 2000; Saklofske et al., 2003). The SSEIT is an established measure of emotional intelligence (EI) based on the theoretical model introduced by Salovey and Mayer (1990). According to this model, EI is a composite of several emotional skills such as: perception, appraisal and expression of emotion; emotional facilitation of thinking; understanding, analyzing and employing emotional knowledge; and reflective regulation of emotions to further emotional and intellectual growth (Schutte et al., 1998; Jaworowska et al., 2005). Prior work on the SSEIT supported a four-factor solution (Petrides & Furnham, 2000; Saklofske et al., 2003) that corroborates the conceptual composites of this measure (appraisal of emotions, social skills, utilization of emotions, and optimism/mood regulation subscale). In this study the subscale optimism/mood regulation was chosen to evaluate emotion regulation. This specific subscale was demonstrated to have convergent validity with other measures of emotion regulation (Cho et al., 2015). Moreover, the optimism/mood regulation subscale was demonstrated to be associated with measures of neuroticism and extraversion from the NEO Five Factor Inventory (Saklofske et al., 2003), which reflect traits that are known to be associated with the regulation of negative and positive mood (Bar-On, 2000) and was shown to be correlated with other emotion-related variables such as coping styles and depressive affect (e.g., Goldenberg et al., 2006). Cronbach’s α for the optimism/mood regulation subscale in this study was 0.81.

2.3. Data analysis

First, a simple analysis of variance (ANOVA) was performed to assess the between-group difference among individuals with AUD and HCs in emotion regulation and impulsivity (attention, non-planning, and motor). An ANOVA was also used to test the between-group differences across basic demographic covariates (i.e., biological sex, age, education).

Next, a path model was estimated. The model included the main variables of interest (i.e., AUD status, emotion regulation, attentional impulsivity, non-planning impulsivity, motor impulsivity, age, education, and biological sex – coded: female = 0, male = 1). The model (see Figure 1) examined whether there was support for an indirect effect of alcohol dependence status on attentional, cognitive, and motor impulsivity via individual differences in emotion regulation. Finally, in additional analyses, we examined associations between emotion regulation and different facets of impulsivity separately in the AUD versus non-AUD group.

3. Results

The average age of onset of drinking problems in the group with AUD was 26.15 ± 9.3 years of age. The average duration of problem drinking was 18.9 ± 11.4 years. Individuals with AUD reported on average of 13.3 ± 10.9 days of drinking during the four weeks preceding their admission to the treatment center, with an average of 17.1 ± 7.2 standard drinks of alcohol drunk during a typical drinking day. An average score on the MAST in this group was 34.7 ± 9.6 , indicative of high severity of AUD symptoms.

Individuals meeting criteria for AUD were significantly older ($F = 28.11$; $p < 0.0001$), more likely to be female ($\chi^2 = 9.8$; $p = 0.007$), less educated ($F = 12.69$; $p < 0.0001$) and scored significantly higher on the MAST ($F = 793.23$; $p < 0.0001$) than HCs. The Bonferroni corrected ($p < 0.008$) between-group comparisons revealed that individuals meeting criteria for AUD obtained significantly higher attentional ($F = 26.38$; $p < 0.0001$), non-planning ($F = 55.96$; $p < 0.0001$) and motor ($F = 22.12$; $p < 0.0001$) impulsivity scores than HCs. Individuals meeting criteria for AUD also reported lower emotion regulation compared to HCs ($F = 19.32$; $p < 0.0001$). The detailed results of comparisons between the AUD and HC groups are presented in Table 1.

Figure 1 depicts estimated parameters for the path model. The proposed path model provided a good fit to the data ($\chi^2[3] = 2.91$, $p = 0.41$; CFI = 1.00; TLI = 1.00; RMSEA = 0.00). Parameters for paths leading from the covariates (i.e., age, education, and biological sex) to emotion regulation were constrained to zero in this model, as the parameter values were not significant. There was evidence for a direct effect of AUD status on emotion regulation, as well as attentional, motor, and non-planning impulsivity. Emotion regulation was negatively associated with attentional and cognitive (non-planning) impulsivity, but not with motor impulsivity. Moreover, there was evidence for two indirect pathways. First, there was evidence for a significant indirect effect of AUD status on attentional impulsivity via emotion regulation. Namely, AUD was associated with impairments in emotion regulation. In turn, poor emotion regulation was associated with greater attentional impulsivity. Second, there was evidence for a significant indirect effect of AUD status on non-planning impulsivity via emotion regulation. Namely, AUD was associated with impairments in emotion regulation. In turn, poor emotion regulation was associated with greater non-planning impulsivity. Since the association between emotion regulation and motor impulsivity was not observed, there was no evidence for an indirect effect of AUD status on motor impulsivity via emotion regulation. Parameter estimates and indirect effects are presented in Table 2.

3.1. Additional analyses

Additional analyses revealed *non-significant* correlations between emotion regulation and BIS total score ($r = -0.175$, $p = 0.09$), and BIS-11 subscales (motor, non-planning or attentional; $p = 0.12$, $p = 0.19$ and $p = 0.25$, respectively) among the HC group. In contrast, additional analyses revealed *significant* correlations between poor emotion regulation and higher BIS total score ($r = -0.246$, $p = 0.001$), as well as higher non-planning ($r = -0.321$, $p < 0.001$) and attentional impulsivity ($r = -0.265$, $p < 0.0001$) among individuals with AUD.

Similar to the HC group, there was not a significant association between emotion regulation and motor impulsivity ($r = -0.003$, $p = 0.9$) among individuals with AUD.

4. Discussion

In the current study, the association between a diagnosis of AUD, three facets of impulsivity, and emotion regulation were analyzed. As expected and consistent with prior reports (Lejuez et al., 2010; Petit et al., 2015), individuals diagnosed with AUD self-reported significantly lower emotion regulation skills and higher levels of impulsivity (in all three analyzed domains: cognitive, attentional, and motor) when compared to non-dependent HCs. Also, as predicted, impairments in emotion regulation were associated with non-planning and attentional impulsivity. However, emotion regulation was not associated with motor impulsivity. Consistent with our hypothesis, there was support for an indirect effect of AUD status on both non-planning and attentional impulsivity via emotion regulation, suggesting that emotion dysregulation partially explains the association between AUD and attentional and non-planning impulsivity. However, contrary to our hypothesis, there was no evidence for an indirect effect of AUD status on motor impulsivity via emotion regulation. In additional analyses, we observed that the association between poor emotion regulation and higher impulsivity was limited to individuals with AUD and was not observed among the non-AUD HC individuals.

While, there were a few studies investigating the association between emotion regulation and impulsivity in various clinical and non-clinical populations, to our knowledge this is the first study that takes into consideration AUD status. Overall, our results indicate a significant association between high impulsivity and poor emotion regulation in individuals with AUD. This is in line with prior work demonstrating that poor emotion regulation may be a contributor to impulsive behaviors among individuals with PTSD, eating disorders, or borderline personality disorder (Messman-Moore et al., 2010; Weiss et al., 2012; Williams et al., 2006), which are all well-established risk factors for alcohol-related problems. Interestingly, in our sample, this association was not present in healthy, non-AUD individuals. This is a novel and interesting finding suggesting that the significant association between poor emotion regulation and high impulsivity may be limited to groups with prior exposure to problematic alcohol use. The biological mechanisms underlying this pattern of findings remain unclear. Yet, it is possible that this link may be related to the influence of alcohol on the central nervous system (CNS), particularly dysfunction of the prefrontal cortex (Wang et al., 2016). As shown in functional magnetic resonance imaging (Brown et al., 2012), as well as event related potential (Messerotti Benvenuti et al., 2015) studies, emotion regulation and impulsivity rely on partially overlapping networks, situated mainly in prefrontal cortex. Therefore, generally speaking, individuals that are more impulsive may need greater response inhibition to counteract the tendency to act without thinking, which is elicited by the combination of high trait-related impulsivity and high emotional arousal. In individuals with AUD, the deleterious impact of ethanol on CNS functioning may result in structural and functional hypofrontality. That is, individuals with AUD may have fewer resources to regulate their emotions and behavior simultaneously. Therefore, individuals with AUD that experience emotional arousal may devote extensive resources to emotion processing, which leaves a limited amount of resources available for successful behavioral

control, cognitive analysis (attention), and careful decision making (Seo et al., 2016). In contrast, HCs may be more efficient, and have more available resources to facilitate both emotional and cognitive processes at the same time, without influencing the behavior of an individual. This may explain why emotion regulation and impulsivity were not associated in the HC group.

Our observation that emotion dysregulation partially explains the association between AUD and attentional and non-planning impulsivity may indicate that impulsive behaviors and drinking alcohol may be mutually enhancing emotion regulation strategies. Cyders and Smith (2008) posited, that rash action - such as heavy drinking - can be a response to intense emotion that provides either relief by providing a distraction from the source of the distress (negative reinforcement) or by enhancing a celebratory mood (positive reinforcement). Effective emotion regulation strategies facilitate non-impulsive goal-directed behavior while an individual experiences negative emotion (Gratz & Roemer, 2004). When facing stressful or challenging situations, individuals with ineffective emotion regulation strategies may struggle to regulate impulsive behaviors or decisions (Patton et al., 1995). Together, it might be posited that in the context of emotional distress, impulsive behaviors/decisions may be strongly related to maladaptive coping mechanisms with emotions, or difficulties controlling behaviors. However, there is no direct association between trait impulsivity and impulsive behaviors. It has been postulated that heightened levels of emotion dysregulation may add to trait impulsivity leading to clinical manifestations of behavioral undercontrol (Linehan, 1993). Although, drinking alcohol might serve as a secondary emotion regulation strategy, over time it could negatively influence brain structures related to behavioral control and affect regulation (prefrontal cortex), resulting in further emotional dysregulation (Kober, 2014) and impulsivity (Wang et al. 2016). Importantly, the cross-sectional nature of our study precludes us from inferring any causal associations. Our analyses focused on a specific pathway, whereby the effect of AUD led to problems with emotion regulation and impulsivity. However, the opposite pathway is also plausible, whereby impulsivity and emotion regulation may exacerbate subsequent AUD symptoms. The latter is also supported by numerous lines of evidence. Pedersen et al. (2016) showed that elevated levels of emotional impulsivity may place children with attention-deficit/hyperactivity disorder at increased risk for alcohol problems in adulthood. Laboratory experiments revealed that in non-dependent young individuals (VanderVeen et al., 2016), negative urgency was associated with amplified alcohol self-administration. Finally, results from recent meta-analyses confirm that problematic alcohol use, specifically amongst older adolescents, may be a consequence of a tendency to act rashly when in a heightened positive or negative mood (Smith & Cyders 2016; Stautz & Cooper 2013). Both paths (AUD to impulsivity and from impulsivity to AUD through emotion regulation) may be considered clinically relevant and feasible. Although, it is not possible to determine causal pathways or *predictors* of AUD symptoms, but rather *characteristics* of individuals with AUD, given the cross-sectional nature of the current data, it is plausible that this relationship is reciprocal. For example, one study that focused solely on urgency indicated that these factors enter into a rapid and vicious cycle, whereby drinking alcohol may lead to impairment in emotion regulation and higher impulsivity, which in turn may lead to more alcohol consumption, which continues the cycle (Riley et al., 2016).

Despite clear overlap between urgency and impulsivity, urgency is not synonymous with impulsivity, but represents only one aspect of this very broad concept. Recent work suggests very little overlap between urgency and behaviorally measured impulsivity (Cyders & Coskunpinar 2011). Importantly, the majority of studies that examine impulsivity, emotion regulation, and alcohol drinking problems focus on urgency as a main measure of interest. Our findings expand the results of studies on urgency and alcohol-related problems by demonstrating that poor emotion regulation may be associated with specific (i.e., attentional and cognitive) domains of impulsivity. More specifically, among individuals with AUD, emotion dysregulation likely impacts: 1) the inability to plan and think deliberately and postpone gratification, and 2) the inability to focus on a specific task and to analyze different elements of the situation. Notably, current research confirms that various dimensions of impulsivity may be related to different areas of alcohol-related problems. Specifically, cognitive impulsivity was shown to be associated with early onset of alcohol drinking problems, motor impulsivity was shown to be associated with poor alcohol treatment outcomes (Rubio et al., 2007), and attentional impulsivity was shown to be associated with higher severity of physical pain in participants with alcohol dependence (Jakubczyk et al., 2016). Contrary to most of the studies testing the association between emotions, impulsivity, and alcohol drinking, which were performed in young adults, our study was conducted in middle-aged participants and included individuals meeting criteria for AUD. These results may be especially significant in this population, given the established impact of impulsivity and emotion dysregulation on poor treatment outcomes among individuals with AUD (Kopera et al., 2014; Loree et al., 2014).

Contrary to our hypothesis, level of motor impulsivity was not associated with emotion regulation, and consequently the association between AUD status and emotion regulation was not influenced by motor impulsivity. Yet, levels of motor impulsivity were different between individuals meeting criteria for AUD and healthy controls. Other studies showed that the BIS subscale of motor impulsivity frequently does not correlate with objective measures of behavioral impulsivity (e.g. Jakubczyk et al., 2012). It is also important to note that the Cronbach's α for the motor impulsivity factor was low (0.59), which may suggest limited utility of this subscale in evaluating behavioral disinhibition. Interestingly, consistent with our results, Schreiber and colleagues (2012) found that in a group of healthy young adults with a history of recreational gambling, participants who reported high levels of emotion dysregulation also scored significantly higher on BIS attentional and non-planning impulsivity subscales, but not on motor impulsivity. Also, these authors did not observe a significant association between emotion regulation and a behavioral measure of motor impulsivity (i.e., Stop-signal Task). However, in a study utilizing a behavioral measure of *cognitive* impulsivity, Tsukue et al. (2015) did report a significant association between more frequent impulsive choices (using a delay discounting paradigm) and higher unfairness sensitivity (which may be considered an emotion regulation measure) in individuals diagnosed with AUD. These results suggest that impairments in emotion regulation are associated with impulsive choices (cognitive impulsivity) rather than impulsive, uncontrolled behaviors.

The assumption that poor emotion regulation may affect the association between AUD status and high impulsivity has significant clinical implications. One potentially important aspect

of effective treatment for addictive behaviors is to reduce the tendency to get involved in reckless, impulsive behaviors when highly emotional. The recent success of other personality-based interventions (Conrod et al., 2011) suggests the value of such approaches. Dialectical Behavior Therapy (Dimeff & Linehan, 2008) is one prominent intervention that emphasizes teaching skills to enable one to respond adaptively to the experience of extreme emotional states (Smith & Cyders, 2016). The possibility that chronic alcohol drinking may significantly complicate the processing of emotional information while making decisions indicates that therapeutic and preventative strategies focused on expanding abilities/resources responsible for these two processes may have utility.

Several study limitations should be noted. Most importantly, the cross-sectional nature of our study precludes us from inferring any causal associations. It will be important to test similar models using longitudinal study designs. Moreover, we recruited participants from an inpatient treatment program for AUD, who were likely to have a more severe course of the disorder. Therefore, it is plausible that our group was comprised of more impulsive individuals. At the same time, due to inclusionary criteria of the treatment program where the study took place, our study group did not include patients with major depressive disorder. While we admit that the restriction of the study group only to inpatients might be a serious disadvantage of the study, we believe that on the other hand, from a clinical perspective, it may still be very valuable to investigate this group of individuals; this group corresponds to patients that are likely realistic targets for therapeutic interventions. Contrary to other studies (e.g., Weiss et al., 2012), the results of our study revealed only partial indirect effects in the analyzed models. Therefore, the presented models represent only one potential mechanism that may underlie complex, mutual associations between AUD status and impulsivity. Another limitation of our study is the fact that the majority of the study group was comprised of men and the prevalence of women was higher in the AUD group. The statistical analyses did not reveal a significant effect of biological sex on the analyzed associations; yet, it is still unclear whether there are any differences in terms of the association between problematic alcohol use, emotion regulation, and impulsivity for men compared to women. It would be informative to conduct such comparisons in future studies. Moreover, although the racial and ethnic distribution in our sample was comparable to the general Polish population (according to government statistics non-White individuals comprise less than 0.01% of the Polish population) and individuals seeking substance use treatment, it is likely that our findings may not generalize to samples that are more racially and ethnically diverse. It will be important that future work investigate these associations in a more diverse sample. Another disadvantage of the project is the reliance on subjective, self-report measures of impulsivity and emotion regulation. Also, the indicators of internal consistency for attentional and, especially, motor impulsivity subscales of the BIS were relatively low. Future studies would strongly benefit from utilizing behavioral measures of these features.

5. Conclusions

Our results provide evidence for an indirect effect of AUD status on impulsivity via poor emotion regulation. However, additional studies using a larger and more representative clinical sample that utilizes more comprehensive evaluations of impulsivity and mood

regulation over time should be conducted to confirm these potentially clinically important findings.

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Figure 1. Path Model. Indirect effects of alcohol dependence status on impulsivity factors via emotion regulation. Estimated unstandardized path coefficients. * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 1

Comparison between individuals with alcohol use disorder and health controls

	AUD	HC	p
Age (years)	44.33±11.4	36.16±13.3	<0.0001
Education (number of classes completed)	12.23±2.8	13.66±3.4	<0.0001
Attentional impulsivity (BIS-11)	18.15±2.9	16.06±3.5	<0.0001
Motor impulsivity (BIS-11)	24.98±4.0	22.66±3.5	<0.0001
Non-planning impulsivity (BIS-11)	28.0±4.6	23.68±4.4	<0.0001
Global impulsivity	71.15±9.2	62.40±9.1	<0.0001
Emotion regulation (INTereg)	36.92±5.5	40.63±8.2	<0.0001
Biological sex (% women)	17.8	5.4	0.007

BIS – Barratt's Impulsiveness Scale, AUD – alcohol use disorder, HC – healthy controls

The data is presented by means±standard deviation

Table 2

Path Model: selected results supporting indirect effects of alcohol dependence status on impulsivity factors via emotion regulation.

	<i>a</i>	<i>b</i>	<i>c</i>	<i>c'</i>	<i>ab</i>	SE	p
ATTENTIONALIMPULSIVITY	-4.030***	-0.104**	2.064***	1.646***	0.417***	0.128	0.001
MOTORIMPULSIVITY	-4.030***	-0.030	2.690***	2.570***	0.121	0.184	0.513
NON-PLANNINGIMPULSIVITY	-4.030***	-0.167***	3.903***	3.230***	0.673***	0.163	< 0.001

Note. Unstandardized path coefficients. *a* = effect of alcohol dependence on emotion regulation; *b* = effect of emotion regulation on impulsivity factors; *c* = total effect of alcohol dependence on impulsivity factors; *c'* = direct effect of alcohol dependence on impulsivity factors. *ab* = indirect effect of alcohol dependence on DV.

*
p < .05,

**
p < .01,

p < .001.