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Behavioral and Biological Indicators of Impulsivity in the Development of Alcohol Use, Problems, and Disorders

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Abstract

Alcohol Use Disorders (AUDs) are a devastating public health problem. The construct of impulsivity is biologically-based and heritable, and its various dimensions are relevant for understanding alcohol use. The goal of the current manuscript is to review recent behavioral and biological research examining various dimensions of impulsivity, and their relation to AUDs from risk for initial use through dependence and relapse. Moreover, we also highlight key psychological variables including affective processes as they relate to current use and early indications of alcohol problems, as well as psychopathology, violence, and aggression in relation to AUDs. Each section includes a critical summary and we conclude the review with future directions focused on issues relevant to measurement, causality, and intervention. Throughout the review, we attempt to be as specific as possible about the dimensions of impulsivity being referenced, while attempting to draw parallels and highlighting differences as the existing literature allows.

Keywords

impulsivity; AUDs; review; behavior; biology

Problematic alcohol use is a devastating public health issue. Recent research has focused on various aspects of impulsivity as important determinants of alcohol use and related problems. Impulsivity is a multidimensional construct that includes biologically-based and heritable components (cf. Evenden, 1999). There is both theoretical and empirical disagreement regarding the most appropriate categorization of the various aspects of impulsivity, ranging from as few as two (Reynolds, Ortengren, Richards, & DeWit, 2006) through as many as five distinct dimensions (Meda et al., 2009). Across these dimensions, however, there are several commonly cited definitions including diminished ability to focus

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on tasks at hand (i.e., attentional impulsiveness) and/or persist or persevere in tasks (i.e., motor impulsiveness); a tendency to act on the spur of the moment and poor future planning (i.e., non-planning impulsiveness; Patton, Stanford, & Barrett, 1995; Eysenck, Eysenck, & Barrett, 1985; Whiteside & Lynam, 2001); diminished ability to delay gratification or heightened discounting of reward as a function of delay as well as hypo- and hyper-sensitivity to reward and punishment (Ainslie, 1975; Gray, 1987; Mischel, Shoda, & Rodriguez, 1989); poor response inhibition and increased passive avoidance (Logan, 1994; Newman, Patterson, & Kosson, 1987); and diminished ability to regulate emotion, sometimes referred to as 'urgency' (Whiteside & Lynam, 2001)¹. Although these categories fit well with theory and empirical evidence and correspond with several widely accepted operational definitions, the exact nature of the dimensions remains controversial, and there is evidence for both relatedness (Reynolds et al., 2006; Bornovalova, Lejuez, Daughters, Rosenthal, & Lynch, 2005) as well as independence (Smith, Fischer, Cyders, Annus, Spillane, & McCarthy, 2007) among the measures.

The goal of this manuscript is to review recent behavioral and biological research aimed at understanding the relationships between impulsivity and alcohol use phenomena. The review is organized into three sections, corresponding roughly to three stages of alcohol use: i) Alcohol use vulnerability and initiation, ii) Current use and early indications of alcohol problems, and iii) Alcohol use disorders (AUDs)². Within each section, we review selected animal and human studies linking impulsive behaviors and alcohol. In particular, we highlight key psychological variables that affect these relationships, including affective processes during the development of alcohol problems, and psychopathology, violence, and aggression once AUDs have developed. Each section includes a critical summary and we conclude the review with future directions focused on issues relevant to measurement, causality, and intervention. Throughout the review, we will attempt to be as specific as possible about the dimensions of impulsivity being referenced, while attempting to draw parallels and highlighting differences as the existing literature allows.

Alcohol Use Vulnerability and Initiation

Several studies with laboratory animals have linked measures of impulsivity to the initiation of alcohol self-administration or acute alcohol responses. One of the most widely cited studies used delay discounting as a predictor of alcohol self-administration (Poulos, Le, & Parker, 1995). These authors reported that male rats showing steeper discounting of delayed rewards subsequently self-administered more alcohol in a test of consumption. Mitchell, Reeves, Li, & Phillips (2006) examined the extent to which discounting predicted the degree of locomotor stimulation after alcohol in mice; locomotor stimulation is considered an indicator of rewarding effects. The mice that discounted more steeply exhibited *less* locomotor stimulation upon initial exposure to ethanol. However, after repeated exposure to alcohol, these same animals exhibited greater alcohol-induced locomotor activity. Thus, in rats, impulsive discounting showed a more general link to alcohol use, whereas with mice, more impulsive discounting was only related in the expected direction after repeated exposure to alcohol.

¹It should be noted that some researchers have included yet other dimensions as aspects of impulsivity, including for example sensation seeking and risk taking propensity/risky decision making (Bechara, Damasio, Damasio, & Anderson 1994; Whiteside & Lynam, 2001). However, for both theoretical and empirical reasons, these may not represent core features of 'impulsivity' (e.g., Lejuez, Aklin, Bornovalova, & Moolchan, 2005; Reynolds, Ortengren, Richards, de Wit, 2006), and therefore, will be mentioned here only when they are directly related to research on one of the four dimensions outlined above.

²As with impulsivity, there is often a lack of consistency across studies in the measurement of alcohol use and patterns. For the purpose of the review, we refer to "social drinking" individuals as those who report no alcohol-related problems, "problem drinkers" as those who report alcohol related problems but did not meet criteria for a DSM-IV diagnosis (or the study did not assess for DSM-IV diagnoses) or those with an AUD as those who met criteria for Abuse or Dependence (APA, 2000).

Several lines of evidence suggest that forms of impulsivity are related to initiation of drinking in humans. Dougherty, Mathias, Tester, & Marsh (2004) used behavioral measures of impulsivity to compare social drinking women who reported early or late onset of drinking. The women were compared using the Immediate (IMT) and Delayed Memory Tasks (DMT), which are considered measures of response inhibition and passive avoidance. Women who reported early onset of drinking made more errors of commission on these tasks compared with the late-onset group. Across the sample, age at first drink was significantly negatively correlated with DMT commission errors. These results support the idea that this form of poor behavioral inhibition is associated with early onset alcohol use, even among non-problem social drinkers. Another study examined delay discounting in social drinking college students in relation to age of first alcohol use (Kollins, 2003). Students who reported earlier alcohol use also discounted delayed, hypothetical rewards more steeply than students who initiated drinking at a later age. Both of these studies suggest that more impulsive individuals, assessed by two different indices of impulsivity, started using alcohol earlier than less impulsive individuals. These studies do not rule out the possibility that alcohol effects may have interacted with developmental trajectories to affect impulsivity indices.

Many studies have examined trait-based aspects of impulsivity as predictors of early use of alcohol and other drugs (Kirisci, Tarter, Reynolds, & Vanyukov, 2006; Clark, Cornelius, & Kirisci, 2005; Tarter, 2002; Lejuez et al., 2007). In an extensive series of longitudinal studies, Tarter and colleagues (1999) have reported that adolescents who exhibit behavioral dysregulation, as measured by both behavioral and self-report measures, are more likely to use drugs and alcohol. Dom, D'Haene, Hulstijn, & Sabbe (2006) found that early-onset problem drinkers scored higher than late-onset problem drinkers on the Barratt Impulsivity Scale (BIS), as well as measures of sensation seeking, and aggressiveness. McGue, Iacono, Legrand, Malone, & Elkins (2001) found that measures of behavioral disinhibition (including inattentiveness, hyperactivity, and impulsivity) at age 11 predicted drinking onset by age 14, and that age of first use of alcohol was associated with antisocial personality characteristics and "lack of constraint." Lejuez et al. (2007) reported that the Eysenck Impulsiveness Questionnaire (EIQ; Eysenck et al., 1985) was correlated with a composite of risk-taking behaviors in 98 African American adolescents, aged 14–18. Scores on the Eysenck scale also were correlated with age at first drink as well as past year frequency, even at this early stage of alcohol experimentation (unpublished data). Von Diemen et al. (2008) found that adolescents ($n = 418$) with AUDs as well as other substance use disorders (SUDs) demonstrated both an earlier age of first drink as well as higher scores on the BIS-11. Taken together, there is considerable accumulated evidence that various indicators of impulsiveness, including both self-report measures and behavioral indices, are predictive of early onset of drinking.

Biological Extensions

The biological mechanisms that link impulsive behaviors to alcohol use are poorly understood. There is some evidence that individuals who are genetically at risk for alcoholism also perform more impulsively on at least one measure of impulsive behavior, delay discounting. Petry, Kirby & Kranzler (2002) found a modest relationship between family history of alcoholism and delay discounting. Women, but not men, with a parental history of alcohol dependence had higher discount rates than women without a parental history, indicating that they preferred more immediate, over more delayed, rewards. Other studies have examined brain morphology in relation to measures of impulsivity and alcohol consumption. Hill et al. (2009) examined the volume of the orbitofrontal cortex (OFC) of a sample of 107 young adults (mean age 17.6) from either high-risk AUD families or control families. The OFC, which is thought to be involved in emotional regulation and impulsivity,

showed decreased right/left volumes in high-risk offspring in comparison with control subjects. Findings also demonstrated relationships between OFC volumes and genetic and self-report information. Specifically, lower right volume was significantly associated with variation in the serotonin transporter (5-HTT) and brain-derived neurotrophic factor (BDNF) genes, and white matter ratios were positively correlated with a self-report measure of self-control from the Multi-dimensional Personality Questionnaire (MPQ; Tellegen & Waller, 1992), demonstrating that reduced OFC white matter is related to self-control aspects of impulsivity (i.e. urgency). These two studies provide limited evidence for a biological association between impulsive tendencies and alcohol consumption.

Integration and Critical Review

We have reviewed evidence from both nonhumans and humans indicating that impulsivity is a predictor of early alcohol use. Although there is evidence in both nonhumans and humans that certain indices of impulsive behavior are related to initiation of alcohol use, the studies vary widely in their methodologies. In particular, they utilize very different measures of impulsivity, both within the dimensions we identified above and also across dimensions. Thus, although poor response inhibition (e.g., the IMT and DMT tasks; Dougherty et al., 2004), greater delay discounting, and in humans, self-report measures of impulsive personality are all related to early alcohol use, few studies use more than one measure, making it difficult to integrate the different findings across species, tasks, and measures of alcohol consumption. Similar problems exist in the self-report assessments. For example the relationships among measures such as the EIQ, BIS, different variations of the BIS, and the MPQ are poorly understood. In the future, it would be valuable for authors to justify clearly their selection of measures, including behavioral tasks, self-report measures, and measures of alcohol consumption, and to integrate their findings with the larger literature.

An understanding of biological mechanisms underlying the relationship between impulsivity and initiation of alcohol use also depends on careful selection and description of the behaviors under study. We reviewed two studies demonstrating relationships between impulsivity and family history of alcoholism; Petry et al. (2002) focused on delay discounting impulsivity, whereas Hill and colleagues (2009) focused on self-control aspects of impulsivity. It would be of great interest to examine self-control in a design such as that used by Petry et al. and to examine discounting in the design used by Hill et al. In sum, future studies will be enriched by inclusion of a range of measures, addressing more than a single dimension of the complex construct of impulsivity, including a clear strategy for differentiation and causal directions encompassing biological evidence.

Current Use and Early Indications of Alcohol Problems

Quite a number of studies have assessed measures of impulsivity in relation to habitual alcohol use, often with the goal of identifying early indicators of problem use. For example, Grau & Ortet (1999) examined the Karolinska Scales of Personality (KSP) and the Eysenck Personality Questionnaire Revised (EPQ-R) in relation to alcohol consumption rates in a sample of 149 social drinking women. They found that alcohol consumption was positively related to disinhibitory personality traits assessed by the KSP, including sensation seeking (termed “monotony avoidance” and defined as “avoiding routine, need for change and action”) and impulsivity (defined as “acting on the spur of the moment, non-planning”). Kollins (2003) found that steeper discounting in a hypothetical money reward task was related to alcohol-related problems (e.g., passing out) in a sample of social drinking college students. Similarly, Vuchinich & Simpson (1998) demonstrated that heavy social drinkers and problem drinkers showed greater delay discounting than lighter social drinkers and also scored higher on various self-report measures, including the Sensation Seeking Scale (SSS; Zuckerman, 1979), the Stanford Time Perspective Inventory (STPI; Zimbardo, 1990), and

the Consideration of Future Consequences scale (CFCS; Strathman, Gleicher, Boninger, and Edwards, 1994). Notably, however, discounting showed only a very modest relationship to the self-report measures of sensation seeking, consideration of future consequences, and time estimation, suggesting that they measure distinct dimensions of impulsivity, if even considered dimensions of impulsivity.

Other lines of evidence have suggested unique relationships between certain dimensions of impulsivity and distinct aspects of problem alcohol use. MacKillop (2007) found that problem drinkers scored higher than social drinkers on several measures of impulsivity, including the EIQ and the STPI but *not* on a delay discounting task. These findings suggest that alcohol misuse in college students may be related in particular to non-planning, a dimension that may not be captured in the discounting procedure. Magid, MacLean, & Colder (2007) found that different personality traits were related to alcohol *use* as compared to alcohol *problems*. In a sample of 310 college students, they found that impulsiveness, measured using the Impulsiveness Monotony Avoidance scale (IMA; Schalling, 1978), was related to alcohol *problems*, whereas a different scale, measuring sensation seeking, was related to alcohol *use* (Magid, MacLean, & Colder, 2007). In a separate study, Magid & Colder (2007) examined the UPPS Impulsive Behavior Scale (Whiteside & Lynam, 2001) in relation to alcohol use and alcohol-related problems in 267 undergraduates. Of the four factors they identified on this scale, sensation seeking and (lack of) premeditation were related to high alcohol consumption, but not to alcohol-related problems, whereas high scores on the urgency subscale and low scores on perseverance were related to alcohol problems, regardless of level of alcohol use.

Affective Processes—In addition to considering impulsivity at a “cold” level, impulsivity may also vary in relation to dynamic affective processes. In particular, a dimension of impulsivity referred to as “negative urgency” (Whiteside & Lynam, 2001) is defined as non-planning tendencies under the influence of negative affective states. When studied in the context of alcohol use, negative urgency mirrors the negative reinforcement model. For example, Anestis, Selby, & Joiner (2007) examined the ability of negative urgency to predict negative reinforcement patterns of drinking (“drinking to cope”) among a sample of college students ($n = 70$) at two time points approximately 1 month apart. Urgency predicted “drinking to cope” at both Time 1 and Time 2, and changes in urgency from Time 1 to Time 2 also predicted changes in “drinking to cope” behaviors. Thus, greater increases in negative urgency were significantly associated with higher levels of negative reinforcement-driven drinking behaviors. This idea that affective processes affect impulsive behaviors offers a promising new direction for studying the role of impulsivity in alcohol use.

Cyders et al. (2007) extended the idea of “urgency” to “positive urgency,” which refers to the tendency to act rashly when experiencing extremely positive emotion. Thus, just as negative emotional states can increase impulsive behaviors, so can positive emotional states (Cyders & Smith, 2008; Whiteside & Lynam, 2001). Cyders et al. (2007) reported that positive urgency was related to both quantity of use and alcohol-related problems, independently of the effect of negative urgency, lack of planning, lack of perseverance, and sensation seeking.

Another valuable direction of research concerns the role of affect and affect dysregulation in the relationship between impulsivity and alcohol use and related early problems. Colder & Chassin (1997) assessed joint effects of self-reported impulsivity, positive affect, and negative affect on adolescent alcohol use and alcohol-related impairment. Impulsivity was assessed in this study using a modified scale based on the Revelle version of Eysenck’s impulsivity scale (Revelle, Humphreys, Simon, & Gilliland, 1980). Specifically, they

focused on the response inhibition and urgency dimensions of impulsivity, and as such, deleted two items from the Revelle scale that focused more on sensation seeking rather than the “core” of impulsivity, which they deem to be urgency. They found that impulsivity moderated the effects of positive affect on both alcohol use and alcohol-related impairment. They also found that impulsive adolescents with low positive affect consumed more alcohol and experienced more alcohol-related impairment compared to impulsive adolescents with high positive affect or non-impulsive adolescents (Colder & Chassin, 1997). Similarly, another study (Simons, Carey, & Gaher, 2004) examined two aspects of affect dysregulation as risk factors for alcohol-related problems in an undergraduate sample. They found that affective vulnerability and impulsivity increased risk for alcohol problems even after controlling for gender and lifetime-use frequency. Further, this relationship was greatest among participants with the highest levels of impulsivity, defined using the emotional control questionnaire (Roger & Najarian, 1989) that assesses control over impulses, both benign and aggressive. The results demonstrate that individual differences in impulse control associated with alcohol-related problems in young adults.

In another study, Simons (2003) examined predictors of alcohol use-related problems in undergraduates over a 30-day interval. Baseline scores on the Eysenck Impulsiveness Scale (EIS; Eysenck & Eysenck, 1977), affective vulnerability, social norms, and perceived conflict between personal strivings and alcohol use predicted alcohol use-related problems at the second assessment. Structural equation models indicated that impulsivity and affective vulnerability were directly related to use-related problems. Impulsivity moderated the association between alcohol use and alcohol use-related problems; that is, use frequency was associated more strongly with use-related problems among more impulsive participants (Simons, 2003). Taken together, these studies indicate the importance of considering emotional processes in the relationship between impulsivity and alcohol.

Representing an important methodological advancement, Simons and colleagues (2005) investigated the relationship between affective states and alcohol consumption using real-time assessment. College students ($n = 56$) first completed a personality measures of impulsivity (EIQ) and then provided experience sampling data for two-weeks including measures of negative and positive affective states, alcohol use and related problems. Although higher levels of both positive and negative affect throughout the day were associated with higher levels of alcohol use that evening, only negative affect was associated with alcohol-related problems (controlling for levels of use). Impulsivity is associated with higher levels of alcohol use as well as problems, and further, impulsivity moderated the relationship between negative affect and problems as well as between alcohol use and problems, such that individuals with higher levels of impulsivity showed higher rates of alcohol-related problems in both scenarios (Simons et al., 2005).

Biological Extensions

Studies with laboratory animals may shed light on the biological mechanisms linking impulsive behaviors with alcohol consumption. Wilhelm & Mitchell (2008) compared delay discounting in ethanol-naïve rats selectively bred for high or low alcohol consumption. High alcohol drinking rats discounted delayed and probabilistic rewards more steeply than low drinking counterparts. Similarly, Oberlin & Grahame (2009) reported that alcohol-naïve mice selected for alcohol preference exhibited steeper discounting than low-preferring mice. Wilhelm, Reeves, Phillips, & Mitchell (2007) examined delay discounting and responding in a go-no go task in short-term selected lines of mice bred to drink high or low amounts of 10% ethanol. Unlike the Oberlin & Grahame (2009) findings, they found no differences between groups in delay discounting. However, using a more circumscribed measure of inhibition, the high drinking mice did make more responses during the pre-cue period on the go-no go task. Thus, although the study found no differences in delay discounting, the mice

bred for higher alcohol consumption had impaired response inhibition on at least one measure. Such response inhibition data are consistent with Logue, Swartz, & Wehner (1998), who assessed the genetic relationship between nose poking efficiency and subsequent consumption of a 10% ethanol solution vs. water in a 2-bottle choice procedure, using 13 inbred strains of mice. Strains that confined their nose poking to periods when it yielded rewards (more efficient behavior) also consumed less ethanol. Logue and colleagues interpreted this as indicating a genetic relationship between impulsivity and ethanol consumption. Using the same procedure, Bowers & Wehner (2001) found that mice evidencing the gene encoding the neuronal-specific γ subtype of protein kinase C (PKC γ) consumed more alcohol and also demonstrated increased impulsivity in an appetitive-signalized nose poke task when compared with control mice. PKC γ may be an important mechanism within the cell that mediates one or more neurochemical pathways relevant to an increased predisposition to alcoholism and behavioral impulsivity (Bowers & Wehner, 2001). Studies delineating additional mechanisms underlying these genetically-based relationships between impulsivity and propensity to develop AUDs will be an important future research direction.

Alcohol can also exacerbate impulsive tendencies. Johansson, Bergvall, & Hansen (1999) found that rats with ibotenic acid-induced neuron loss in the basal forebrain showed increased alcohol consumption, which augmented aggression, insensitivity to punishment, and inattention to future rewards. These characteristics resemble those of alcohol abusers who demonstrate high rates of violence, impulsivity, and signs of reduced serotonin functioning in the brain. Interestingly, the same results were not evident with serotonin depletion alone, suggesting that these effects are also mediated by non-serotonergic mechanisms in the forebrain. More recently, Oliver, Ripley & Stephens (2009) examined the acute effects of alcohol on attention and response control aspect of impulsivity using a 5-choice serial reaction time task in two mouse strains. Although alcohol did not affect attention and impulsivity in the standard procedure, it did increase premature responding in the long inter-trial-interval sessions. Using rats, Nasrallah, Yang and Bernstein (2009) examined whether alcohol exposure in adolescence alters decision making and risk preference in adulthood. Using a probability-discounting instrumental responses task that offered a choice between a large uncertain reward and small certain rewards, these authors assessed risk-based choice either 3 weeks or 3 months following alcohol exposure. While control animals' performance closely conformed to the predictive model of risk-neutral value matching, rats that consumed high levels of alcohol during adolescence violated the predictive model and showed greater risk preference. Furthermore, evidence of such risk preference was also present when choice was assessed 3 months following discontinuation of alcohol access when the animals were adults. These recent studies indicate that acute alcohol exposure and high levels of repeated and continuous alcohol exposure especially in adolescents increases premature responding and risky decision making respectively. It has yet to be determined whether such alcohol effects on impulsivity are persistent or affect development of alcohol problems.

In humans, the electrophysiological measure of P3 amplitude has been associated with specific dimensions of disinhibited personality (impulsivity, social deviance, harm avoidance, and excitement seeking), and disinhibition appears to mediate the association between P3 and alcohol problems. Justus, Finn, & Steinmetz (2001) found that social deviance, impulsivity, and alcohol problems in young adults were associated with lower P3 amplitude, but only in male subjects. Social deviance mediated this association between P3 and alcohol problems, as well as the association between impulsivity and alcohol problems. Thus, smaller P3s are strongly associated with antisocial, defiant, and impulsive traits, which might, in turn, increase the risk for continued alcohol problems.

Integration and Critical Review

Among social drinkers and early problem users, alcohol consumption is related to self-report aspects of impulsivity, including sensation seeking and non-planning, as well as behavioral measurement of impulsivity (i.e., steeper delay discounting). Although the dimensions of impulsivity (e.g., delay discounting and non-planning) are often independent of one another, it has also been reported that certain dimensions of impulsivity are more related to alcohol use than to alcohol-related problems. These findings with different dimensions of impulsivity and their relation to alcohol-related outcomes both in behavioral and biological research support the need to distinguish between distinct constructs or dimensions of impulsivity, to fully understand the role of this category of behaviors in alcohol use. As compared to vulnerability, an issue of important consideration for use and alcohol problem development is the exacerbating role that alcohol may begin to have on impulsivity, producing a bi-directional relationship (Anestis, Selby, & Joiner 2007). Controlled studies examining this interactive relationship are lacking and should provide a focus for future research. In addition, future studies should consider the extent to which different dimensions of the construct are more or less influenced by continued alcohol use and associated problems.

Finally, the studies reviewed in this section indicate the importance of considering the interactive roles of emotion, affect regulation, and biological factors in the relationship between impulsivity and alcohol consumption and alcohol-related problems. Consistent and standardized methodologies should be used across studies, to investigate the complex interaction between the factors. Moreover, real-time assessments involving either behavioral tasks or experiential sampling are to be preferred over retrospective or subjective self-assessments, as they enable real examination of how emotion and affective factors may influence responding in the moment. Additionally, assessments of relations between affect and behavior should be completed in more heterogeneous subjects samples (older, nonstudent, psychiatrically more complex), and for longer assessment periods to gain a more thorough understanding of these relationships.

Alcohol Use Disorders (AUDs)

Impulsivity is also associated with problem alcohol use, including diagnoses of both alcohol dependence and abuse. Using a longitudinal design, Sher, Bartholow, & Wood (2000) demonstrated that impulsivity, defined as the “novelty seeking” (NS) subscale of the Tridimensional Personality Questionnaire (TPQ), prospectively predicted AUDs six years later in a high-risk college sample, thus suggesting that sensation seeking aspects of impulsivity may be relevant in understanding the development of AUDs. Von Demien et al. (2008) found that drinkers who met DSM-IV criteria for abuse or dependence scored higher on the BIS than non-alcohol abusing controls. Further, when age of first drink was included in the model, the strength of the relationship between BIS scores and SUDs decreased, suggesting that “part of the effect of impulsivity on the odds of SUDs is explained by a reduction in age of first drink” (von Demien et al., 2008). Ketzenberger & Forrest (2000) also found that BIS scores differed in individuals who did or did not meet DSM-IV criteria for an AUD. These relationships were evident in individuals grouped by decade from age 20 to 60, suggesting that the relationship does not vary substantially with the progression of problem drinking.

Numerous studies have found that alcohol dependent individuals demonstrate high rates of delay discounting compared to non-alcohol dependent individuals (Bjork, Hommer, Grant, & Danube, 2004; Finn, 2002; Mitchell, Fields, D’Esposito, & Boettiger, 2005). For example, Petry et al. (2001) compared discounting rates between money and alcohol rewards in 19 current alcohol abusers, 12 currently abstinent former alcohol abusers, and 15

controls (no history of problem drinking). They used a titration procedure to determine indifference points at various delays, for each of the following rewards: \$1,000, \$100, 150 bottles of an alcoholic beverage, and 15 bottles of an alcoholic beverage. Active alcohol dependent individuals exhibited the most rapid discounting across three conditions of money and alcohol rewards, whereas currently abstinent former alcohol abusers were intermediate, and discounting was lowest for controls. In sum, the data demonstrated alcohol abusers to have steeper rates of delay discounting compared to controls, and in particular, the current users demonstrated the steepest rates of discounting.

Psychopathology, Violence, and Aggression—Psychopathology is also linked to alcohol use in psychiatric populations. For example, Rubio et al. (2007) indicated individuals with an AUD differed from controls on some dimensions of impulsivity such as delay discounting, but those with an AUD and a cluster B personality disorder evidenced additional impulsivity related impairment in other aspects of impulsivity, such as sustained attention and response inhibition. Moreover, results indicated some distinction among the cluster B personality disorders themselves (e.g., more commission errors on a continuous performance test associated with borderline personality disorder and poorer efficiency in a differential reinforcement of low rate responding associated with antisocial personality disorder). Specific to antisocial features, Whiteside & Lynam (2003) compared self-reported impulsivity using the UPPS Impulsive Behavior Scale (Whiteside & Lynam, 2001) among alcohol abusers high or low in antisocial personality traits and a control group. Individuals high on antisocial personality traits had significantly high scores on all UPPS scales (urgency, lack of premeditation, lack of perseverance, and sensation seeking), while the individuals low in antisocial personality traits and controls only had significant elevations on the Urgency subscale. However, when analyses were conducted controlling for rates of psychopathology, no differences in impulsivity were found among groups, suggesting that impulsivity may be more closely related to psychopathology than alcohol abuse alone. These results indicate the importance of considering concurrent psychopathology for understanding the link between impulsivity and AUDs.

Impulsivity and alcohol use are also closely tied to violence and aggression. Alcohol dependence is associated with an increased risk for aggressive behavior, which may be related to certain forms of impulsivity. This association may either result from pre-existing impulsive tendencies, or it may result from—or be exacerbated by—alcohol use, which further increases the risk of aggression. Dom, Hulstijn, & Sabbe (2006) compared impulsivity, sensation seeking and aggression in early- and late-onset alcohol dependent individuals. Those with early onset scored higher on measures of aggression than those with late onset, and further, the groups differed on questionnaire measures of impulsivity even after controlling for the effect of aggressiveness (Dom et al., 2006). Bjork and colleagues (2004) examined these relationships using behavioral measures of impulsivity, comparing detoxified alcohol dependent individuals ($n = 130$) to healthy controls ($n = 41$) on behavioral measures including two risk-taking tasks designed by the authors, a delay discounting task, and self-report measures (SSS, BIS and a self-report measure of aggression). Compared to the healthy controls, alcohol dependent individuals demonstrated poorer response inhibition, higher rates of delay discounting, higher rates of risk responses in the risk-taking paradigm, and higher self-reported BIS and aggression scores. Interestingly, aggression was significantly correlated with steepness of delay discounting for the entire sample, supporting the idea that aggression is related to at least one behavioral form of impulsivity. Further, alcohol dependent males with an earlier age of problem drinking onset and with a problem-drinking parent demonstrated higher rates of aggression compared to those with later onset problem drinking and no problem-drinking parent (Bjork et al., 2004). Parrott & Giancola (2006) also investigated the relationship between alcohol use, impulsivity and aggression in

male and female drinkers with an AUD between 21 and 35 years of age, using the BIS. Although alcohol dependence did not mediate the relationship between impulsivity and aggression, the attentional and motor impulsivity subscales of the BIS mediated the relationship between alcohol dependence and physical aggression in men.

Biological Extensions

Genetic mechanisms—Studies with both humans and nonhumans suggest that the traits associated with AUDs have a genetic basis. Thus, identifying the genes involved could help to identify neurochemical pathways that are important in the development of alcohol abuse. Preuss et al. (2001) investigated the association between impulsivity as measured by the BIS in relation to the G-1438A polymorphism in the promoter region of the 5-HT_{2A} receptor gene in alcohol dependent individuals. Participants with low scores on the BIS reported a later age of onset of alcoholism than the subjects with high scores, and impulsivity was associated with 5-HT_{2A} 1438A alleles (Preuss et al., 2001). Limosin et al. (2003) investigated the dopamine receptor D2 (DRD2; specifically the TaqI A polymorphism) gene in relation to BIS scores among alcohol dependent individuals ($n = 92$). A2/A2 and A1/A2 genotypes scored higher on global impulsiveness than A1/A1 genotype. The authors propose that delay discounting and other aspects of reward-dependent impulsiveness may constitute a risk factor for the development of alcohol dependence, and that this could be partly mediated by the DRD2 gene (Limosin et al., 2003). Angheliescu et al. (2005) examined the association of the tryptophan hydroxylase (TPH) intron 7 A218C alleles with dimensions of the Temperament and Character Inventory (TCI; Cloninger & Syrakic, 1997) in alcohol-dependent individuals and age-matched controls. The TPH enzyme, which is implicated in serotonin biosynthesis, has been linked to alcoholism and impulsivity (Nielsen et al., 1998; Virkkunen, Godman, Nielsen, & Linnoila, 1995). Although TCI scores were not related to the TPH genotypes in the complete sample the alcohol-dependent group included a higher proportion of individuals homozygous for certain TPH genotypes. Thus, although TPH polymorphisms may not explain variation in the expression of impulsivity in either alcohol dependent individuals or healthy controls, the difference in the TPH genotype distribution in alcohol dependent individuals suggests that there is a connection between serotonin function and alcoholism.

Neuroanatomical substrates—Recent evidence has suggested a disordered reward-based impulsivity in alcoholism. Wrase and colleagues (2007) reported that abstinent alcohol dependent individuals exhibited blunted engagement of ventral striatum (a key reward system brain region) during an incentive processing task that did not involve alcohol-related stimuli, but enhanced ventral striatal engagement when presented with an alcohol cue that induced craving. A more recent study (Beck, Schlagenhauf, Wustenberg, Hein, Kienast, Kahnt, et al., 2009) extended upon this evidence by demonstrating that detoxified alcohol dependent individuals showed lower ventral striatum activation during a monetary incentive delay (MID) task (which used visual cues to predict whether a rapid response to a target stimulus would lead to monetary gain, avoidance of monetary loss, or no consequences). Further, reduced activation of the ventral striatum and anterior cingulate during anticipation of monetary gain was also correlated with high impulsivity (assessed using the BIS-10); however, this finding was moderated by alcohol dependence status, such that low activation in these neural areas was only correlated with high impulsivity in alcoholics and not in control subjects. Because the ventral striatum is believed to signal the likelihood of reward (Schultz, Apicella, Scarnati, & Ljungberg, 1992), these findings suggest that disordered neural signaling of alcohol vs. non-alcohol reward availability could contribute to impulsive alcohol use behavior in clinical samples. To date, findings from fMRI and PET studies suggest that hard-to-resist urges to use drugs involve the ventral striatum, insula, OFC, and

lateral prefrontal cortex, all regions that comprise aspects of the extended reward system (Olbrich et al., 2006; Hermann et al., 2006; de Greck et al., 2009; Park, et al., 2007).

Some recent studies suggest that alcohol dependence affects the function of brain regions engaged for successful behavioral control. Li et al. (2009) reported the results of an fMRI study comparing alcohol dependent individuals and health control subjects using a Stop-Signal Task, designed to provide a trial-by-trial assessment of response inhibition, error processing, post-error behavioral adjustment, and risk taking by assessing speed and accuracy. Results of the study demonstrated that alcohol dependent individuals demonstrated less activation of the right dorsolateral prefrontal cortex during post-error behavioral adjustment compared to healthy controls, and further, alcohol dependent individuals who reported stronger alcohol urges at the time of the fMRI were particularly impaired in dorsolateral prefrontal activation compared to those that did not report any urges. In addition, alcohol dependent individuals showed less activation in cortical and subcortical structures including the putamen, insula, and amygdala compared to controls during the timed risk-taking trials. (Li et al., 2009). This research is also supported by other studies that have begun to delineate complex cortical-subcortical networks crucial for successful behavioral control (Stevens, Kiehl, Pearson, & Calhoun, 2007).

There also appear to be individual differences in neural activation related to externalizing symptoms, psychopathology, and affect. For example, Tarkka and colleagues (2001) reported that alerting tones were processed in the frontal lobe by violent alcohol abusers, but in medial temporal brain areas by non-violent alcohol abusers and normal subjects; violent alcohol abusers responded to deviant tones simultaneously in frontal and temporal areas, whereas these areas were affected sequentially in other groups (Tarkka et al., 2001). These findings imply alterations in arousal and involuntary adaptive processes in cortical networks associated with impulsive violent behavior and AUDs (Tarkka et al., 2001). The observation that impaired cognitive control is related to abnormal prefrontal cortex function is consistent with numerous neuroimaging and EEG studies of Attention-Deficit/Hyperactivity Disorder, Conduct Disorder, and ASPD (Seidman et al., 2004; 2005). The similarity of deficits between these two lines of research potentially points to abnormalities representing shared vulnerability to disruptive/antisocial behavior disorders and AUDs which has long been observed to co-occur clinically (Sher & Trull, 1994). Although this evidence may not provide specificity in the neural substrates implicated in AUDs as they relate to impulsivity, it does demonstrate commonalities in the dysfunction across the related disorders.

Electrophysiological data—There is also a large body of research on the role of the P3 amplitude in addictions and impulsive behaviors. For instance, Chen and colleagues (2007) assessed the relationship between impulsivity (measured by the BIS) and alcohol dependence, and their correlations with P3 amplitude. Healthy control subjects and subjects with DSM-IV diagnosis of alcohol dependence were assessed with a visual oddball task. Alcohol dependent subjects had lower target P3 amplitudes, which was also associated with lower activity in the cingulate, medial, and superior frontal regions. Alcohol dependent subjects had significantly higher scores on the BIS than nonalcohol using individuals, and BIS scores were negatively correlated with P3 amplitude. In sum, results demonstrated a strong frontal focus of reduced activation during processing of visual targets in alcohol dependent subjects and individuals with higher impulsivity (Chen et al, 2007).

Integration and Critical Review

These findings highlight the importance of developing our understanding of the bidirectional relationship of impulsivity and alcohol both at the behavioral and biological level including gene expression and neurobehavioral functioning. Further, these findings show that both

behavioral (i.e., delay discounting) and self-report measures of impulsivity can distinguish subtypes of individuals presenting with AUDs, and do so in a reasonably systematic manner. In addition to more straightforward relationships between impulsivity and AUDs, identifying these factors, such as psychopathology, violence/aggression, and biological mechanisms can serve to further elucidate the relationship between impulsivity and alcohol use. The limited research on psychopathology suggests that certain dimensions of impulsivity may manifest differently or to a greater extent for some individuals than others given issues of comorbidity. However, this literature is largely limited to focusing on Cluster B Personality Disorders, and thus more work is needed in examining how other types of PDs and Axis I disorders may also influence the relationship between impulsivity and AUDs. The reviewed studies also illustrated how co-occurring factors, such as violence and aggression, may shed light on aspects of the relationship between impulsivity and AUDs. Specific aspects of impulsivity, including attention and motor control, have been shown to most closely explain the high rates of physical aggression found in alcohol dependent men (Parrott & Giancola, 2006). Further work is needed in this area to build on the heterogeneous designs and methodologies of studies examining the role of violence and aggression, as well as to more clearly elucidate gender differences.

Despite recent progress, continued progress will require targeted and systematic replication of these reported associations, which may be especially difficult in the genetic and neurobehavioral domains where sufficiently powered studies are difficult to conduct. Additionally, since most studies have relied on cross-sectional designs, future longitudinal studies are crucial to clearly differentiate the impulsivity-related factors that underlie AUDs versus those factors that are related but not causal, including factors that are the result of AUDs. In this way, there is great potential for advances in this area through the continuing assessment of large cohorts of “at-risk” samples, such as collected in the Collaborative Studies on Genetics of Alcoholism (COGA) and similar longitudinal studies in the United States, Europe and New Zealand that have assessed alcohol use patterns throughout child to early adult development. These platforms provide a powerful basis upon which to graft targeted neurobiological assessment (especially genome-wide association studies) onto rigorously-characterized samples whose alcohol use and disorder status has been carefully tracked over time.

Future Directions of Research

Taken together, there is an emerging body of research that provides a solid foundation for the association between impulsivity and alcohol abuse, but moving forward it is important to target key areas that provide greater structure and depth, including more research directed at better understanding the multidimensional nature of impulsivity, expanding the scope of impulsivity measurement to aversive stimuli, ascertaining causal, mechanistic relationships between impulsivity and alcohol use phenomena, and impulsivity as a target for alcohol interventions.

Expanding the Measurement of Impulsivity to Focus on Aversive Stimuli

From the review presented above, the role of the delay discounting dimension of impulsivity is clear. It is notable that in these studies delay discounting is studied largely in relation to the delay of positive reinforcement/reward. However, delay discounting also may be used to understand the discounting of aversive events, as a larger later aversive event may be preferred over a smaller current aversive event. This focus on aversive events fits well with alcohol, as its use can be viewed under some circumstances as a strategy to avoid immediate or short-term aversive events or aversive affective experiences (i.e., in line with negative reinforcement theory; Baker et al., 2004), despite the fact that the longer-term aversive events associated with alcohol use are often greater in magnitude. Although this form of

delay discounting may be most appropriately applied to alcohol withdrawal, it can also be understood in the context of current use, such as when someone turns to alcohol when faced with environmental stressors, or even initiation of use, for example when youth combat influences such as peer pressure to drink; drinking alleviates peer pressure in the moment, even though worse consequences may be around the corner. This type of a paradigm would add an important alternative methodological approach that could be done hypothetically or with real events in laboratory or naturalistic settings. This paradigm would provide an important negative reinforcement conceptual complement to the current positive reinforcement approach that dominates the delay discounting and impulsivity literature. Critically important to this expanded focus is research that juxtaposes the study of disordered sensitivity to reinforcement with abnormal cognitive control. As reviewed above, these are two major conceptualizations within the study of impulsive behavior. The relationship of these variously-defined constructs not only to alcoholism, but also to each other, will provide key insights for understanding how impulsive behavior promotes alcohol use disorders.

Arguably the best example of this approach in the current literature involves what has been called a cost-discounting paradigm with three studies available beginning to test its psychometric properties in relation to discounting of gain (Holt, Green, Myerson, & Estle, 2008; Murphy, Vuchinich, & Simpson, 2001; Mitchell & Wilson, 2009) and specific to cigarette smoking (Baker, Johnson, & Bickel, 2003). These provide a useful starting point but it is important for this work now to be expanded to focus directly on alcohol and to consider using more ecologically valid stimuli that simulate more closely the types of aversive events in the real world using both hypothetical self-report and choice with real consequences.

Ascertaining Causal Relationships

Despite the breadth of research examining the relationship between impulsivity and alcohol use initiation, maintenance, and development of AUDs, the issue of causality in this relationship remains ambiguous. For example, studies in the majority of areas reviewed do not provide sufficient evidence to ascertain whether impulsivity predates AUDs as a risk factor, whether impulsivity co-occurs with AUDs perhaps a consequence or associated feature of persistent drinking, or rather co-exists as a result of a third common factor. Alternatively, impulsivity may be both a causative factor and a consequence of AUDs. Interactive effects often exist with both the genetic predisposition to impulsivity as well as effects of AUDs. For instance, an individual may have a predisposition to impulsivity, while certain consequences of excessive alcohol use (e.g., brain function abnormalities) may further exacerbate this predisposition. As noted above, biological research has made some progress teasing apart causality. Although still quite preliminary, there is more progress in this domain compared to studies using behavioral and self-report measures of impulsivity, where very limited progress has been made. Finally, to truly understand causality among these numerous components of impulsivity and the development of AUDs, we must first come to a consensus on our definitions of impulsivity, thus facilitating a smoother connection to be made across studies and a platform to build upon existing findings to meaningfully incorporate biological evidence.

Impulsivity as a Target of Alcohol Interventions?

Although personality constructs such as impulsivity often are viewed as stable, unvarying traits, recent evidence has suggested that impulsivity may change over time, particularly throughout the course of adolescence, and this *change* in impulsivity may also affect alcohol use. For example, a study by Littlefield, Sher, & Wood (2009) examined the relationship between changes in problematic alcohol involvement and personality (measures of

impulsivity, neuroticism, and extraversion) from ages 18 to 35 in a cohort of college students at varying risk for alcohol use disorders. They captured the non-planning dimensions of impulsivity using 10 items derived from the EIQ and the TPQ. Latent growth models indicated that both normative and individual changes in alcohol involvement occurred between ages 18 and 35, and that these changes were associated with changes in neuroticism and impulsivity. Moreover, environmental factors, such as marital status and parental role status, did not explain the associated changes in alcohol involvement and personality. Findings suggest that personality change may be an important mechanism in understanding the onset and progression of alcohol use (Littlefield et al., 2009).

In considering this notion of the malleability of personality, there is great potential utility in focusing on treatment efforts that target personality vulnerabilities. In a study conducted by Conrod, Castellanos, & Mackie (2008), the authors investigated how early intervention targeting at-risk personality factors (negative thinking, anxiety sensitivity, impulsivity, and sensation seeking) could defer premature alcohol abuse and binge drinking in at-risk youth. Study participants (median age = 14) were randomly assigned to one of two treatment conditions: personality-targeted interventions that integrated a psycho-educational, motivational interview, and cognitive-behavioral approach; or no intervention. Treatment consisted of two supervised 90-minute sessions, and follow-ups were conducted at 6 and 12 months post-intervention. Conrod and colleagues found that personality-targeted interventions provided a 6-month delay in alcohol misuse and could extend up to 12 months in all aspects of drinking behavior (i.e., increases in quantity and frequency of drinking, binge drinking) compared to controls. Although targeted intervention was shown to be effective in all groups, individuals with high-risk drinking behaviors and at-risk personality characteristics (e.g., sensation seeking drinkers) showed the most robust improvements post-6 and 12 month intervention. Additional support for this personality-matched approach has been provided in previous studies in older youth with problem patterns of drinking (Conrod, Stewart, Comeau, & Maclean, 2006) and adult female substance users (Conrod et al., 2000), again with significant effects for the impulsivity and sensation seeking interventions, with stronger effects for the latter. Currently this approach does not match based on the particular dimensions of impulsivity discussed in this review, but this may be a useful direction for future efforts.

Beyond applications of behavioral data, it will be a distinct challenge to devise ways to make use of neuroimaging data in any concrete “clinical” application, so for now its value lies primarily in better understanding neural aspects of AUDs. One possibly fruitful future direction will be to integrate fMRI or similar functional neuroimaging assessments into trials of emerging experimental pharmacological approaches designed to reduce urges to drink. Such research might discover how exactly these medications influence neural substrates associated with impulsive alcohol use in ways that could dramatically improve our understanding of links between impulsivity and AUDs. Additional neurobehavioral research targeting the link between impulsivity and alcohol use is needed, particularly studies that build on findings specific to unique dimensions of impulsivity as they relate to distinct stages in the development of AUDs

Conclusion

In summary, numerous studies have established a role for impulsivity in the initiation of alcohol use, continued use and escalation associated with developing AUDs. However, the measures used have been highly heterogeneous, which makes it difficult to identify the neuropsychological mechanisms underlying these relationships. In addition, studies establishing the relationship between impulsivity and alcohol use were not designed to assess whether these relationships were founded on causal mechanisms. Thus, two

recommendations are made for future research in this area. First, it is important to link psychological definitions of impulsivity with variations in neurological processes using a broad array of complementary neuroscience techniques. Second, it is important to examine causal relationships utilizing study designs including longitudinal, interventional and animal models.

Additionally, there exists a lack of systematic progression and connection across studies among the different aspects of alcohol use and the varied definitions and methodologies for assessing impulsivity. This being said, the existing literature does represent an emerging body of work that provides a solid framework to begin to build more depth and draw connections, as much about the ways in which impulsivity and alcohol are related as well as the ways in which null results are evident. In this way, what becomes most relevant are studies that isolate the various dimensions of impulsivity with differential predictions of how the distinct dimensions relate to alcohol use, as well as further delineating the components of “alcohol use” as a main outcome. Moreover, the extent to which this work can be used to guide intervention is a clinical necessity.

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