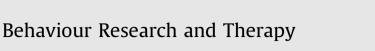
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A laboratory model of impulsivity and alcohol use in late adolescence



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ABSTRACT

Impulsivity is a core characteristic of externalizing problems and a robust predictor of alcohol use in adolescence. There is little evidence on the causal mechanisms through which impulsivity influences drinking or how they are affected by key social factors (peer influence). This study reports the development of the first comprehensive laboratory model of adolescent impulsivity and alcohol use. One-hundred and twenty adolescents (50% female) of legal drinking age (M = 19.47 years, SD = 1.12) in Australia (18+ years) were subjected to 1 of 3 experimental manipulations to increase impulsive behavior (reward cue exposure, negative mood induction, ego depletion). Changes in disinhibition (stop-signal task) and reward-seeking (BAS-Fun Seeking) were measured before completing a laboratory drinking task alone or with a heavy-drinking confederate. Reward cue exposure increased alcohol consumption, with the effect mediated by increased reward-seeking. Negative mood induction increased disinhibition, but not drinking. The presence of a heavy-drinking peer directly increased alcohol consumption in an additive fashion. Findings provide causal evidence that extends survey-based research by highlighting the role of reward-related impulsivity in adolescent alcohol use. The new laboratory model can provide novel insights into the psychological processes underlying adolescent impulsivity and impulsivity-related drinking.

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1. Introduction

Alcohol use during adolescence can have a profound, longlasting impact on future health. Alcohol use disorders are most prevalent in the late adolescence-to-young adulthood developmental period (18–29 years) and, in developed countries, alcohol is responsible for 1 in 4 adolescent deaths (Connor, Haber, & Hall, 2016; Toumbourou et al., 2007). Impulsivity is a robust predictor of problematic alcohol use and may be particularly important to teenage drinking, given the elevations observed in this trait during adolescence (Chartier, Hesselbrock, & Hesselbrock, 2010; Gullo & Dawe, 2008; Moffitt et al., 2011). Despite this, little is known about how impulsivity influences alcohol use. Critically, there is a dearth of evidence on causal effects and how such effects might be moderated by social factors and the nature of the impulse that leads to drinking (Franken & van de Wetering, 2015; Gullo & Potenza, 2014; Lejuez et al., 2010). This paper reports the development of a comprehensive laboratory model of adolescent impulsivity and alcohol use that incorporates key psychological and social influences.

Impulsivity and other approach-related personality traits have been consistently associated with problematic alcohol use (Gullo, Loxton, & Dawe, 2014; Iacono, Malone, & McGue, 2008; Stautz & Cooper, 2013; Verdejo-García, Lawrence, & Clark, 2008). Moffitt and colleagues' (2011) large, birth cohort study found the power of childhood self-control ratings to predict future adult health behaviors (e.g., substance dependence, overweight, sexuallytransmitted infection) approximated that of intelligence and social class. Similar findings have been reported with behavioral measures. Fernie et al. (2013) reported that a range of behavioral impulsivity measures, including the stop-signal task, consistently predicted adolescent alcohol use at 6-month follow-ups over a two-year period. In a multinational sample of 1593 14-year-olds, Whelan et al. (2012) observed reduced orbitofrontal cortex (OFC) activity during stop-signal task inhibition was associated with early adolescent drug use, suggesting OFC hypofunctioning may underlie inhibitory control deficits linked to early substance use (i.e., disinhibition). In a subset of the same sample, Nees et al. (2012) found self-reported impulsivity traits were more predictive of early onset drinking in 324 adolescents than behavioral measures of risktaking and reward-related brain activity. Impulsivity, whether measured by self-report ratings, behavioral performance, or its underlying neural activity, is clearly associated with adolescent alcohol use.

While impulsivity is generally regarded as a multidimensional trait, debate continues as to the number and nature of underlying dimensions or subtraits. Prominent models specify the existence of 2-5 dimensions (Berg, Latzman, Bliwise, & Lilienfeld, 2015; Dawe & Loxton, 2004; Depue & Collins, 1999; Potenza & Taylor, 2009; Sharma, Kohl, Morgan, & Clark, 2013; Steinberg, 2008; Whiteside & Lynam, 2001). When considering impulsivity in the context of substance use, however, there is a consensus emerging that two dimensions are uniquely involved (Gullo et al., 2014; Hamilton, Littlefield, et al., 2015; Hamilton, Mitchell, et al., 2015; King, Patock-Peckham, Dager, Thimm, & Gates, 2014; Sharma et al., 2013; Stautz, Dinc, & Cooper, 2017). These dimensions are characterized by reward sensitivity and disinhibition. The proceeding discussion will focus on one of these two-factor models of impulsivity and substance use that has been applied specifically to adolescence: the 2-Component Approach to Reinforcing Substances (2-CARS; Gullo & Dawe, 2008).

Impulsivity can arise from high reward sensitivity, leading to strong approach motivation and subsequent reward-seeking (Dawe, Gullo, & Loxton, 2004; Ernst, Pine, & Hardin, 2006; Hamilton, Mitchell, et al., 2015; Potenza & Taylor, 2009). This conveys risk for alcohol misuse through heightened sensitivity to positive reinforcement and incentive salience (Dawe et al., 2004). Reward sensitivity is a key mechanism of impulsive behavior insomuch that the strength of the approach impulse requiring inhibition is negatively associated with the likelihood of successful inhibition (Dawe & Loxton, 2004; Gray, 1975; Padmala & Pessoa, 2010). Basing their conceptualization of reward sensitivity on Gray's Behavioral Approach System (BAS; Gray, 1975), Gullo and Dawe (2008) used the analogy of two automobiles braking at different speeds (i.e., 2-CARS). The vehicle traveling at higher speed (i.e., stronger approach impulse) will take longer to stop despite both having equally effective brakes (i.e., inhibitory control). Padmala and Pessoa (2010) provide empirical evidence consistent with this hypothesis. They experimentally increased impulsive responding on the stop-signal task simply by rewarding correct "go" approach responses in healthy adults. Not only did participants demonstrate greater disinhibition (driven by reward), but also a pattern of reduced activity in the inferior frontal gyrus and other regions typically observed in addicted populations.

The other key dimension of impulsivity involved in substance use is disinhibition, or "rash" impulsiveness (Dawe & Loxton, 2004; Dawe et al., 2004). Disinhibition reflects a reduced capacity for inhibition of prepotent approach responses due, in part, to less consideration of negative future consequences (Dawe et al., 2004; Ernst et al., 2006; Hamilton, Littlefield, et al., 2015; Potenza & Taylor, 2009). The model (and measures) of impulsivity proposed by Barratt (1993), Eysenck (1993), Zuckerman (Zuckerman & Kuhlman, 2000), and Cloninger (1987) align more closely with this dimension (Dawe & Loxton, 2004). Returning to the automobile analogy, this dimension relates to the strength and efficiency of vehicle brakes, irrespective of travel speed (Gullo & Dawe, 2008). Disinhibition conveys risk for alcohol misuse through a reduced capacity to inhibit drinking (especially continued drinking) in light of future negative consequences (Dawe et al., 2004; Ernst et al., 2006; Hamilton, Littlefield, et al., 2015; Potenza & Taylor, 2009).

The preceding discussion should not be taken to suggest that other dimensions of impulsivity do not exist. Rather, it is argued that they probably do not convey risk for adolescent alcohol use independent of their relationship with reward sensitivity and disinhibition (Stautz et al., 2017). For example, some have proposed a unique dimension of trait impulsivity related to negative affect (e.g., urgency; Whiteside & Lynam, 2001). Indeed, negative affect has been shown to increase impulsive behavior and patients with major depressive disorder evidence deficits in inhibitory control (Snyder, 2013; Tice, Bratslavsky, & Baumeister, 2001). However, negative affect can also reduce reward sensitivity (Gullo & Stieger, 2011) and the evidence relating trait urgency to youth substance use is mixed. Most studies do not find urgency prospectively predicts substance use when controlling for other impulsivity traits (for a review, see Gullo et al., 2014; Lopez-Vergara, Spillane, Merrill, & Jackson, 2016). Youth alcohol use is predominantly motivated by social rewards with tension-reduction motives gaining prominence in older adulthood (Kuntsche, Knibbe, Gmel, & Engels, 2005; Nicolai, Moshagen, & Demmel, 2012). In summary, while 2-CARS and other two-factor models of impulsivity do not place a strong emphasis on the unique role of negative affect in reward-seeking or disinhibition in youth substance use, other models do. If negative affectivity/urgency-related traits do play a unique role in adolescent drinking, it is likely to be complex, and controlled laboratory studies observing actual behavior are well-placed to elucidate this. This is an empirical question.

Impulsivity is commonly operationalized at the trait level as scores on a self- or observer-rated questionnaire reflecting general behavioral tendencies over time. Despite the stability of these generalizations (i.e., mean impulsivity), there is high within-person variability in prototypical impulsive behaviors across contexts (Fleeson, 2001). In a series of studies employing ecological momentary assessment, Fleeson (2001) reported greater withinperson than between-person variability in behaviors derived from approach- and impulsivity-related traits. This within-person variability was related to the presence of trait-relevant cues in the environment, such that greater increases in extraverted behavior while in the presence of others predicted one's overall variability in extraversion. These findings are consistent with biologically-based models of impulsivity, which conceptualize the trait as individual differences in baseline thresholds of activation to specific classes of stimuli (e.g., rewards and punishments; Cloninger, 1987; Depue & Collins, 1999; Gray, 1975). Both theoretical frameworks allow for the experimental induction of impulsive behaviors by external stimuli ("state impulsivity"), irrespective of an individual's average frequency of impulsive behaviors ("trait impulsivity"). Thus, in theory, reward-seeking could be experimentally induced by exposure to reward cues. Disinhibition could be induced by an affective state previously shown to reduce inhibitory control (e.g., negative mood), or by a less affectivelycharged manipulation designed to directly undermine selfcontrol through exertion or fatigue (e.g., "ego depletion"; Baumeister, Bratslavsky, Muraven, & Tice, 1998; Inzlicht, Schmeichel, & Macrae, 2014). The ability to manipulate impulsivity, at least in the short-term, provides rich opportunities for experimental research and the delineation of key causal effects.

A comprehensive laboratory model of adolescent drinking should take into account the unique effect of peers during this developmental period. Peer alcohol use is a key predictor of youth Download English Version:

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