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 increased alcohol consumption independent of experimental manipulations. 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.

Keywords: impulsivity, alcohol, adolescence, reward, disinhibition, mood

1. Introduction

Alcohol use during adolescence can have a profound, long-lasting 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, sexually-transmitted infection) approximated that of intelligence and social class. Similar findings have been reported with behavioral measures. Fernie and colleagues (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 1,593 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 risk-taking 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 inasmuch 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 et al., 2004; Dawe & Loxton, 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 within-person 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 affectively-charged manipulation designed to directly undermine self-control 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 drinking and there is robust experimental evidence that the presence of heavy-drinking peers increases consumption (Chartier et al., 2010; Quigley & Collins, 1999). The presence of a peer does increase both reward-related neural activity in adolescents and

risk-taking on behavioral tasks in experimental studies (Albert, Chein, & Steinberg, 2013; Chein, Albert, O'Brien, Uckert, & Steinberg, 2010). What is not clear is if such social forces influence the causal effect of impulsivity on drinking. In the laboratory, a heavy-drinking peer may facilitate the translation of an impulsive state into heavier drinking in two ways. First, it may increase the perceived social rewards of drinking by engaging in behavior consistent with that of a peer. In other words, a reward seeking-related process. Second, it may lessen concerns about it being inappropriate to imbibe a large quantity of alcohol in a research context, i.e., a disinhibition-related process.

To further advance the field, a new laboratory model was developed to comprehensively investigate the causal effect of impulsivity on adolescent alcohol use. The *Experimental Paradigm to Investigate Impulsive Consumption (EPIIC)* was designed to allow for the controlled study of adolescent drinking caused by impulsivity arising from three theoretically-derived psychological processes: reward-seeking (reward cue exposure), disinhibition (non-affective ego depletion), and negative affect (mood induction). Participants are allocated to one of these three arms and receive the corresponding experimental manipulation in one testing session (e.g., reward cue exposure) and a control in the other session (e.g., neutral cue exposure; counter-balanced). Within-subject change in laboratory alcohol consumption across these two sessions quantifies the causal impact on alcohol use. Within-subject change on measures of reward-seeking and disinhibition will isolate mechanisms affected by experimental manipulation. Primary outcomes of interest were administered as repeated measures in order to reduce the potential impact of individual differences on induction effects. Laboratory alcohol consumption is influenced by body weight, sex, alcohol metabolism, drinking history, and many other factors that can be controlled for in a within-subjects design (Bujarski & Ray, 2016). The moderating effect of a

heavy-drinking peer on alcohol consumption was also incorporated, such that half of all participants will engage in laboratory consumption with a confederate across both sessions (or not; between-subjects). This was intended to identify important contextual effects that may facilitate or attenuate the translation of an impulsive state into drinking. Self-report measures of trait impulsivity (and potential covariates) were administered to allow confirmation of whether random allocation produced balanced groups.

The study had two overarching aims. First, to test whether distinct impulsive states (reward seeking or disinhibition) can be induced experimentally by three distinct procedures (reward cue exposure, negative mood induction, ego depletion). Second, to determine whether these impulsive states causally increase adolescent alcohol use. Figure 1 depicts *EPIIC* and the hypothesized effects for adolescent drinkers.

INSERT FIGURE 1 HERE

Based on the 2-CARS model (Dawe, Gullo, & Loxton, 2004; Dawe & Loxton, 2004; Gullo & Dawe, 2008), it was hypothesized that only reward cue exposure and ego depletion would increase adolescent alcohol consumption. It was also hypothesized that the effect of reward cue exposure would be mediated by increased reward-seeking, and the effect of ego depletion mediated by increased disinhibition. Negative mood induction was predicted to have no ultimate effect on adolescent alcohol use because it will both increase disinhibition and decrease reward-seeking, producing an overall null effect on consumption. Experimental effects were predicted to be moderated by a heavy-drinking peer, such that their presence would strengthen experimental effects on drinking.

2. Method

2.1 Participants

One hundred and twenty participants (50% female) were recruited through advertisements placed at local university and community college campuses in Brisbane, Australia. The mean age of the sample ($M = 19.47$ years, $SD = 1.12$) is consistent with *late adolescence* as defined in recent studies and by the World Health Organization (Casey, 2015; Ernst, 2014; Stautz & Cooper, 2013; World Health Organization, 2015). Minimum age of participants was 18 years due to jurisdictional drinking age restrictions. There is no consensus on when the adolescent period ends (Casey, 2015; Ernst, 2014; Roenneberg et al., 2004; Stautz & Cooper, 2013). While most commentators suggest the early 20s, others argue adolescence should not be defined in terms of chronological age (Casey, 2015; Petersen, Silbereisen, & Sorensen, 1996). For the purposes of this study we chose a maximum age of 21 years.

The majority of the sample were in college (95; 79.2%) and were White/Caucasian (86.6%), with 12 (10.1%) Asian, and 4 (3.4%) 'Other' participants. Additional inclusion criteria aside from age 18-21 years were: recent consumption of alcohol (i.e., within the last 2 weeks), never been diagnosed with an alcohol use disorder, a zero Blood Alcohol Level (BAL = 0.00%) at the time of testing, not currently suffering from a medical condition or taking medication where alcohol consumption is contraindicated, not pregnant, and having normal or corrected-to-normal color vision. Participants were reimbursed with a AUD\$40 gift voucher for vendors that do not sell alcohol. All participants provided written, informed consent. The study was approved by the relevant university human research ethics committee.

2.2 Measures

2.2.1 Self-reported alcohol use. The timeline followback (TLFB) interview schedule was administered to measure *quantity (in grams of ethanol)* and *frequency (days)* of alcohol use over the past 28 days. It has strong psychometric properties and is one of the most widely used measures of alcohol use (Sobell & Sobell, 1992). The Alcohol, Smoking and Substance Involvement Screening Test (ASSIST, version 3.0) is an interview schedule to screen for the presence and severity of substance use, including *hazardous alcohol use*. The alcohol-specific substance involvement composite has been shown to discriminate between non-problematic alcohol use, abuse, and dependence, and has good internal consistency and test–retest reliability (Humeniuk et al., 2008).

2.2.2 Self-reported trait impulsivity. The 24-item Sensitivity to Reward (SR) scale (Torrubia, Ávila, Moltó, & Caseras, 2001) was used as a measure of *trait reward sensitivity*. Cronbach’s alpha for the SR scale in this study was .67. *Trait rash impulsiveness* was measured with the 19-item impulsiveness scale of the I₇ Questionnaire (S. B. G. Eysenck, Pearson, Easting, & Allsopp, 1985). This is one of the most commonly used impulsivity measures and has been shown to load on a factor distinct from reward sensitivity with other measures of the construct (Dawe & Loxton, 2004). Cronbach’s alpha for the scale in this study was .83.

2.2.3 Alcohol consumption. Laboratory alcohol consumption was measured using a Cocktail Taste Rating Task (C-TRT) based on Chipperfield and Vogel-Sprott (1988). Various alcohol taste rating tasks have been used in a large number of laboratory studies since their introduction by Marlatt, Demming, and Reid (1973). Consumption on these tasks has previously been shown to correlate with self-reported consumption, to differentiate individuals with and without a diagnosis of alcohol dependence, and to be sensitive to peer

modeling and expectancy effects (Chipperfield & Vogel-Sprott, 1988; Conrod, Stewart, & Pihl, 1997; Marlatt et al., 1973; Quigley & Collins, 1999). As in Chipperfield and Vogel-Sprott (1988), the C-TRT used in this study involved participants rating 3 alcoholic cocktails (marked A-B-C) on various taste indices (sweetness, bitterness, etc.) under the guise of exploring taste perceptions. Each of the 700ml cocktails contained vodka and soda mixtures (6.6% alcohol by volume, %v/v) designed to look like premixed spirits (“alcopops”), which are popular among Australian adolescents (White & Bariola, 2012). Mixers for the cocktails were lemon squash (A), blood orange (B), and passionfruit flavored soda (C). Cocktails were presented in 750ml transparent acrylic cocktail shakers (Bartender Acrylic Cocktail Shaker, Donaldson Enterprises Pty Ltd). Participants were provided a 185 ml transparent sampling cup, Card Index booklet of 100 taste adjectives, printed one per card, and a response form on which they were to rate how accurately each adjective described the cocktail on a 1-10 scale. Participants were informed that they could sample as much of each drink as they needed in order to rate the cocktails. After being given instructions for the task, the researcher exited the room for an unspecified time and returned after 15 minutes to end the task. At the end of the task in session one, participants were told that they would be able to complete the task in the next session. No participant consumed all the available alcohol or completed the ratings for all cocktails in the allotted time, reflecting no ceiling effects. *Total consumption (ml) was the outcome variable.*

In this study, consumption on the C-TRT predicted drinking quantity ($r = .44, p < .001$) and drinking frequency ($r = .19, p = .04$) over the next 7 days, and correlated with past-month drinking quantity ($r = .26, p = .003$), drinking frequency ($r = .15, p = .06$), and problematic drinking on the ASSIST ($r = .19, p = .01$). There was good temporal stability across testing sessions (1 week) despite the administration of an experimental manipulation

designed to affect consumption ($r = .47, p < .001$). For reference, Cohen's (1988) effect size conventions for r are: small = .10, medium = .30, large = .50. Together, this provides preliminary validation of the C-TRT (see also Chipperfield & Vogel-Sprott, 1988; Scheveneels, Boddez, Vervliet, & Hermans, 2016).

2.2.4 Disinhibition. A computerized stop-signal task, *STOP-IT* (Verbruggen, Logan, & Stevens, 2008), was administered to calculate *Stop-Signal Reaction Time (SSRT)*, a measure of 'state' disinhibition (Hamilton, Littlefield, et al., 2015). Stop-signal paradigms are widely used in both clinical and preclinical research, and the SSRT has strong reliability and validity (Hamilton, Littlefield, et al., 2015; Kindlon, Mezzacappa, & Earls, 1995). The stop-signal task is also less sensitive to practice and order effects than other disinhibition measures because there is no learning component and the stop-signal delay adjusts in response to participant performance (Hamilton, Littlefield, et al., 2015; Kindlon et al., 1995). STOP-IT involves a choice reaction time task in which participants discriminate between a square and circle presented on screen. Trials start with the presentation of a fixation sign for 250 ms, which is replaced by a square or circle. The stimulus remains on the screen until participants respond or 1,250 ms elapse. On a random 25% of trials, an auditory stop signal is presented that requires participants to inhibit their approach response. The delay between the visually presented shape and the auditory stop signal is varied according to a staircase tracking procedure, whereby unsuccessful inhibition results in a 50 ms increase in stop-signal delay and a successful inhibition in a 50 ms decrease. The procedure leads to a stop-signal delay with a .50 probability of response disinhibition, the SSRT. STOP-IT comprises an initial practice phase of 32 trials followed by 3 blocks of 64 trials. Interstimulus interval is 2,000 ms that is independent of RT.

2.2.5 Reward Seeking. The 4-item BAS-Fun Seeking scale from the BAS/BAS Scales (Carver & White, 1994) was modified for the purposes of this study to measure ‘*state*’ *reward seeking*. The BAS scales have been previously shown to be sensitive to experimental manipulations (Schmeichel, Harmon-Jones, & Harmon-Jones, 2010). This scale was chosen because of its short number of items that could be modified for a “right now” focus. The scaling of the items was also modified to a 9-point Likert scale, resembling the “right now” version of the Approach and Avoidance of Alcohol Questionnaire (i.e., I agree with this statement... 0 = *Not at all* to 8 = *Very Strongly*). Cronbach’s alpha in this study was .84.

2.2.6 Covariates. Experimental manipulations were intended to increase impulsivity. However, the manipulations might produce other effects that were not of interest to the study. Changes in craving, positive affect, and negative affect were measured so that they could be statistically controlled, if necessary. *Craving* was measured using the ‘Right Now’ version of the 5-item Inclined/Indulgent scale of the Approach and Avoidance of Alcohol Questionnaire (AAAQ; McEvoy, Stritzke, French, Lang, & Ketterman, 2004). The full AAAQ comprises two approach subscales (Inclined/Indulgent and Obsessed/Compelled) and one avoidance subscale (Resolved/Regulated) and has been validated on college drinkers and alcohol-dependent outpatients (Klein, Stasiewicz, Koutsky, Bradizza, & Coffey, 2007; McEvoy et al., 2004). Only the approach motivation scales predict cue reactivity in drinkers with an alcohol dependence diagnosis and, in those without such a diagnosis, only the Inclined/Indulgent scale predicts unique variance in self-reported consumption (Klein et al., 2007). Only the Inclined/Indulgent scale was used in the present study (Cronbach’s alpha in this study was .83). *Affect* was measured using the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a widely-used measure assessing the degree to which participants are currently experiencing 10 positive and 10

negative emotions on a 5-point scale, which can be summed to provide an overall index of positive affect and negative affect, respectively. Cronbach's alpha in this study were .84 (positive) and .68 (negative).

2.3 Procedure

2.3.1 Randomization and Design. The study involved an Impulsivity Induction (2) x Induction Type (3) x Peer Influence (2) experimental design. Impulsivity Induction was a within-subjects (repeated measures) variable with two levels: experimental and control. The order of experimental and control sessions for each participant was determined in advance using randomized counterbalancing, conducted by an independent person (detailed below). Induction Type was a between-subjects variable with three levels: reward cue exposure ($n = 40$), negative mood induction ($n = 40$), ego depletion ($n = 40$). Peer Influence was a between-subjects variable with two levels: heavy-drinking peer present ($n = 60$) or absent ($n = 60$). Random allocation to between-subjects variables was stratified by gender to ensure an equal number of male and female participants in each condition (see Supplemental Material available online for details on randomisation and blinding). This was solely for the purpose of enhancing generalizability of results and is not a variable of interest. A recent meta-analysis by Cross et al. (2011) found no sex differences on SSRT or BAS-Fun Seeking. Baseline differences in alcohol consumption are controlled for by the within-subjects experimental design and use of random intercepts in analyses (detailed below). There was no *a priori* reason to expect sex differences in the effect of increased state impulsivity on drinking or behavioral performance (i.e., interactions). An increase in state impulsivity was expected to produce the same increase in alcohol consumption for men and women, even though baseline levels may differ.

Participants were tested individually across two experimental sessions (see Figure 2). Participants were asked to fast for 4 hrs prior to each session, and ingest no non-prescription drugs or alcohol 24 hrs before each session (Chipperfield & Vogel-Sprott, 1988; Miller & Fillmore, 2014). Breath samples were analyzed at the beginning of each session with a Lion Alcolmeter 500 to verify zero blood alcohol level (BAL). The TLFB and ASSIST interview schedules were then administered, followed by trait impulsivity measures (SR and I₇), and baseline measures of reward-seeking, craving, and affect.

INSERT FIGURE 2 HERE

2.3.2 Manipulations. In the experimental session, participants randomly allocated to the *Reward cue exposure* manipulation were shown a 10-min film clip depicting characters in a ‘fun’ social context relevant to the population being studied (a college party). While alcohol consumption is present in the clip, what is more salient is the depiction of young people having fun, acting on the spur of the moment, and being disinhibited. These stimuli were chosen because the perceived social rewards of alcohol use are more potent motivators of youth drinking than the physical response to alcohol (Engels, Hermans, van Baaren, Hollenstein, & Bot, 2009; Kuntsche et al., 2005). In the control session, participants were shown a neutral clip involving the same characters as the experimental clip for that induction type. For male participants, clips were extracted from the film *American Pie 2* (Engels et al., 2009; Moore, Zide, & Rogers, 2001). For female participants, the experimental clip comprised scenes from the *Gossip Girl* episode “Dare Devil” (Rosenfeld & Babbit, 2007). The control clip was taken from the episode “The Blair Bitch Project” (Steinberg & Tobin, 2008).

Participants allocated to the *Negative mood induction* experimental manipulation watched a 6-min film clip rated by a large sample of youth ($M = 19.6$ yrs) as one of the saddest of a pool of 824 clips (Schaefer, Nils, Sanchez, & Philippot, 2010). The clip was taken from *City of Angels* (Roven, Steel, & Silberling, 1998). This was chosen based on evidence suggesting sadness and depression are more robust predictors of youth drinking than anxiety (O'Neil, Conner, & Kendall, 2011). In the control condition, an affectively neutral clip from the same film was shown. All film clips were shown under the guise of exploring perception/attitudes that participants would be later asked to rate.

Participants allocated to the *Ego depletion* group were administered the *Crossing-Out Letters Task* (Baumeister et al., 1998), a complex mental task used in previous studies to 'deplete' self-control. The task involves two parts. The first part requires participants to cross out as many instances of the letter "e" on an A4 page of text within 5 mins. The text is taken from a statistics textbook. In the experimental session, part two involves providing participants 5 mins and another page of text to perform the same task, but this time obeying complex rules. Specifically, the researcher read a script to the participant advising them to cross out all instances of the letter 'e' except if there was another vowel adjacent to the 'e' or one letter removed from it. Because of the dominant approach response set created in part one, part two requires response inhibition and leads to a subsequent reduction in impulse control after completion (Hagger, Wood, Stiff, & Chatzisarantis, 2010). The precise mechanisms involved in this process are debated, although accumulating evidence suggests it may be due to fatigue and change in task motivation (Inzlicht et al., 2014). In the control session, part two is performed without the complex rules, as in previous studies (Baumeister et al., 1998). Participants in all groups completed measures of reward-seeking, affect, and craving after the experimental/control manipulation and before the stop-signal task

(measuring disinhibition). This was followed by administration of the C-TRT to assess impact on alcohol consumption.

Participants allocated to the *Heavy Drinking Peer* condition completed the C-TRT whilst sitting opposite a gender-matched ‘heavy-drinking’ confederate, believed to be another participant. As in previous studies (Chipperfield & Vogel-Sprott, 1988), the confederate was trained to drink at a standardized pace to 700ml (45 sips, at a rate of 3 sips per minute, to drink a total of 700ml after 15 mins). Prior to providing the cocktails, the experimenter left the room to collect a participant who was ‘being tested in another room’ and they were seated opposite the participant approximately 2 m away. The experimenter served the cocktail decanters, tasting cups, and read standardized instructions stressing that participants were free to sample as much of each beverage as they needed to rate the cocktails on each taste dimension, and asked them not to interact or discuss their ratings with each other (Chipperfield & Vogel-Sprott, 1988). The experimenter then left the room saying that he/she would return shortly. As in Chipperfield and Vogel-Sprott, participants were not informed how long the C-TRT would last and there were substantially more adjectives in the card index than could be completed during the task. Confederates did not initiate conversation with participants but, if spoken to, replied in a friendly manner without continuing the discussion. Participants allocated to the *Peer Absent* condition completed the C-TRT alone.

After completing the C-TRT, participants were given filler questionnaires for 15 mins before being breathalysed, in order to obtain accurate readings. The confederate was returned to their testing room to ‘complete these questionnaires’. Participants with a BAL > 0.05% were asked to wait until their BAL fell below 0.05%, and were provided with magazines during this time. At the end of the 2nd session, participant knowledge of the true aims of the study were assessed with a funnel debriefing procedure (Aronson, Ellsworth, Carlsmith, &

Gonzales, 1990). Participants were then fully debriefed and asked to re-affirm written consent for their data to be included in the study. All participants reaffirmed consent.

2.4 Data Analysis

Hypotheses were tested in multilevel regression to account for the nested structure of the data (experimental sessions nested within individuals). Analyses were conducted in *MLwiN* (version 2.30) using full iterative generalized least squares (IGLS) estimation, which automatically addresses missing data using “gold standard” maximum-likelihood procedures (Graham, 2009; Hox, 2002). Contrast-coded (centered) variables were created to model experimental effects of *a priori* interest (Figure 1). The alcohol consumption model was specified as follows:

$$\begin{aligned} \text{Alcohol Consumption}_{ij} = & \beta_{0j} + \beta_1 \text{Peer Influence}_j + \beta_2 \text{Contrast 1 (Reward Cue + Ego} \\ & \text{Depletion vs. Negative Mood)}_j + \beta_3 \text{Contrast 2 (Reward Cue vs. Ego Depletion)}_j + \\ & \beta_4 \text{Induction}_{ij} + \beta_5 \text{Peer Influence} \times \text{Induction}_{ij} + \beta_6 \text{Contrast 1} \times \text{Induction}_{ij} + \beta_7 \text{Contrast} \\ & \text{2} \times \text{Induction}_{ij} + e_{ij} \end{aligned}$$

$$\beta_{0j} = \beta_0 + u_{0j}$$

where $\text{Alcohol Consumption}_{ij}$ is the amount of alcohol consumed (ml) by person j in session i . A similar model was specified for disinhibition and reward-seeking, with SSRT (or BAS-Fun Seeking) as the response variable and contrast-coded variables testing relevant hypotheses. Peer influence parameters were omitted. In all models, Level 2 predictors were grand mean-centered and Level 1 predictors were group mean-centered (Enders & Tofighi, 2007). Random intercepts were specified in all models due to large variance partition coefficients (VPC; or intra-class correlation) for alcohol consumption (.49), disinhibition (.35), and reward-seeking (.54), indicating that 49% of variance in consumption was

attributable to inter-individual differences (35% and 54% for disinhibition and reward-seeking, respectively). There were no *a priori* predictions concerning random slopes and, when specified in the models, did not significantly improve fit. They were not retained.

Predictors of interest were tested for significance using $\Delta\chi^2$ to compare the fit of nested models to the data using $-2 \times \log$ likelihood, in which the parameter of interest was constrained to zero or not. This method tends to have greater statistical power than the Wald test (Hox, 2002). Assumptions of normality and linearity were tested for each model through examination of residuals (Hox, 2002). These assumptions were met for all models tested. Mediation was evaluated using the joint significance procedure, whereby a significant association between IV and mediator (path *a*) combined with a significant association between mediator and DV (path *b*) provides evidence for mediation (Krull & Mackinnon, 2001; Pituch, Whittaker, & Stapleton, 2005). Where there was evidence for mediation, the product-of-coefficients method was used to estimate the unstandardized mediation effect (Tofghi & MacKinnon, 2011).

3. Results

3.1 Data screening and descriptive statistics

Descriptive data are reported in Table 1 (Level 2 variables) and Table 2 (Level 1 variables). All participants completed both testing sessions. Of the total 120 participants, 8 (6.7%) had missing data on C-TRT alcohol consumption for one session. This was due to participants spilling cocktails during the task ($n = 5$), continuing to drink after the task had finished ($n = 1$), or the confederate unexpectedly not attending a heavy-drinking peer session ($n = 2$). For disinhibition, 6 (5%) participants had missing SSRT data for one session due to misunderstanding task instructions, and 13 (10.8%) inhibited responses significantly more or

less than 50% of the time ($p < .05$) on one/both administrations, preventing reliable calculation of SSRT (Verbruggen et al., 2008). During the funnel debriefing, 3 (2.5%) participants revealed a suspicion the confederate was not a genuine participant or was there to influence alcohol consumption, and 9 (7.5%) raised varying degrees of suspicion that total consumption was being measured as part of the C-TRT. Controlling for this knowledge did not affect reported results and data from these participants were retained. Missing data were handled as part of the IGLS estimation, an optimal means of handling missing data (Graham, 2009; Hox, 2002).

There were no significant differences between experimental groups on Table 1 variables resulting from random allocation, except for 28-day alcohol frequency ($p = .045$), whereby mood induction participants ($M = 8.78$, $SD = 4.78$) reported more days of alcohol use than reward cue exposure participants ($M = 6.60$, $SD = 3.25$). Controlling for this variable did not affect model parameter estimates and, thus, was not retained as a covariate. There were no significant differences between (non-randomized) Peer Influence groups on Table 1 variables ($ps < .05$), with allocation stratified by gender to ensure equal distribution of men and women. Intercorrelations between variables are reported in Table 3.

INSERT TABLE 1 HERE

INSERT TABLE 2 HERE

INSERT TABLE 3 HERE

3.2 Covariates and nonspecific effects

All participants completed “right now” measures of craving, positive affect, and negative affect after the experimental and control manipulations, regardless of the type of

induction they were randomly assigned to. Prior to the main analysis, the impact of the experimental manipulations on craving, positive affect, and negative affect were investigated in separate models to examine nonspecific effects and control for them, if necessary. As intended, none of the manipulations produced increased craving after the experimental condition compared to the control condition ($ps > .05$). As expected, only participants receiving the negative mood induction reported an increase in negative affect after the experimental manipulation, $\Delta\chi^2(1) = 14.40, p < .001$. For positive affect, there was a significant reduction after the negative mood and ego depletion manipulations ($\Delta\chi^2[1] = 4.08, p = .04$), while the reward cue exposure had no effect, $\Delta\chi^2(1) = 3.93, p = .05$. Including positive affect and negative affect as covariates did not affect any tested models and, thus, were not retained as covariates. There were no direct effects (or interactions) involving sex on disinhibition ($\Delta\chi^2[1] = 0.19, p = .66$) or reward-seeking ($\Delta\chi^2[1] = 0.47, p = .49$). There was a direct effect of sex in the alcohol consumption model (females drank less; $\Delta\chi^2[1] = 20.91, p < .001$), but sex did not moderate any experimental or peer effects ($ps < .05$), nor did it moderate the association between increased state impulsivity and alcohol consumption in mediation tests ($\Delta\chi^2[1] = 0.02, p = .88$).

3.3 Direct effects on alcohol consumption

On average, 387.73 ml ($SD = 216.27$) of alcoholic cocktail was consumed during the C-TRT. As shown in Table 4, there was no direct main effect of experimental manipulation on alcohol consumption (β_4). Contrary to hypothesis, reward cue exposure and ego depletion, as a group, did not produce a direct increase in alcohol consumption greater than the negative mood induction (β_4). However, reward cue exposure produced a significantly greater increase in alcohol consumption compared to ego depletion (β_7 , Table 4). Compared to the ego

depletion group, participants in this condition consumed approximately 100.80 ml more cocktail after reward cue exposure than after the control film clip. The increase in alcohol consumption after reward cue exposure was not significantly greater than the increase after negative mood induction, despite being in the predicted direction (51.15 ml, $\Delta\chi^2 [1] = 1.26, p = .26$). The presence of a heavy-drinking peer directly increased cocktail consumption by approximately 61.93 ml (β_1) and this effect was statistically significant ($p7$, Figure 1). However, peer influence did not moderate the effect of experimental manipulations on consumption ($p8$, Figure 1).

INSERT TABLE 4 HERE

3.4 Mediating mechanisms and indirect effects on consumption

Change in reward-seeking ('state' BAS-FS) was a mediator of interest. As shown in Table 5 (upper panel), there was no main effect of experimental manipulation on reward-seeking (β_3). As hypothesised, there was a significant interaction indicating that reward cue exposure produced a significantly greater increase in reward-seeking than the other manipulations (β_4 , Table 5; $p1$). Critically, change in reward-seeking predicted subsequent alcohol consumption, *unstandardized coefficient* = 5.13, *SE* = 2.07, $\Delta\chi^2 (1) = 19.22, p < .001$ ($p5$, Figure 1). This provides evidence for mediation (significant path *b*; mediator $\rightarrow$ DV). Using the product-of-coefficients method (Tofighi & MacKinnon, 2011), the unstandardized indirect/mediated effect of reward cue exposure on alcohol consumption was +12.93 ml (95% CI = 0.27, 32.03). The negative mood induction did not significantly reduce reward-seeking to a greater extent than ego depletion (β_5 , Table 5).

INSERT TABLE 5 HERE

Change in disinhibition (SSRT) was another mediator of interest. As shown in Table 5 (lower panel), there was no main effect of experimental manipulation on disinhibition (β_3). There was partial support for disinhibition hypotheses (p_3, p_4). Specifically, while negative mood induction and ego depletion, as a group, did not induce greater disinhibition than reward cue exposure (β_4), negative mood induction produced greater disinhibition than ego depletion (β_5). Participants evidenced slower SSRT after negative mood induction compared to ego depletion, indicating greater disinhibition. When the model was re-run to contrast negative mood induction effects with all other conditions, consistent results were obtained, indicating greater induced disinhibition (+16.90ms, $\Delta\chi^2(1) = 3.67, p = .055$). However, change in disinhibition was unrelated to alcohol consumption, $\Delta\chi^2(1) = 0.33, p = .57$ (p_6 , Figure 1). Therefore, there was no evidence for mediation (non-significant path b ; mediator $\rightarrow$ DV). A summary of findings is depicted in Figure 3.

INSERT FIGURE 3 HERE

Interestingly, a similar pattern of findings emerged when using trait impulsivity measures to predict outcomes. Neither measure predicted disinhibition ($ps > .05$). Trait Sensitivity to Reward predicted both C-TRT alcohol consumption (*unstandardized coefficient* = 11.85, *SE* = 5.02, $\Delta\chi^2(1) = 55.41, p < .001$) and state reward-seeking (*unstandardized coefficient* = 0.30, *SE* = 0.15, $\Delta\chi^2(1) = 45.19, p < .001$). By contrast, I_7 (Impulsiveness), which taps more into disinhibition-related processes, did not predict C-TRT alcohol consumption (*unstandardized coefficient* = -0.75, *SE* = 4.24, $\Delta\chi^2(1) = 45.19, p = .860$), but

was related to state reward-seeking (*unstandardized coefficient* = 0.30, *SE* = 0.12, $\Delta\chi^2(1) = 31.07, p < .001$).

4. Discussion

This study reports the first comprehensive investigation into the causal effect of impulsivity on adolescent alcohol use. Applying the newly developed *EPIIC* paradigm, results support the hypothesized causal role of reward-driven impulsivity in adolescent alcohol use. Reward cue exposure increased state reward-seeking which, in turn, increased alcohol consumption. Consistent with the 2-CARS model (Gullo & Dawe, 2008), negative affect selectively increased disinhibition (stop-signal reaction time), but not reward-seeking or alcohol consumption. Peer influence was not found to moderate the causal effect of impulsivity, but instead directly increased adolescent drinking in an additive fashion. Taken together, these findings demonstrate the utility of a comprehensive experimental approach to understanding the interplay of psychological and social (peer) factors affecting adolescent impulsivity and alcohol use.

Three theoretically-derived experimental manipulations were used in the present study. Reward cue exposure and negative mood led to increases in reward-seeking and disinhibition, respectively. However, only reward-driven impulsivity increased alcohol consumption in adolescents. This finding is consistent with a large body of non-experimental research on the role of reward sensitivity/drive in impulsive behavior and alcohol use (George, Connor, Gullo, & Young, 2010; Gullo, Dawe, Kambouropoulos, Staiger, & Jackson, 2010; Kabbani & Kambouropoulos, 2012; Sharma et al., 2013; Stautz & Cooper, 2013; Wood, Dawe, & Gullo, 2013). It is also consistent with the drinking motives and expectancies literature showing that expectations of positive (social) reinforcement are key

drivers of adolescent heavy drinking (Kuntsche et al., 2005). These expectancies have been linked to the reward sensitivity component of impulsivity specifically, and are targeted in prevention programs for impulsive adolescents (Conrod, Castellanos, & Mackie, 2008; Gullo et al., 2010). Our study provides causal evidence supporting this pathway of risk and its continued focus in adolescent alcohol prevention.

Negative mood induction led to increases in disinhibition and negative affect, consistent with previous experimental studies (Schaefer et al., 2010; Tice et al., 2001). However, as hypothesized, it did not increase adolescent alcohol consumption. The role of negative affect and underlying avoidance-based neural systems in substance use is complex. Bivariate associations are inconsistently reported (Sher, Grekin, & Williams, 2005), experimental effects can depend on the specific emotion elicited (Gullo & Stieger, 2011), and its role in drinking may depend on an individual's stage of development (Kuntsche et al., 2005) and/or past exposure to alcohol (Koob & Volkow, 2010). When controlling for the overlap with reward-related impulsivity traits, negative affect-related impulsivity traits do not reliably predict youth alcohol use (Gullo, Loxton, & Dawe, 2014; Stautz et al., 2017). However, there is clear evidence of a role for negative affect in dependent substance use and relapse after treatment (Koob & Volkow, 2010; Witkiewitz & Villarroel, 2009).

These results are consistent with Gullo and Dawe's (2008) 2-CARS model of impulsive substance use in adolescence. Poor inhibitory control would not be expected to increase the risk of substance use in the absence of an impulse to approach substances that requires inhibition (i.e., high disinhibition combined with reward-seeking). Such an impulse should arise more easily in drinkers with an alcohol use disorder experiencing negative affect-induced disinhibition when alcohol is available (King et al., 2014; Koob & Volkow, 2010; Sinha, 2009). Unlike adolescents, the drinking behaviour of alcohol-dependent patients

is more strongly motivated by tension-reduction and negative reinforcement expectancies (Li & Dingle, 2012; Nicolai et al., 2012). For this population, there is a stronger association between negative affect and reward-seeking, specifically alcohol-seeking. Our results show that in non-dependent adolescent drinkers, impulsivity triggered by reward cues is more likely to increase drinking. Studies utilizing *EPIIC* with problem drinkers (or older drinkers) in whom tension-reduction has been shown to motivate drinking would further test this hypothesis (Nicolai et al., 2012).

The ego depletion manipulation did not increase alcohol consumption or disinhibition. This is consistent with a recent preregistered multi-lab replication reporting no effect of ego depletion on subsequent self-control (Hagger & Chatzisarantis, 2016). These results appear against a backdrop of mixed meta-analytic findings concerning the effectiveness of ego depletion, with some reports of a significant pooled effect (Hagger et al., 2010) and others finding no effect (Carter, Kofler, Forster, & McCullough, 2015). The Crossing-Out Letters Task was chosen for this study because it is one of the more affectively neutral procedures, clearly differentiating it from the reward cue and negative mood manipulations. It also involved suppression of a habitual approach response, which is theoretically more relevant to the present study than other disinhibition approaches that involve the suppression of emotions or thoughts (Hagger et al., 2010; Muraven, Lorraine Collins, & Nienhaus, 2002). To our knowledge, no previous study has used the Crossing-Out Letters Task manipulation in an attempt to influence SSRT or laboratory alcohol consumption. Although, not long after commencing our study, Xu et al. (2014) failed to replicate disinhibition effects on handgrip perseverance and Stroop inhibition across 4 studies with the Crossing-Out Letters Task manipulation. Additionally, the recent preregistered multi-lab replication included a computerised Crossing-Out Letters Task and found no effect on subsequent self-control

(Hagger & Chatzisarantis, 2016). Ego depletion was the only experimental manipulation in our study that did not affect either measure of state impulsivity. Given these recent findings reported by other researchers, it is likely that ego depletion does not increase disinhibition. An alternative approach to inducing non-affective disinhibition may be required in future studies.

The presence of a heavy-drinking peer increased alcohol consumption, but did not influence the effect of impulsivity on drinking. The former is consistent with results from previous experimental studies on peer effects (Quigley & Collins, 1999). With respect to impulsivity, the present findings suggest the role of peer influence in adolescent drinking may be additive. That is, regardless of the level of impulsivity, adolescents in general are likely to drink more alcohol in the presence of heavy drinkers. Being impulsive would simply add further to this risk. However, it is possible that the nature of the peer influence employed in this study contributed to the lack of moderating effect. Recent work by Fairbairn and colleagues (2015) suggests increased responsiveness to specific social cues (e.g., Duchenne smiling) may underlie the alcohol-related reward experienced by extraverted individuals. Extraversion is closely aligned with trait reward sensitivity/drive, and there is evidence suggesting individual differences in reward sensitivity lies at the core of extraversion (Dawe & Loxton, 2004; Lucas & Diener, 2001). Given that the peer manipulation employed in the present study was highly standardized and instructed participants and confederates not to talk to each other, this may also explain the lack of hypothesized moderation effect. It should be noted, however, that Fairbairn et al. did not measure laboratory alcohol consumption in their study, but rather self-reported mood and social reward.

The peer influence manipulation used in this experimental setting lacked a number of elements common to adolescent drinking contexts. While highly controlled to provide strong

internal validity, future research would benefit from incorporating other social elements (e.g., Duchenne smiling, scripted conversation; Fairbairn et al., 2015). Another limitation was the relatively narrow behavioral assessment of impulsivity, in that only experimental effects on one measure (of disinhibition) was assessed. Studies utilizing other behavioral disinhibition measures, as well as behavioral measures of reward-seeking, will provide a more complete understanding of mediating mechanisms. Inter-individual differences in psychiatric symptoms (e.g., depression) may have affected paradigm response (e.g., reward-seeking), but were not assessed. This is an area for future research. The repeated measures design controlled for the large inter-individual differences in alcohol consumption (due to weight, sex, alcohol use history). Although largely consistent with self-report studies on younger adolescents, the generalizability of laboratory results obtained with 18-21 year-old adolescents may differ in important ways and this should be taken into account when interpreting findings. Indeed, the greater susceptibility of experimental studies in general to chance findings and unintended design effects (e.g., behavioral measure used) highlight the need for replication. However, the similar overall pattern of findings observed with trait self-report measures is encouraging. Lastly, despite producing the intended effects, differences in the length of film clips used for reward cue exposure and negative mood induction reduces standardization across manipulations. Future studies should seek to employ clips of equal length across domains. The ego depletion manipulation was the only manipulation not to involve a film clip and, despite being standardized by the use of a script, was nonetheless more prone to experimenter effects due to lack of blinding.

These limitations should be considered alongside some notable strengths. These include a comprehensive experimental approach that incorporated different methods of inducing impulsivity, incorporating social factors, the measurement and control of

nonspecific experimental effects, and the matched mode of delivery for reward cue and negative mood inductions with experimenter blinding.

In summary, this is the first comprehensive study of the causal effect of impulsivity on adolescent alcohol use. Only reward-driven impulsivity increased alcohol consumption, suggesting impulsivity-targeted prevention should maintain a focus on positive outcome expectancies and alternative sources of reward. Negative mood increased disinhibition but not adolescent drinking. Mood may be a more relevant intervention target for clinical populations. Peer influence had a direct effect on alcohol consumption, independent of impulsivity, and is likely an additive risk factor for heavy drinking. The new *EPIIC* paradigm shows promise as a methodology for advancing the understanding of adolescent impulsivity and alcohol use. Future studies could investigate the role of other hypothesized moderators and mediating mechanisms within this comprehensive model (Craske, 2016), or just focus on one of the manipulations in greater detail. Future translational research could employ *EPIIC* to evaluate the specific effects of intervention techniques within a controlled context (Bujarski & Ray, 2016). It is hoped such efforts will eventually inform new approaches to early intervention with impulsive youth.

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Figure Legends

Figure 1. Experimental Paradigm for Investigating Impulsive Consumption (EPIIC) and hypothesized effects in adolescent drinkers.

Note. Solid lines depict hypothesized direct causal effects of a positive (+) or negative (-) direction. Dashed lines depict hypothesized moderation effects.

Figure 2. Procedure flowchart for Experimental Paradigm for Investigating Impulsive Consumption (EPIIC).

Note. Levels of between-subjects variables appear in bold. Levels of within-subjects (repeated measures) variables appear in italics. BAL = Blood Alcohol Level.

Figure 3. Summary of key findings.

Note. Unstandardized effects are reported.

$*p < .05$; $**p < .01$; $***p < .001$.

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Table 1

Descriptive Statistics for Self-Reported Alcohol Use and Impulsivity (N = 120; 50% male).

	Reward Cue Exposure (n = 40)	Negative Mood Induction (n = 40)	Ego Depletion (n = 40)	<i>F</i>	<i>p</i>
Age (years)	19.58 (1.13)	19.50 (1.04)	19.33 (1.21)	0.52	.60
28-Day Alcohol Quantity (grams)	439.29 (359.86)	602.49 (383.50)	439.91 (378.80)	2.53	.08
<i>Quantity in U.S. standard drinks</i>	31.38 (25.70)	43.04 (27.39)	31.42 (27.06)	-	-
28-Day Alcohol Frequency (days)	6.60 ^a (3.25)	8.78 ^b (4.78)	7.35 (3.56)	3.18	.045
ASSIST Alcohol SSI	10.13 (6.51)	12.40 (6.74)	11.15 (5.86)	1.28	.283
Sensitivity to Reward	10.72 (3.90)	11.34 (3.18)	11.38 (3.73)	0.41	.66
I ₇ (Impulsiveness)	6.83 (4.88)	7.26 (4.08)	7.34 (4.23)	0.16	.86

Note. Means with different superscripts are significantly different at $p < .05$. A U.S. standard drink contains 14g of pure alcohol (ethanol). ASSIST = Alcohol, Smoking and Substance Involvement Screening Test; SSI = Specific Substance Involvement.

Table 2

Means and standard deviations (SD; in parentheses) for Level 1 variables.

		Reward Cue Exposure (<i>n</i> = 40)	Negative Mood Induction (<i>n</i> = 40)	Ego Depletion (<i>n</i> = 40)
Reward-Seeking (BAS-Fun Seeking)	Control	19.78 (6.59)	21.73 (6.06)	22.08 (6.80)
	Experimental	20.88 (5.28)	19.30 (7.19)	21.63 (6.37)
Disinhibition (SSRT; ms)	Control	256.29 (56.94)	254.46 (37.45)	259.53 (33.15)
	Experimental	256.44 (41.54)	263.42 (41.33)	247.74 (42.18)
Alcohol Consumption (C-TRT; mls)	Control	355.40 (201.49)	380.84 (198.72)	401.42 (193.45)
	Experimental	421.22 (271.45)	380.66 (198.43)	387.58 (230.72)
Blood Alcohol Level (BAL) after C-TRT	Control	.028 (.024)	.035 (.021)	.035 (.017)
	Experimental	.032 (.025)	.033 (.019)	.043 (.053)

Note. SSRT = Stop-Signal Reaction Time; C-TRT = Cocktail Taste Rating Task.

Table 3

Intercorrelations between study variables.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Age	-												
2. Sex (0 = Male, 1 = Female)	.134	-											
3. TLFB Quantity	-.051	-.214*	-										
4. TLFB Frequency	.215*	.010	.499**	-									
5. Alcohol SSI	.072	.033	.350**	.301**	-								
6. Sensitivity to Reward	.069	-.074	.377**	.177	.262**	-							
7. I ₇ Impulsiveness	-.022	.148	.195*	.118	.214*	.478**	-						
8. Reward-Seeking (Control)	-.049	.153	.131	.067	-.013	.253**	.405**	-					
9. Reward-Seeking (Exp)	-.086	-.050	.209*	-.022	-.065	.295**	.220*	.544**	-				
10. Disinhibition (SSRT, Control)	-.041	.078	.059	-.045	-.104	.052	.082	-.036	.016	-			
11. Disinhibition (SSRT, Exp)	-.167	-.072	.151	.137	.112	.047	.054	-.072	-.118	.355**	-		
12. Cocktail consumed (ml; Control)	-.061	-.335**	.256**	.229*	.213*	.191*	.123	.123	.216*	-.012	.225*	-	
13. Cocktail consumed (ml; Exp)	-.007	-.443**	.268**	.081	.169	.258**	.098	.014	.256**	.039	.066	.473**	-

Note. TLFB = 28-day Timeline Followback; SSI = Substance Specific Involvement; Exp = Experimental; SSRT = Stop-Signal Reaction Time (ms).

* $p < .05$

** $p < .01$

*** $p < .001$

Table 4

Experimental and peer effects on alcohol consumption (N = 120)

Parameter	Unstandardized coefficient	SE	$\Delta\chi^2(1)$	p
<i>Fixed effects</i>				
Intercept, β_{0j}	373.68	15.23	-	-
Peer Influence, β_1	61.93	30.47	4.04	.044
(C1) Reward Cue + Ego Depletion vs. Negative Mood, β_2	10.47	32.50	0.10	.751
(C2) Reward Cue vs. Ego Depletion, β_3	-0.30	37.46	0.00	1.00
Induction, β_4	0.99	18.65	0.003	.956
Peer Influence x Induction, β_5	-12.27	37.32	0.11	.740
C1 x Induction interaction, β_6	0.75	39.69	0.00	1.00
C2 x Induction interaction, β_7	100.80	46.04	4.72	.030
<i>Random effects</i>				
σ_e^2	19013.04	2592.13		
σ_{u0}^2	16815.87	3770.51		
Deviance (-2*log likelihood)	2944.72			

C1 = Contrast 1 (Reward Cue, +0.33; Ego Depletion, +0.33; Negative Mood, -0.66); C2 = Contrast 2 (Reward Cue, +0.5; Ego Depletion, -0.5; Negative Mood, 0).

Table 5

Experimental effects on 'state' reward-seeking and disinhibition (N = 120)

Parameter	Unstandardized coefficient	SE	$\Delta\chi^2(1)$	p
Reward-Seeking ('state' BAS-FS)				
<i>Fixed effects</i>				
Intercept, β_0	20.91	0.51	-	-
(C1 ^a) Reward Cue vs. Ego Depletion + Negative Mood, β_1	-0.88	1.09	0.65	.420
(C2 ^a) Negative Mood vs. Ego Depletion, β_2	-1.30	1.25	1.07	.301
Induction, β_3	-0.56	0.54	1.08	.299
C1 x Induction interaction, β_4	2.52	1.16	4.64	.031
C2 x Induction interaction, β_5	-1.91	1.33	2.04	.153
<i>Random effects</i>				
σ_e^2	17.51	2.25		
σ_{u0}^2	22.39	4.20		
Deviance (-2*log likelihood)	1514.25			

Disinhibition (Stop-Signal Reaction Time; SSRT)

Fixed effects

Intercept, β_{0j}	257.89	3.10	-	-
(C1 ^b) Negative Mood + Ego Depletion vs. Reward Cue, β_1	0.31	6.76	0.003	.956
(C2 ^b) Negative Mood vs. Ego Depletion, β_2	4.90	7.46	0.43	.512
Induction, β_3	-0.72	4.11	0.13	.718
C1 x Induction interaction, β_4	3.19	8.96	0.13	.718
C2 x Induction interaction, β_5	20.21	9.90	4.11	.043

Random effects

σ_e^2	850.92	121.49
σ_{u0}^2	609.61	157.83

Deviance (-2*log likelihood) 2097.63

^aC1 = Contrast 1 (Reward Cue, +0.66; Ego Depletion, -0.33; Negative Mood, -0.33); C2 = Contrast 2 (Reward Cue, 0; Ego Depletion, -0.5; Negative Mood, +0.5).

^bC1 = Contrast 1 (Reward Cue, -0.66; Ego Depletion, +0.33; Negative Mood, +0.33); C2 = Contrast 2 (Reward Cue, 0; Ego Depletion, -0.5; Negative Mood, +0.5).

Figure 1

Figure 2

Figure 3

Highlights

- Impulsivity is robust predictor of adolescent alcohol use, but causal evidence lacking
- Developed first laboratory model of adolescent impulsivity and alcohol use
- Reward exposure increased reward-seeking and adolescent alcohol use
- Negative mood increased disinhibition, but not alcohol use
- Impulsivity effects independent of sizeable peer influence on drinking

Supplemental Material

Randomisation and Blinding

Stratified, restricted randomisation was conducted by an independent person using *Random Allocation Software* (Saghaei, 2004). Random allocation was stratified by gender to ensure an equal number of male and female participants in each condition. It was also stratified by the peer influence variable because allocation to the heavy-drinking peer condition was limited by the availability of gender-matched confederates, who were student volunteers. Therefore, peer influence was a quasi-experimental variable. Confederates were blind to experimental condition, and various measures were in place to ensure the experimenter was blind to experimental condition for the reward cue exposure and mood induction groups. Film clips were played on a laptop computer through headphones and commenced with a 10-s fade in and ended with a fade to black. The experimenter started the clips using computer desktop shortcuts to conceal file names, which were set up by an independent person. Due to the nature of the crossing-out letters task, which requires experimenter involvement, the experimenter was not blind to condition in the ego depletion group.

Order effects

There were order effects for C-TRT consumption and SSRT. On average, participants consumed 60.52 ml ($SE = 21.61$) more alcohol during session two, $t(119) = 2.80, p = .005$. For SSRT, participants were 28.54 ms ($SE = 3.63$) faster during session two, indicating less behavioral impulsivity, $t(119) = 7.85, p < .001$. The counterbalanced design was intended to control for possible order effects. Statistically controlling for session order did not affect model parameter estimates and was not included as a covariate.

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