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Acute alcohol effects on impulsive choice in adolescents

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Abstract

Background: Neurodevelopmental and alcohol-induced changes in decision-making have been proposed to critically influence impulsive behaviour in adolescents.

Objective: This study tested the influence of acute alcohol administration on impulsive choice in adolescents.

Methods: Fifty-four males aged 18–19 years were tested in a single-blind placebo-controlled cross-over design. During alcohol administration (infusion resulting in an arterial blood alcohol concentration of 80 mg%) and placebo condition (saline infusion), participants performed a task battery providing estimates of delay discounting, probability discounting for gains, for losses and loss aversion, and also rated subjectively experienced alcohol effects. Additionally, baseline alcohol consumption (Alcohol Use Disorders Identification Test, blood phosphatidylethanol levels), motives (Drinking Motive Questionnaire, Alcohol Expectancy Questionnaire and Obsessive Compulsive Drinking Scale), family history and self-report measures of impulsivity (Barratt Impulsiveness Scale, Substance Use Risk Profile Scale) were provided.

Results: No overall effects of treatment on choice behaviour were found. However, individual differences were observed. In the alcohol condition, more impulsive choice tendencies for delay discounting were associated with higher subjectively experienced alcohol effects. Further, higher risk aversion for probabilistic gains and higher loss aversion during alcohol condition were related to higher levels of real-life alcohol consumption and a family history of alcohol problems, respectively. Finally, the time to make a decision was substantially shortened for choices involving negative prospects.

Conclusions: Contrary to common beliefs, acute alcohol intoxication did not generally incite impulsive decision-making. It rather appears that alcohol-induced behavioural changes in adolescents vary considerably depending on prior experiences and subjective effects of alcohol.

Keywords

Computer-assisted Alcohol Infusion System (CAIS), discounting, risk, loss aversion, adolescence, decision-making

Introduction

Adolescence is a time of substantial change in social, emotional, cognitive and neurobiological development (Dahl, 2004), which often coincides with the first experiences of unrestricted alcohol consumption (WHO, 2001). Adolescence in general and alcohol intoxication in particular are associated with an increased vulnerability to harm such as injuries, car accidents, violent crimes and unprotected sexual activities (Cherpitel, 1993; Pernanen, 1976; Swahn et al., 2004). At least some of these problematic behaviours are thought to be the consequence of alterations in impulsive decision-making (Balogh et al., 2013; Story et al., 2014).

Impulsivity is a multifaceted construct (Evenden, 1999) that consists of at least two distinct yet related dimensions: ‘impulsive action’ and ‘impulsive choice’ (MacKillop et al., 2016). Accordingly, established behavioural instruments have been classified to broadly map onto these dimensions (Weafer et al., 2013). Typically, the ability to refrain from pre-potent responses, which can be measured in Stop-Signal or Go/No-go tasks (Donders, 1969; Logan et al., 1984) assess impulsive action. In contrast, impulsive choice can be assessed through (a) delay discounting (DD), reflecting the devaluation of future consequences as a function of delay to their occurrence (Ainslie, 1975), and (b) probability discounting, a conceptually similar but distinct process in which a consequence is devalued due to its probabilistic occurrence (Rachlin et al., 1991).

Impulsivity has traditionally been conceived as a stable internal disposition (McCrae et al., 2000). Yet, pronounced reductions in impulsivity occur during the transition from adolescence to young adulthood (Roberts et al., 2006). Impulsivity can even fluctuate within short intervals; acute alcohol consumption, for example, has repeatedly been shown to trigger increases in impulsive action (De Wit et al., 2000; Fillmore and Vogel-Sprott, 1999; Loeber and Duka, 2009; Marciszinski et al., 2005). On the other hand, findings regarding acute effects of alcohol on impulsive choice are less clear (see Mayhew and Powell, 2014).

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Alcohol-dependent subjects exhibit higher rates of DD compared to nondependent social drinkers (Bernhardt et al., 2017; MacKillop et al., 2011). Consequently, acute alcohol intoxication in non-dependent drinkers has been theorized to dynamically affect DD making them more impulsive. However, results so far are inconsistent. While the majority of studies show no significant alcohol effect on DD (Bidwell et al., 2013; Reynolds et al., 2006; Richards et al., 1999), trend-level reduced (Ortner et al., 2003) or increased (Reynolds et al., 2006) rates following alcohol administration have also been reported. Likewise, variable findings exist for probability discounting with either no effect (Richards et al., 1999) or reduced discounting rates following alcohol intake in non-dependent drinkers (Bidwell et al., 2013; Johnson et al., 2016).

Discrepancies may arise from procedural variability including route of alcohol administration, setting (laboratory/bar), employed task, commodity and amount of reward but also sample composition (age, gender). Such factors may confound interpretation of acute alcohol effects (Bjork and Gilman, 2014), and additionally question generalizability to alcohol-induced impulsive choice behaviour in adolescents. Notably, adolescents are less affected by neuropsychological deficits in decision-making due to previous alcohol exposure than older participants. Further, studies so far have focused on discounting of rewards (predominantly monetary) warranting additional investigations on choice behaviour facing punishments or losses. This is particularly important since decision-making when intoxicated can often result in negative outcomes.

In an effort to address these open questions, the purpose of this study was to examine how a highly standardized ethanol exposure modulates different facets of impulsive choice in adolescence. We focused on socially drinking 18–19-year-old males without a history of alcohol dependence. According to German regulations, this represents the youngest age group allowing experimental alcohol administration. In a randomized crossover design, participants performed four independent paradigms, (1) DD, (2) probability discounting for gains (PDG), (3) probability discounting for losses (PDL) and (4) mixed gambles (MG), once during acute alcohol administration inducing a stable blood alcohol concentration of 80mg% and once during placebo administration.

During DD, decisions for smaller immediate over larger later rewards signify high DD representing an inability to wait for delayed gratification and, thus, impulsive choice patterns (Hamilton et al., 2015). During PDG, repeated decisions for smaller certain over larger uncertain rewards signify high probability discounting and represent risk-averse choice behaviour. Within the framework of impulsivity a more risk-seeking choice pattern is considered impulsive. This view is supported by the, albeit weak, negative correlation between discounting rates of DD and PDG (Mitchell and Wilson, 2010; Myerson et al., 2003; Richards et al., 1999). The interpretation of discounting behaviour in PDL is inverted due to the negative sign of option outcomes. Here, repeated decisions for larger uncertain losses over smaller certain losses indicate high probability discounting, that is, a higher degree of risk seeking to avoid sure losses. The common choice tendency of risk-seeking behaviour in the domain of losses (Kahneman and Tversky, 1979) and investigations in alcohol use disorder patients suggest that lower PDL (i.e. favouring small but definite losses) represents more impulsive behaviour (Bernhardt et al., 2017). Finally, the MG task measures the degree

of loss aversion via repeated decisions to accept or reject a 50/50% gamble of variable gains and losses (Tom et al., 2007). Low loss aversion has been suggested to indicate reduced punishment sensitivity and increased impulsivity (Brevers et al., 2014; Ernst et al., 2014).

Thus, we hypothesized more impulsive choice behaviour during alcohol compared to placebo infusion, that is, higher DD, lower PDG, lower PDL and lower loss aversion in the MG task. Additionally, associations of choice behaviour with subjective alcohol effects, self-reported impulsivity, measures of alcohol consumption and alcohol-related questionnaires were explored.

Methods

The study protocol was approved by the ethics committee of the Technische Universität Dresden (EK 227062011) and fully complied with the World Medical Association Declaration of Helsinki as revised in 2013.

Participants

The study was part of a prospective longitudinal study of a community sample (randomly sampled by the local resident's registration office) within a large German research consortium investigating the role of learning in the development and maintenance of alcohol use disorders (LeAD, DFG FOR1617). Main cohort participants ($n = 201$) completed the program's standard assessment battery, including the Composite International Diagnostic Interview (CIDI; Jacobi et al., 2013), questionnaires, cognitive and decision-making tasks as well as learning tasks in an independent fMRI session (Nebe et al., 2018). Native German speaking males, 18 years of age, reporting social drinking (≥ 2 drinking days per month during the last three months) were included. Exclusion criteria comprised a history of substance use disorder according to DSM-IV (except nicotine) and other major psychiatric or neurological disorders. Participant sampling for subsequent experiments involving acute alcohol infusion has been previously described (see Jünger et al., 2017). Briefly, adolescents were screened for normal levels of aspartate-amino transferase, alanine-amino transferase and γ -glutamyl transpeptidase to (a) ensure proper liver function and (b) exclude participants with elevated liver enzymes indicating excessive alcohol use. Further exclusion criteria were current medication that could interact with alcohol, a positive drug screen on testing days, and self-reported consumption of alcohol at the test day or the day before. Of 59 initially included participants, four experienced complications due to the infusion ($n = 2$ during venepuncture, $n = 2$ nausea and vomiting) and one participant was lost to day two. Our final sample consisted of 54 males, aged 18 ($n = 44$) or 19 years ($n = 10$).

All subjects gave written informed consent prior to taking part in the experiments.

Measures and tasks

Self-report measures

Temperament traits. The German short versions of the Barratt Impulsiveness Scale (BIS-15 with subscales non-planning, motor and attentional impulsivity; Meule et al., 2011) was used to assess self-reported impulsivity. The Substance Use Risk

Profile Scale (SURPS; Woicik et al., 2009) contains four personality trait scales (anxiety sensitivity, hopelessness, sensation seeking, impulsivity), which are predictive for substance use-related problems during late adolescence and early adulthood (Castellanos-Ryan et al., 2012; Jurk et al., 2015).

Alcohol related constructs. The Alcohol Use Disorders Identification Test (AUDIT) was used to examine alcohol consumption and related problems (Saunders et al., 1993). It comprises 10 questions using either a three-point or five-point Likert scale. AUDIT scores are associated with other subjective and objective measures of alcohol consumption (Coulton et al., 2006; Kip et al., 2008). AUDIT scores ≥ 8 represent the threshold for risky alcohol use and related problems (Reinert and Allen, 2007).

Current alcohol-related problems were further quantified using the German version of the Obsessive Compulsive Drinking Scale (OCDS-G; Mann and Ackermann, 2002), a quick and reliable 14-item self-rating instrument measuring some cognitive aspects of alcohol 'craving' (Anton et al., 1995).

The Brief Alcohol Expectancy Questionnaire (AEQ-G; Demmel and Hagen, 2002) was used to describe anticipated reinforcement from alcohol. It comprises the scales tension reduction (9 binary items) and social facilitation (10 binary items), which are associated with both quantity and frequency of alcohol consumption (Demmel et al., 2006).

The Drinking Motives Questionnaire (DMQ-R; Kuntsche et al., 2006) was used to assess reasons to drink on four motivational dimensions: Drinking to be sociable and to celebrate parties (DMQ-Social), drinking because it makes you forget about problems (DMQ-Coping), drinking to feel better or to be able to do things otherwise impossible (DMQ-Enhancement) and drinking because others do or the social pressure to fit in (DMQ-Conformity). All scales are associated with alcohol consumption and related adverse outcomes (Kuntsche et al., 2014).

Biological marker. Baseline blood phosphatidylethanol (PEth) levels were used as biomarker to quantify alcohol consumption (see Supplementary methods online). Blood concentrations of PEth have been found to highly correlate with self-reported alcohol intake (Aradottir et al., 2006).

Impulsive choice behaviour. Impulsive choice was assessed using a value-based decision-making (VBDM) battery including four independent tasks to measure (1) DD, (2) PDG, (3) PDL and (4) loss aversion (Bernhardt et al., 2017; Pooeh et al., 2018). In all tasks, participants repeatedly had to decide for one of two offers that were presented simultaneously on a computer screen, based on which is the more appealing to them (forced choice). Offers were randomly assigned to the left or to the right of the screen. There was no time limit to make a choice between the options presented in each trial. Outcomes of gambles were never presented during the experiment. To ensure realistic choices and increase task relevance, subjects were informed that at the end of the experiment one trial per task was selected randomly, played by the computer according to their choice and credited to their compensation.

The DD task consisted of 30 trials, during which participants had the choice to either take a small immediate or a larger later

reward. Delays were set to the values 3, 7, 14, 31, 61, 180 and 365 days. Immediate and delayed monetary rewards ranged from €0.30–10. The PDG task consisted of 30 trials, during which participants had the choice to either take a sure monetary reward, or gamble between a larger and no reward. Gambles were played with five possible probability values: 2/3, 1/2, 1/3, 1/4 and 1/5. Monetary rewards ranged from €0.30–10. The PDL task consisted of 30 trials, during which participants had the choice to either take a sure loss, or gamble for no loss or a larger loss. Gambles were played with five possible probability values: 2/3, 1/2, 1/3, 1/4 and 1/5. Monetary losses varied within a limited €0.3–10 interval. During 40 trials of the MG task, participants had the choice to either accept or decline a gamble for a 50% chance of winning or losing a certain amount of money. Amounts were set to €1–40 and €5–20 for wins and losses, respectively. At the beginning of the MG task, subjects were told to receive €10 'house money' and informed of the risk of gaining or losing money during the experiment.

All tasks including instructions, binary choice trials, and outcomes were presented using MATLAB release 2010a (The MathWorks, Inc., Natick, MA, USA) and the Psychtoolbox 3.0.10 based on the Psychophysics Toolbox extensions (Brainard, 1997; Pelli, 1997). To provide behavioural estimates of impulsive choice (k , λ) and consistency (β), an adaptive procedure for binary choice presentation was used. For a detailed description of the mathematical framework, see Pooeh et al. (2018). Briefly, a trial-by-trial adaptive approach was chosen to present participants with choice options near their individual indifference points at each trial, which are most informative and allow for fast assessment of the individual parameters.

For DD, a simple hyperbolic discounting function, which states that the subjective value (V) of an outcome of amount A obtained following delay D declines hyperbolically; $V = A / (1 + kD)$ was used (Mazur, 1987) to estimate the degree of discounting.

Analogous to DD, the degree of discounting of probabilistic gains and losses was estimated using $V = A / (1 + k\theta)$ with θ being the odds against the event of winning or losing, respectively.

For mixed prospects (MG task), we used $V = 1/2(G - \lambda L)$ to estimate a behavioural measure of loss aversion (λ), computed as the ratio of the contribution of the loss magnitude L to that of the gain magnitude G to the subject's decision (Tom et al., 2007).

Additionally, estimates of deliberation time were computed for each task separately. Given that behavioural estimates were assessed with this adaptive approach, the distribution of parameter estimates over task progression was initially plotted and found to converge well yielding stable final estimates of choice behaviour (Supplementary Figure 1). Further quality control of collected data (Supplementary Table 1) resulted in a final inclusion of all data for analysis.

Subjective alcohol effects. Subjectively experienced alcohol effects for both infusion conditions were assessed as previously described (Jünger et al., 2017). We used reports obtained shortly before the VBDM battery (120 min), for stimulation: 'Right now, I am experiencing stimulating alcohol effects, e.g. cheerful, excited, full of energy, full of zest for action...'; sedation: 'Right now, I am experiencing sedating alcohol effects, e.g. relaxed, tired, sluggish...'; feeling drunk: 'I am feeling drunk right now.'; and negative effects: 'Right now, I am experiencing negative

alcohol effects, e.g. nausea, dizziness, ringing in the ear...'. Statements were presented sequentially on a computer screen and answered using vertical visual analogue scales anchored at 0 (not at all) and 100 (extremely).

General experimental procedure

Participants underwent two experimental days, separated by 6–22 days (median = 7), that involved intravenous infusion of alcohol or placebo in a randomized crossover design. Subjects were misinformed that they would receive 'different alcohol dosages' on either day in order to sustain their expectancy of alcohol. Experimental sessions were started at the same daytime to exclude circadian effects. For both alcohol and placebo administration, an 18G i.v. line on the non-dominant arm was established to deliver either a 0.9% saline and/or a 6.0% (v/v) ethanol solution in 0.9% saline (Alkohol Konzentrat 95% Braun, Melsungen, Germany) via the Computer-assisted Alcohol Infusion System (CAIS; O'Connor et al., 1998). Participant's age, sex, height and weight were entered into a Physiologically-Based Pharmacokinetic (PBPK) model (Plawecki et al., 2012; Ramchandani et al., 1999). During the ascending limb phase, arterial blood alcohol concentrations (aBACs) were linearly increased up to the target level of 80 mg% (0.08%) within 25 min. After that, aBAC levels were clamped, that is, kept stable for two hours and repeatedly validated using an Alcotest 6810med breathalyser (Draeger Sicherheitstechnik, Lübeck, Germany). Breathalyser data were entered into the CAIS software to improve individual pharmacokinetic models in real time and to adapt the infusion rates accordingly. The breathalyser measured alcohol concentration in end-expiratory breath, which is closely related to aBAC during intravenous ethanol infusion, providing a reliable estimate of brain alcohol exposure. During the stable aBAC phase, participants completed a set of experiments including the VDBM battery (Figure 1) and ratings of subjectively experienced alcohol effects. At the end of each session, the i.v. line was removed and participants were paid task-specific winnings. At the end of the second experimental day, participants were debriefed and received €100 compensation.

Data analysis

A priori power calculations were carried out using G*Power (<http://www.gpower.hhu.de/>), based on previous work reporting behavioural parameters that were measured after drinking moderate alcohol doses in humans with observed effect sizes between 0.2 and 2 (Cohens' d : $M = 0.9$) (George et al., 2005; Guillot et al., 2010; Loeber and Duka, 2009; Ramaekers and Kuypers, 2006). We computed the required sample size for two-tailed t -tests for matched pairs with an effect size of $d = 0.4$ for condition (alcohol vs. placebo), an alpha level of 0.05 and a power of 0.8, which was $N = 52$ for a t -test and $N = 54$ for a Wilcoxon signed-rank test. Choice parameters k and λ were used on log scale. Deliberation time analysis was performed using the individual median over all trials. Shapiro Wilk Tests and visual inspection of Q-Q-plots were used to judge normality ($p < .05$). In accordance, interrelations between measures were analysed with Pearson's or Spearman's correlation and group effects of alcohol infusion were tested using paired sample t -tests or

Wilcoxon signed-rank tests. Linear regression analyses were done with behavioural choice parameters during alcohol administration as dependent variable. For explorative analyses individual differences between conditions were computed subtracting parameters obtained during placebo condition from alcohol condition ($\text{Diff_scores} = x_{80\text{mg\%}} - x_{\text{placebo}}$) followed by explorative correlational analyses with variables of interest including measures of subjective effects, personality and alcohol use. The significance level for all analyses was set to $\alpha = .05$ (two-tailed). Analyses were conducted using MATLAB, Statistics Toolbox Release 2014a (The MathWorks, Inc., Natick, MA, USA) and SPSS (IBM Corp, Released 2013, Version 22.0, Armonk, NY, USA).

Results

Sample characteristics

All participants were normal weight (BMI mean = 22.8, SD = 2.54). The majority (80%) had >10 years of school education, 6% had a migrant status (parents born outside Germany) and 29.6% were regular smokers. Participants had their first alcoholic drink between 10 and 16 years of age and AUDIT scores (range 3–25; Table 1) indicated risky alcohol consumption and related problems in 37% of participants (AUDIT score ≥ 8). Further characteristics are presented in Table 1.

Experimental parameters

The average breathalyser-measured alcohol concentration was 0.0 mg% for the placebo condition and 0.79 mg% (SD = 0.04) for the alcohol condition (time point 125 min, just before VDBM battery). Accordingly, all subjective alcohol effects were rated higher during alcohol compared to placebo infusion (all p -values $< .001$; Table 2).

Alcohol effects on impulsive choice

Paired sample t -tests showed that there was no statistical difference in the group means comparing placebo and alcohol condition in any of the four decision-making parameters (Figure 2(a)).

To explore the possibility that there was a more complicated interaction between treatment and choice preference, we examined the parameter distribution in the two treatment conditions across our sample using a linear regression (Figure 3). In all models, choice behaviour during placebo explained a significant proportion of variance in choice behaviour during alcohol administration (DD: ($F(1,53) = 142.97, p < .000, R^2 = .733$; PDG: ($F(1,53) = 14.67, p < .000, R^2 = .496$; PDL: ($F(1,53) = 4.04, p = .05, R^2 = .273$; MG: ($F(1,53) = 46.59, p < .000, R^2 = .687$). Also the regression coefficients for all four models were positive and significant (DD: $\beta = 0.831, t(53) = 11.97, p < .001$; PDG: $\beta = 0.477, t(53) = 3.83, p < .001$; PDL: $\beta = 0.357, t(53) = 2.01, p = .05$; MG: $\beta = 0.692, t(53) = 6.83, p < .001$). As can be seen in Figure 3(a), some of the individual values are located above the unity line, indicating that choice parameters were higher in the alcohol condition relative to the placebo condition. Other values were located below the unity line, indicating that parameters were lower in the alcohol condition relative to the

Figure 1. Study design. Top: Timeline of infusion and tasks including measures of subjective alcohol effects and impulsive choice (value-based decision-making). Arterial blood alcohol concentration (aBAC) was clamped at 80 mg% from 25 min to 145 min during alcohol infusion, while during placebo participants received a physiological NaCl solution. Bottom: Experimental tasks were performed in the presented order. Examples of binary choice presentations for DD: delay discounting, PDG: probability discounting for gains, PDL: probability discounting for losses and MG: mixed gambles, as presented to the participants.

placebo condition. Note that as indicated by the consistency parameters choices are not just more random during alcohol administration (Supplementary Figure 2).

As a measure of the deviation from the unity line, individual Diff_scores = (alcohol – placebo) were calculated for each task (DD: range -2.87 – 3.62 , $M = -0.13$, $SD = 1.43$; PDG: range -1.62 – 2.26 , $M = 0.16$, $SD = 1.01$; PDL: range -4.20 – 3.10 , $M = 0.03$, $SD = 1.27$; MG: range -1.67 – 1.44 , $M = -0.01$, $SD = 0.58$). High Diff_scores in DD, representing more impulsive choice during alcohol administration, were found to associate with higher subjectively experienced stimulation during alcohol infusion ($r(54) = .298$, $p = .029$), self-reported individual craving for alcohol (OCDS, $\rho(54) = .279$, $p = .041$) and higher scores assessing motives to drink for its enhancing properties (DMQ-E, $\rho(54) = .288$, $p = .035$). Higher risk aversion during acute alcohol administration was associated with higher real-life alcohol consumption (blood PEth levels, $\rho(54) = .283$, $p = .044$; AUDIT $\rho(54) = .486$, $p < .001$) and higher scores assessing motives to drink alcohol for coping (DMQ-C, $\rho(54) = .333$, $p = .014$). Higher risk-seeking in the alcohol condition for PDL was associated with more self-reported individual craving for alcohol (OCDS-G, $\rho(54) = .3312$, $p = .014$). And finally, higher loss aversion when intoxicated was associated with a positive FH ($r(54) = .362$, $p = .007$) and lower scores on motives to drink alcohol for coping (DMQ-C, $\rho(54) = -.375$, $p = .005$). These explorative findings are supported by additional split-group analyses (see Supplementary material) and thus suggestive of meaningful individual differences in impulsive decision-making during alcohol administration.

Alcohol effects on deliberation times

Implemented in the task design, participants were offered a theoretically unlimited time to deliberate over each choice before making a decision. Individual deliberation times correlated consistently between alcohol and placebo infusion within each task, (DD: $\rho(54) = .710$, $p < .001$; PDW: $\rho(54) = .583$, $p \leq .001$; PDL: $\rho(54) = .544$, $p < .001$; MGA: $\rho(54) = .701$, $p < .001$). In addition, deliberation times correlated highly between the four tasks (Supplementary Table 3) and significantly increased with task complexity ($\chi^2(3) = 79.82$, $p < .001$, $MG < PDW < PDL < DD$; $\chi^2(3) = 87.13$, $p < .001$, $MG < PDW = PDL < DD$) for placebo and alcohol conditions. Comparisons between infusion conditions showed that deliberation times were significantly lower during alcohol than placebo infusion for the tasks involving possible losses (PDL $r = 0.354$; MG $r = 0.2$), while no differences were found for DD and PDG (Table 2, Figure 2).

Discussion

Our data contribute to the understanding of acute alcohol effects on impulsive choice in adolescents. The main findings were as follows: On the group level, alcohol intoxication did not significantly influence choice behaviour in the domains of time preference and risk-taking for monetary wins, losses, or mixed prospects. This contrasts with our hypothesis of generally increased impulsive choice behaviour during alcohol administration. At the same time, additional explorative analyses provide preliminary evidence for the existence of divergent individual

choice tendencies associated with the sensitivity towards alcohol as well as prior experiences with alcohol consumption. Further, when intoxicated, the time to make a decision was substantially shortened solely for choices involving negative prospects.

Table 1. Descriptive statistics of alcohol consumption, related problems and personality constructs.

<i>N</i> = 54	Variable	α	Mean (SD)
Alcohol-related measures	Age of 1st drink (years)	–	14.2 (1.32)
	PEth (ng/mL)	–	125 (194)
	AUDIT score ^a	.79	7.66 (4.42)
	OCDS-G total score ^a	.61	3.33 (2.88)
	AEQ-G total score ^a	.88	29.4 (4.81)
	DMQ-Enhancement ^a	.79	12.2 (4.60)
	DMQ-Social ^a	.79	13.7 (4.44)
	DMQ-Conformity ^a	.78	5.69 (1.44)
	DMQ-Coping ^a	.65	6.78 (2.26)
SURPS	FH (%)	–	27.8
	Anxiety sensitivity ^a	.59	10.7 (1.88)
	Hopelessness ^a	.88	12.3 (3.04)
	Impulsivity ^a	.48	9.44 (1.76)
BIS-15	Sensation seeking ^a	.64	15.9 (2.92)
	Attention ^a	.47	8.67 (1.72)
	Motor ^a	.69	9.67 (2.12)
	Non-planning ^a	.73	10.9 (2.61)
	Sum score ^a	.76	29.2 (4.85)

α : Cronbach's α as a measure of internal consistency; BIS-15: Barratt Impulsiveness Scale; SURPS: Substance Use Risk Profile Scale with subscales. Age of first drink was derived from the standardized Composite International Diagnostic Interview. Peth: phosphatidylethanol; OCDS-G: Obsessive Compulsive Drinking Scale; AEQ-G: Alcohol Expectancy Questionnaire; DMQ-R: Drinking Motives Questionnaire. FH: at least one first- or second-degree relative identified as a problem drinker using the Family Tree Questionnaire (derived from Mann et al., 1985).
^aSums of scale items.

Prior studies examining alcohol effects on discounting in humans tested older samples using oral alcohol administration procedures. The present study therefore was the first to investigate the effects of intravenous alcohol administration on temporal and probability discounting in adolescents. Nevertheless, we failed to find a general effect of alcohol, just like the majority of the previous studies (Bidwell et al., 2013; Reynolds et al., 2006; Richards et al., 1999). In spite of evidence for both trait- and state-like features (Bickel et al., 2015; Friedel et al., 2016), overall little evidence exists that DD is sensitive to drug treatments in humans. This stands in contrast to a range of studies in laboratory animals (De Wit and Mitchell, 2010). Procedural differences in time framing (seconds versus days-years), the use of secondary rewards, and the lack of directly experienced delay and reward within sessions may underlie a lack of sensitivity in the human assessment. Such considerations however remain elusive for the less intensively studied domain of probability discounting as well as for loss aversion, for which acute alcohol effects have not been assessed at all prior to this report.

Yet, individual differences in impulsive choice behaviour when intoxicated may also explain prior research findings. Evidence suggests meaningful individual variation in tests of impulsive action during acute alcohol administration, for example, predicting ad libitum drinking (Gan et al., 2014; Weafer and Fillmore, 2008). Thus, individual differences in impulsive choice when intoxicated might predict real-life drinking and the longitudinal development of consumption patterns from adolescence into young adulthood. This could be a promising line of future investigations.

Even at identical BACs, individuals experience acute effects of alcohol in variable degrees. Such individual differences in the response to alcohol have been found using self-report (subjective) (e.g. Schuckit, 1980), physiological (e.g. Brunelle et al., 2007; Schuckit, 1985) and behavioural measures (Beirness, 1987; Quinn et al., 2013). In our study, more impulsive choice (higher DD) in the alcohol condition was associated with higher subjective

Table 2. Summary statistics of experimental parameters: subjectively experienced alcohol effects and value-based decision-making.

	Variable	Placebo		Alcohol (80 mg%)		Statistical difference	
		<i>M</i>	SD	<i>M</i>	SD	value	<i>p</i>
Subjective measures	sedation	23.5	24.2	48.9	21.7	–5.32 ^c	<.001
	stimulation	14.1	17.7	52.6	25.3	–6.05 ^c	<.001
	negative effects	0.53	2.94	9.23	15.4	–4.37 ^c	<.001
	feeling drunk	9.8	14.2	53.4	24.4	–6.39 ^c	<.001
DD	log(<i>k</i>)	–4.12	2.69	–4.24	2.61	0.64 ^b	.522
	deliberation time ^a	2.16	0.69	2.16	0.81	0.22 ^c	.826
PDG	log(<i>k</i>)	0.27	0.96	0.43	0.97	–1.18 ^b	.248
	deliberation time ^a	1.37	0.37	1.38	0.42	–0.42 ^c	.670
PDL	log(<i>k</i>)	–0.067	0.85	–0.101	1.17	0.19 ^b	.846
	deliberation time ^a	1.55	0.68	1.35	0.45	2.07 ^c	.038
MG	log(λ)	0.5	0.73	0.49	0.74	0.02 ^b	.978
	deliberation time ^a	1.23	0.29	1.11	0.20	3.68 ^c	<.001

N: sample size, *M*: mean, SD: standard deviation, DD: delay discounting, PDG: probability discounting for gains, PDL: probability discounting for losses, MG: mixed gambles, log(*k*)/log(λ): behavioural parameter; for correlations between behavioural parameters see Supplementary Table 2.

^ameasured in seconds.

^b*t* (paired *t*-test).

^c*Z* (Wilcoxon matched pairs rank sum test).

Figure 2. Alcohol effects on impulsive choice and task performance. (a) No significant group effect is found comparing alcohol to placebo condition in any of the four behavioural parameters of impulsive choice. (b) The time to make decision was significantly shortened for PDL and MG. Presented are means and standard errors of the mean (error bars), DD: delay discounting, PDG: probability discounting for gains, PDL: probability discounting for losses and MG: mixed gambles; Significance codes: *** $p < .001$, * $p < .05$.

Figure 3. Correlations between estimated parameters in the placebo and alcohol condition across all subjects for task separately. Each point represents the proportions in both states for a single subject. The black line represents the least square fit. The grey line represents the unity line (i.e. when the values in the two states are equal). DD: delay discounting, PDG: probability discounting for gains, PDL: probability discounting for losses and MG: mixed gambles.

stimulant-like effects, which is in line with similar observations for alcohol-related behavioural disinhibition (Quinn and Fromme, 2016). Overall, stronger subjectively experienced alcohol effects being associated with more impulsive choice tendencies in DD, PDL and PDG further support the notion proposed by Quinn and Fromme (2016) that subjective alcohol sensitivity may have implications for the immediate behavioural consequences of intoxication.

Participants in the present study were not naïve to alcohol, but had varying amounts of pre-exposure. Through repeated experience with a drug, individuals develop expectancies or beliefs about its effects (Brown et al., 1987). Thus, the individual history

of alcohol consumption could have been associated with baseline (i.e. placebo) levels of impulsive choice and/or the effect of alcohol on choice behaviour. Indeed, participants who were more risk averse (i.e. less impulsive) in PDG during alcohol administration, showed increased rates of baseline alcohol consumption and alcohol-related questionnaire scores. This might indicate that participants with more frequent lifestyle consumption were more impulsive regarding probabilistic gains in general but got more cautious when intoxicated due to having more experience with the effects of alcohol. Expectations of alcohol-induced impairment can produce adaptive responses to alcohol that serve to reduce the degree of behavioural impairment (Fillmore and

Blackburn, 2002). As such, the presented findings may result from interactions between the expected and actual pharmacological effects of alcohol.

We found that participants made quicker decisions when intoxicated during PDL and MG. Response times in behavioural tasks have been typically found to increase with acute alcohol consumption (Baylor et al., 1989; King, 1975) resulting in the 'global-slowness' hypothesis (e.g. Maylor and Rabbitt, 1993). However, decreasing response times during acute alcohol administration have also been reported (McManus et al., 1983; Tiplady et al., 2001) providing support for a more complex behavioural pharmacology of alcohol (Ryan et al., 1996). Since changes in response time may be more attributable to cognitive than motor impairment (Hernández et al., 2006), they are thought to reflect attention. Thus, our findings of shorter deliberation times when making decisions regarding monetary losses may be attributable to an attentional bias. This seems plausible as alcohol has been found to not only generally alter sensitivity to option outcomes (Vogel-Sprott, 1967) but also to produce a greater (motivational) sensitivity to gains and a reduced (motivational) sensitivity to risky losses (Lane et al., 2004).

Besides several strengths, including the use of a balanced within-subject placebo-controlled design and multi-modal assessment of outcomes, such as measures of alcohol consumption with blood markers and questionnaires, there are also some limitations of the current study. First, behavioural choice in a laboratory setting applying a constant level of alcohol does not match real life consumption which produces fluctuating aBAC levels. Alcohol consumption increases both stimulation and sedation (Hendler et al., 2013). Initially, euphoric-like stimulant effects are reported as blood alcohol levels are rising during the ascending limb of the curve whereas the depressant-like sedative effects are more predominant during the descending portion. Differences in decision-making during these phases are likely and phasic effects have been reported for discounting for probabilistic, but not delayed, rewards (Bidwell et al., 2013). Testing occurring during a steady 0.08% aBAC thus cannot account for potential effects of the ascending and descending limbs of alcohol intoxication on participant decision-making. Similarly, it cannot be excluded that changes of perceived stimulation and sedation over time during alcohol clamping may have contributed to our null findings. This limitation arises from the study design and application of the impulsive choice tasks 100 min after reaching the target BAC, because adolescents completed other paradigms before (see Jünger et al., 2017; Obst et al., 2018). Nevertheless, intravenous alcohol clamping yields several advantages compared to real-life drinking, as it eliminates biological differences in alcohol pharmacokinetics and reduces inter-individual variation in aBAC and expectancy effects (O'Connor et al., 1998). Next, we employed a relatively new assessment of impulsive choice with an adaptive Bayesian approach because of its efficient parameter estimation (Supplementary Figure 1; Pooch et al. 2018). In addition, monetary rewards used in our study are relatively small and a higher range has been discussed to enhance conflict between choices which might lead to higher sensitivity to observed effects. Nevertheless, the same behavioural assessment was ample to replicate prior findings and illustrate significant group differences in patients with alcohol use disorder compared to controls in all tasks (Bernhardt et al., 2017). Moreover, our sample size

was not small for analysis of an alcohol challenge on the group level but might lack power for the study of inter-individual differences. Additionally, post hoc analyses were explorative and complex, raising questions about hazards of doing multiple comparisons. Nevertheless reported findings may inform future studies designed to investigate inter-individual differences in the effects of acute alcohol on decision-making. Finally, an all-male sample has the benefit of avoiding confounding gender effects, but also precludes us from generalizing these findings to adolescent women. Therefore, studies on gender differences and the relationship between acute alcohol intake and impulsive choice may be valuable, although they are complicated by gender differences in preferred blood alcohol levels and susceptibility to adverse alcohol effects (Jünger et al., 2016).

Conclusion

This study suggests that adolescents do not exhibit generally higher impulsive choice behaviour when under the influence of alcohol. Explorative findings rather advocate the importance of individual differences as a promising path for further investigations into adolescents' impulsivity and risk taking.

Authors' note

The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request.

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Supplementary material

Supplementary material for this article is available online.

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