Dimensions of impulsivity among heavy drinkers, smokers, and heavy drinking smokers: Singular and combined effects

Nathasha R. Moallem, Lara A. Ray

Department of Psychology, University of California, Los Angeles, United States

ABSTRACT

Alcohol use and cigarette smoking commonly co-occur. The role impulsivity may play as a common underlying mechanism in alcohol use and cigarette smoking is of particular interest due to emerging evidence of it being a critical component across multiple forms of addiction. Impulsivity can be examined through several constructs including, risky decision-making, response inhibition, and delay reward discounting. Impulsivity and each of these specific constructs play significant roles in the initiation of drug use, continued use despite negative consequences, and potential to relapse. This study used three behavioral tasks to measure risky decision-making (Balloon Analog Risk Task; BART), response inhibition (Stop Signal Task; SST), and delay reward discounting (Delay Discounting Task; DDT). This study advances research on impulsivity and substance use by parsing out the various components of impulsivity and examining them across three groups, heavy drinkers only (HD) (N = 107), smokers only (S) (N = 67), and heavy drinking smokers (HDS) (N = 213). Participants completed questionnaires, interviews, and neurocognitive tasks including the SST, BART, and DDT. Analyses supported an additive effect of alcohol and nicotine use in delay reward discounting. Heavy drinking smokers displayed steeper delay discounting of small rewards than did smokers only (p < .05) and heavy drinkers only (p < .05). This additive effect of smoking and drinking was not observed for risky decision-making and response inhibition, suggesting specificity of the effects for delay reward discounting. These findings indicate that those who both drink heavily and smoke cigarettes daily have increased delay reward discounting, than those in the S and HD groups. Future studies should examine these constructs longitudinally, as well as incorporate genetic and/or a neuroimaging component to these group comparisons in order to ascertain the biological bases of these behavioral findings.

© 2012 Elsevier Ltd. All rights reserved.

1. Introduction

Alcohol use and cigarette smoking commonly co-occur (Dawson, 2000). Impulsivity has been studied as a common mechanism across addictive disorders, including alcohol and nicotine use (Aragues, Jurado, Quinto, & Rubio, 2011; Doran, Cook, McChargue, & Spring, 2009; Goldstein & Volkow, 2002; Harrison, Coppola, & McKee, 2009; Jenisch & Taylor, 1999; Rubio et al., 2008). Therefore it is critical to study how impulsivity affects alcohol use and cigarette smoking, both separately and co-occurring. Impulsivity is traditionally defined as acting suddenly and without plan to satisfy an immediate desire (Kreek, Nielsen, Butelman, & LaForge, 2005) and consists of multiple facets including risky decision-making, response inhibition, and delay reward discounting (De Wit, 2009; Fernie, Cole, Goudie, & Field, 2010).

Alcohol use has been associated with risky decision-making (Fernie et al., 2010) and studies found that the Balloon Analog Risk Task (BART) differentiated between smokers and nonsmokers (Lejuez et al., 2003). There has been much evidence that heavy drinkers (e.g., Boettiger et al., 2007; Courtney et al., 2011; Field, Christiansen, Cole, & Goudie, 2007; Lejuez et al., 2010) and smokers (Baker, Johnson, & Bickel, 2003) have increased delay reward discounting, that is, impulsively choosing a smaller, immediate reward over a larger, delayed reward (Kirby, Petry, & Bickel, 1999). Response inhibition concerns an individual’s ability to inhibit his/her thoughts or behaviors and poor response inhibition has been associated with alcohol use among social drinkers (Easdon & Vogel-Sprott, 2000; Lejuez et al., 2010). Nicotine may enhance response inhibition, such that smokers were found to perform worse on a response inhibition task following abstinence (Powell, Dawkins, & Davis, 2002), though some have argued that nicotine may simply improve attention (Bekker, Bocker, Van Hunsel, van den Berg, & Kenemans, 2005).

Because impulsivity is so commonly associated with substance use, it is critical to further delineate its role on substance use co-occurrence. To that end, this study parsed out various dimensions of impulsivity and examined them across groups of heavy drinkers only (HD), smokers only (S), and heavy drinking smokers (HDS). While substance-use samples reliably differ from controls on multiple
measures of impulsivity, this study compares across substance using groups to examine unique and additive effects of impulsivity.

2. Methods

2.1. Participants

Participant groups consisted of heavy drinkers only (HD; N = 107), smokers only (S; N = 67), and heavy drinking smokers (HDS; N = 213). Inclusion criteria for all three groups were: (1) age between 21 and 55; (2) current heavy drinker and/or daily smoker. Exclusion criteria for all three groups were: (1) serious medical illness within the past 6 months; (2) use of illicit drugs (other than marijuana) in the previous 60 days (verified by toxicology screen); (3) lifetime psychotic disorders, bipolar disorders, or major depression with suicidal ideation. Inclusion criteria for both smoking groups were: (1) daily smoker (≥ 10 cigarettes/day); (2) ≤ 3 months of smoking abstinence in the past year. Those in the heavy drinking groups must be currently drinking heavily according to the National Institute on Alcohol Abuse and Alcoholism (NIAAA): > 14 drinks per week (>7 for women) or ≥ 5 drinks (≥4 for women) per occasion at least once per month over the past year. Non-smokers in the HD group were defined as having smoked < 50 cigarettes in their lifetime. Non-heavy drinkers in the S group fell below the NIAAA guidelines for heavy drinking.

2.2. Procedures and measures

The three groups were recruited from the Los Angeles community as part of three large-scale studies involving either alcohol administration or pharmacotherapy. Following a phone screen, participants were invited for an in-person visit in which they signed the consent form and completed the following study assessments.

2.2.1. The Fagerstrom Test of Nicotine Dependence and 30-day Timeline Follow Back

The Fagerstrom Test of Nicotine Dependence (FTND; Heatherton, Kozloski, Frecker, & Fagerstrom, 1991) and the 30-day Timeline Follow Back (TLFB; Sobell & Sobell, 1980) were used to assess nicotine dependence and quantity and frequency of alcohol and cigarette use over the past 30-days.

2.2.2. The Delay Discounting Task

The Delay Discounting Task (DDT; Kirby et al., 1999) required participants to choose between 27 hypothetical monetary situations in which they must decide between smaller immediate rewards and larger delayed rewards. Patterns of choice were analyzed to estimate hyperbolic discounting functions (Mazur, 1987). k scores were derived and indexed according to the preference for smaller immediate rewards relative to larger delayed rewards.

2.2.3. The Stop Signal Task

In the Stop Signal Task (SST), participants were shown a series of arrows and were instructed to respond as quickly and accurately as possible to the corresponding arrow; however if presented with an audible beep, they were instructed to withhold their response to that arrow (as described in Moallem & Ray, 2012). An average Stop Signal Delay (mSSD) indexed the time delay the participant needed in order to inhibit their response 50% of the time. The Stop-Signal Reaction Time (SSRT), a sensitive measure of response inhibition, was calculated by subtracting mSSD from median go reaction time (MGRT; Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003).

2.2.4. The Balloon Analog Risk Task

In the Balloon Analog Risk Task (BART) (Lejuez et al., 2002), participants were instructed to pump up a balloon to earn money. If the balloon explodes, the participant loses the earnings for that trial and a new balloon will be presented. This study utilized two versions of the BART in which the risk of balloon explosion followed a uniform or normal distribution. To allow comparisons between groups, a z-score was calculated for each variable to standardize across versions (Lejuez, Aklin, Bornova, & Moocla, 2005). Since the inclusion of pumps made in trials that resulted in explosions may negatively bias the mean, adjusted mean pumps (AMP) were used as a variable of risky-decision making (Lejuez et al., 2002) and the variance in adjusted mean pumps was also examined (VAMP).

2.3. Statistical analysis

A series of ANCOVAs were conducted in SAS Statistical Software, using the general linear model (PROC GLM). An initial omnibus test was conducted across the three groups (HD, S, and HDS) and significant results were followed-up with two-group planned comparisons. Final models also include significant demographic covariates. The dependent variables of delay reward discounting as measured by the DDT include: (a) k small, (b) k medium, and (c) k large scores. The dependent variables of response inhibition as captured by the SST include: (a) MGRT, (b) mSSD, and (c) SSRT. The dependent variables of risky decision-making indexed by the BART were (a) AMP and (b) VAMP.

3. Results

3.1. Power analysis and baseline comparisons

Statistical power was estimated using the program GPower 3.1.2. Power was estimated for the required ANOVA omnibus test with 3 groups and a total sample size of 387 participants and at an alpha level of p<.05. Results indicated that the sample size was adequately powered (≥.80) to detect a small to medium effect size (f = .16 or larger). Demographic characteristics and group differences are presented in Table 1.

3.2. Analyses of study aims

3.2.1. Delay discounting

Initial omnibus tests revealed a significant main effect of group on k value, small (F(2, 370) = 10.64, p < .01), after controlling for gender and ethnicity (ps > .01), as they differed across groups (see Fig. 1). Follow-up comparisons revealed a significant difference between HD and HDS on k small (F(1, 304) = 21.82, p < .01), such that HDS displayed steeper discounting for small rewards, as compared to HD. Likewise, there was a significant difference between S and HDS on k small (F(1, 264) = 3.84, p < .05), such that HDS displayed steeper discounting for small rewards, as compared to S. There was no main effect of group on k medium (F(2, 368) = 1.30, p = .27) but a trend level effect on k large (F(2, 379) = 2.21, p = .09). Follow-up comparison revealed a significant difference between HD and HDS on k large (F(1, 309) = 5.17, p < .05), after controlling for education (p < .01), such that HDS displayed steeper discounting for large rewards, as compared to HD.

3.2.2. Response inhibition

Initial omnibus tests revealed no significant main effect of group on SSRT (F(2, 376) = 1.42, p = .24) or MGRT (F(2, 379) = 2.43, p = .09), after controlling for age (p < .01). However, there was a significant difference between HDS and S on MGRT (F(1, 273) = 4.66, p < .05), such that HDS had slower MGRT as compared to S. Omnibus tests revealed no significant main effect of group on mSSD (F(2, 380) = 1.14, p = .32). None of the remaining planned comparisons reached statistical significance (ps > .10).

3.2.3. Risky decision-making

Omnibus tests revealed no significant main effect of group on AMP (F(2, 375) = 1.32, p = .27), after controlling for gender (p < .05) and
Delay reward discounting may be a predictor of future substance use for small rewards. It is plausible that those exhibiting higher delay reward discounting will not only be more likely to drink or smoke, but may also be more likely to use substances involved in the co-occurrence of alcohol and tobacco use.

4. Discussion

By comparing singular and combined effects of smoking and drinking on various constructs of impulsivity, this study sought to elucidate the additive effects of these substances of abuse. This is the first study to investigate the combined effects of smoking and drinking on impulsivity. Analyses revealed a significant additive effect of the two substances in delay reward discounting. HDS displayed steeper delay discounting of small rewards than did both S and HD groups. This additive effect of smoking and drinking was not observed with medium and large rewards, suggesting specificity of the effects for small rewards.

This initial evidence lends support to the idea that impairments in delay reward discounting may be a predictor of future substance use (Dom, D’Haene, Hulstijn, & Sabbe, 2006; Kollins, 2003). This is consistent with previous studies suggesting that higher delay reward discounting functions as a predisposing risk factor for alcohol and nicotine use (e.g., Anker, Zlebnik, Gliddon, & Carroll, 2009; Audrain-McGovern et al., 2004; Perry, Nelson, Anderson, Morgan, & Carroll, 2007). It is plausible that those exhibiting higher delay reward discounting will not only be more likely to drink or smoke, but may also have a higher probability of concurrent use.

The absence of significant additive effects on risky decision-making and response inhibition demonstrates the importance of comparing multiple dimensions of impulsivity. Although some studies on the acute effects of alcohol and nicotine suggest an effect of these substances on response inhibition (e.g., Abroms, Fillmore, & Marczinski, 2003), results of the present study do not support these findings. The present study measured the effects of chronic substance use on response inhibition whereas the previous literature focused on acute effects, which may explain the difference between findings. In addition, although previous research has found support for the association between risky decision-making and smoking (Lejuez et al., 2003) and alcohol use (Fernie et al., 2010) the current study suggests that singular and combined effects of alcohol and nicotine use do not differ on this dimension of impulsivity.

It is possible that these participant groups are simply not different in regard to response inhibition and risky decision-making; however an alternative explanation is that the SST and BART are not as sensitive as the DDT. Although both measures have reliably differentiated substance users from controls (e.g., Lejuez et al., 2003; Li, Luo, Yan, Bergquist, & Sinha, 2009) it may be the case that these measures cannot distinguish between substance using groups. Interestingly, functional neuroimaging studies have found associations between smoking (Galvan, Poldrack, Baker, McGlennen, & London, 2011) and drinking (Courtney et al., in review; Schuckit et al., 2012) variables and patterns of neural activation during performance of the SST in the scanner. This suggests that neuroimaging paradigms may detect meaningful variation that differentiates within-group substance use severity, while behavioral measures may not be as sensitive.

These results should be interpreted in the context of the study’s strengths and limitations. Strengths include the use of behavioral measures, which allowed for an objective measure of these facets of impulsivity. A limitation is the lack of a control group; however, the literature has convincingly established that substance users and non-users are different across these constructs of impulsivity (MacKillop et al., 2011). Future research should expand on the current study by incorporating a genetic and/or neuroimaging component to these comparisons, as biological measures may be more sensitive than behavioral assays. Further research should also examine these dimensions of impulsivity longitudinally, in order to ascertain their causal role in the co-occurrence of alcohol and tobacco use.

Role of Funding Source

This study was supported by grants from ABMRF, the Foundation for Alcohol Research, from the Pfizer Global Award for Nicotine Dependence (GRAND), the Tobacco Related Disease Research Program of California (TRDRP), and the UCLA Clinical and Translational Science Institute, National Institutes of Health (MO1-RR00865), awarded to Dr. Lara Ray.

Contributors

NRM conducted the analysis of data and produced the manuscript. Together, NRM and LAR interpreted the findings. LAR edited and approved the manuscript.
Conflict of interest

The authors declare that they have no competing financial interests or conflict of interest relating to the data included in this manuscript. The authors declare that, except for income received from their primary employer, no financial support or compensation has been received from any individual or corporate entity relevant to the data in this manuscript. There are no personal financial holdings that could be perceived as constituting a potential conflict of interest for this manuscript.

Acknowledgments

The authors would like to thank Kitty Lunny, Eliza Hart, Anida Heydari, Pauline Chin, Jessica Webb, James Ashenhurst, Kelly Courtney, Spencer Buijski, Molly Tartter, Ellen Chang Bellinda De La Torre, Zenova Williams, Ana Heydari, Taylor Rohrbaugh, Anna Sheng, Ryan Arellano, Marliana Mansour, and Ryan Tschida for their contribution to data collection and data management for this project.

This study was supported by grants from ABMRF, the Foundation for Alcohol Research, from the Pfizer Global Award for Nicotine Dependence (GRAND), the Tobacco Related Disease Research Program of California (TRDRP), and the UCLA Clinical and Translational Science Institute, National Institutes of Health (M01-RR00865), awarded to Dr. Lara Ray.

References


