Article

Impulsivity Moderates Subjective Responses to Alcohol in Alcohol-Dependent Individuals

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Abstract

Aims: Studies of social drinkers indicate that subjective response (SR) to alcohol and impulsivity are risk factors for the development of alcohol use disorder which may be related. It is unclear, however, whether there are significant relationships between SR and impulsivity among individuals with alcohol dependence. Using data from an intravenous (IV) alcohol challenge study, the present study is the first to explore the relationship between impulsivity and SR during alcohol administration among alcohol-dependent individuals.

Methods: Non-treatment-seeking, alcohol-dependent individuals (N = 42) completed the Delay Discounting Task to measure impulsivity and then completed two counterbalanced, placebo-controlled IV alcohol administration sessions, which included assessments of SR at breath alcohol concentration (BrAC) levels of 0.00, 0.02, 0.04 and 0.06 g/dl.

Results: Analyses revealed that more impulsive participants experienced higher subjective stimulation and positive mood in response to rising BrACs as compared to less impulsive individuals. More impulsive participants also experienced increased sedation over time regardless of condition (i.e. alcohol vs. saline).

Conclusion: These findings suggest that among alcohol-dependent individuals, impulsivity is positively associated with the hedonic effects of alcohol as compared to placebo. High impulsivity may characterize a subset of alcohol-dependent individuals who drink to experience the rewarding effects of alcohol.

INTRODUCTION

Subjective response (SR) to alcohol, which refers to one’s subjective experiences of the pharmacological effects of alcohol, and impulsivity, a personality trait defined by a failure to delay rewards and to control thoughts and behaviors, are two well-established predictors of problematic drinking (Dick et al., 2010; Ray et al., 2016). There are two predominant views of SR as a risk factor for alcohol use disorder (AUD). Building on the well-established familial nature of alcohol dependence (AD; Verhulst et al., 2014), Schuckit and colleagues (i.e. Schuckit, 1994b) developed the low level of response model, which posits that blunted SR represents an AUD risk factor due to the need to consume more alcohol to achieve comparable levels of subjective intoxication. Longitudinal studies have shown that family history positive individuals exhibit a low response to the intoxicating effects of alcohol as compared to family history negative participants (Schuckit, 1994a). Furthermore, independent of family history, low responders were over four times as likely to develop AD at a 10-year follow-up than high responders (Schuckit, 1994b). Other findings are more closely aligned with the differentiator model (Newlin and Thomson, 1990), which proposes a 2D model of SR where the predominant response domain differs based on the limb of intoxication. Specifically, stimulation is more prevalent along the ascending limb...
and sedation more prevalent along the descending limb. The differen-
tiator model further posits that at-risk drinkers experience enhanced
stimulatory responses along rising blood alcohol concentration (BAC)
and blunted sedation as BAC falls. Recent studies involving the differ-
tiator model have focused primarily on drinking history and SR
domain with less emphasis on limb of intoxication. King et al. (2016)
reported that, among heavy social drinkers, individuals who experi-
enced high stimulation and low sedation at peak breath alcohol con-
centration (BrAC) reported more binge drinking and AUD symptoms
at both 2- and 6-year follow-ups and that these response patterns
were fairly stable.

The vast majority of SR research thus far has focused on social
drinkers, leaving a relative void in literature pertaining to SR in indi-
viduals with AD. Drobes et al. (2004) found that participants with
AD experienced greater subjective stimulation than social drinkers
in the hour following consumption of a gender-adjusted dose of
alcohol (0.40 g/kg for males and 0.34 g/kg for females). King et al.
(2016) examined SR in participants with low (M = 0.9, SD = 0.2),
intermediate (M = 3.2, SD = 0.2) and high (M = 6.4, SD = 0.6)
AUD symptoms at baseline and at 5-6 years follow-up. Individuals
with moderate and high AUD symptoms experienced greater sub-
jective stimulation, wanting and liking during a follow-up session,
as compared to the low AUD group. Taken together, these studies sug-
gest a positive correlation between alcohol use severity and reward-
motivated drinking. By contrast, Bujarski et al. (2015) reported greater
stimulation in light-to-moderate drinkers and in those with AD than in
heavy drinkers, and no group differences in sedation. However, stimu-
lation was more strongly associated with alcohol craving among
non-dependent heavy drinkers as compared to subjects with AD. We
propose that examining associations between impulsivity and SR may
clarify these differences.

The Delay Discounting Task (DDT; Kirby et al., 1999) is a well-
validated behavioral measure of impulsive choice, which assesses an
individual's preference for smaller immediate rewards versus later
delayed rewards as a function of the length of the delay and the
magnitude of the monetary difference between rewards. Studies
comparing the DDT to various self-report measures of impulsivity
have observed null to moderate (r's = 0.39-0.50) associations
(Madden et al., 1997; Vuchinich and Simpson, 1998; Lane et al.,
2003), suggesting that impulsivity may not be a single trait, but
rather several inter-related traits that may confer independent or
joint risk on AUD. To that end, as compared to other behavioral
and self-report measures, the DDT appears to be a stronger correlate
of alcohol and substance use disorders (Courtney et al., 2012).

Studies of the relationship between impulsivity and SR in social
drinkers have produced mixed results. An early investigation of
drinkers without AD found that self-reported impulsivity was not
associated with self-reported energy/vigor and friendliness during
alcohol administration (Nagoshi et al., 1991). However, impulsivity
was positively correlated with sedation in males along falling BAC.
Erblich and Earleywine (2003) identified a positive relationship
between behavioral under-control (i.e. impulsive sensation seeking)
and subjective stimulation on the ascending limb of the BAC curve
in college students who did not report drinking problems. A recent
investigation of social drinkers reported a positive association
between response inhibition failure and sedation along rising BrAC
and a negative relationship between response inhibition failure and
stimulation as BrAC fell though there was no relationship between
self-reported impulsivity and SR (Shannon et al., 2011). Two studies
of undergraduate social drinkers did not observe associations
between self-reported impulsivity and SR (Rose and Grunsell, 2008;
Magrys et al., 2013). Considered together, the aforementioned stud-
ies suggest that relationships between impulsivity and SR in social
drinkers are poorly understood and contingent upon study design
and measurement tools.

Leeman et al. (2014) were the first to examine impulsivity and
SR in social drinkers at precise alcohol concentrations in a highly
controlled environment. They achieved this by using an intravenous
(IV) alcohol clamping method, which reduces some sources of vari-
ability in absorption rate and facilitates the maintenance of target
BrACs enabling experimenters to better control alcohol exposure to
the brain. The IV alcohol clamping method enabled Leeman et al.
(2014) to test SR at low (0.04 g/dl) and high (0.10 g/dl) BrACs while
minimizing sources of pharmacokinetic variability and responses to
alcohol-related cues. More impulsive individuals were more respon-
sive to the stimulant and less responsive to the sedative effects of
both alcohol doses. No study to date has explored the relationship
between impulsivity and SR in individuals with AD.

Studies have highlighted the influence of the A118G single-
nucleotide polymorphism (SNP) of the mu-opioid receptor (OPRM1)
on SR in heavy drinking participants. While evidence linking this SNP
to AD is mixed (Arias et al., 2006), several studies found that G-allele
 carriers experience the rewarding subjective effects of alcohol more
strongly than do A-allele homozygotes (Ray and Hutchison, 2007;
Ray et al., 2013). This SR pattern was replicated in individuals with
AD in the primary analysis of the present data, which oversampled
for G-allele carriers. Given the prospective genotyping in this data set,
OPRM1 genotype was accounted for in the analyses for the present
study.

The current investigation is a secondary analysis of an alcohol
challenge study that aims to expand upon studies of SR and impul-
sivity. Specifically, this study is the first to investigate the moderating
effects of impulsivity on SR in participants with AD. To achieve this
aim, a well-validated behavioral indicator of impulsivity, the DDT
and a placebo-controlled, ascending limb, IV alcohol administration
paradigm were employed in a well-characterized sample of particip-
ants with AD. We hypothesized that impulsivity would be posi-
tively associated with the stimulant and hedonic subjective effects
of alcohol and negatively associated with sedative effects along rising
BrAC.

MATERIALS AND METHODS

Participants

Participants were non-treatment-seeking alcohol-dependent indivi-
duals (N = 42) recruited from the Los Angeles area. Inclusion cri-
tera were the following: (a) between 21 and 65 years old, (b) met
DSM-IV criteria for current (i.e. past month) AD and (c) consumed
a minimum of 48 standard drinks in the month preceding the
screening visit. Participants were excluded if they: (a) were currently
receiving treatment for alcohol problems or planned on seeking
treatment in the near future, (b) abstained from alcohol for 21 days
prior to screening, (c) met diagnostic criteria for lifetime bipolar or
any psychotic disorder and (d) reported clinically significant with-
drawal symptoms as indicated by a Clinical Institute Withdrawal
Assessment for Alcohol-Revised (CIWA-AR) score ≥10.

Procedures

During the screening visit participants completed interviews, ques-
tionnaires and the DDT to assess for inclusion and exclusion criteria
and to measure individual differences. Participants also submitted a
saliva sample for genetic analysis. Of the 295 participants who completed the screening visit, 43 individuals were randomized for participation in experimental sessions. Prospective genotyping for the A118G SNP of the OPRM1 gene accounted for most of the attrition from screening and allele frequencies were in agreement with Hardy-Weinberg equilibrium ($\chi^2(1) = 0.425, P = 0.514$) (Ray et al., 2013). A physical examination was conducted to ensure that participants were healthy enough to receive alcohol.

Individual differences measures

Measures collected at screening and used to determine eligibility included: (a) the 30-day Timeline Follow back interview (Sobell and Sobell, 1981) to assess frequency and quantity of alcohol consumption, (b) the Structured Clinical Interview for DSM-IV (First et al., 1995) to assess alcohol and substance use, mood and psychotic disorders and (c) the CIWA-AR (Sullivan et al., 1989) which measured the presence and severity of alcohol withdrawal symptoms. The Alcohol Dependence Scale (ADS; Skinner and Allen, 1982), Drinkers Inventory of Consequences (DRI-2; Miller et al., 1995) and Penn Alcohol Craving Scale (PACS; Flannery et al., 1999) were also administered on the screening visit to assess severity of alcohol-related problems. The first question of the Smoking History Questionnaire (Brown et al., 2002) was administered to assess smoking status. Participants reported whether they had smoked <50 cigarettes, between 50 and 100 cigarettes and >100 cigarettes in their lifetime. Responses were collapsed so that only individuals who reported smoking >100 cigarettes were considered regular smokers. Table 1 provides a description of sample demographics, smoking status and alcohol use variables.

The DDT was administered during the screening visit to assess trait impulsivity (Kirby et al., 1999). Participants were presented with 27 hypothetical scenarios containing a smaller immediate reward (Option A) and a delayed larger reward (Option B) and asked to respond to rewards as if they were real. For example, in one scenario, participants were asked if they preferred $27 today or $50 in 21 days. Reward values were derived from a previously validated measure of discounting (Kirby et al., 1999) and ranged from $11 to $85 with delays from 7 to 186 days. From these hypothetical choice data, hyperbolic discounting rate $k$-scores were calculated to reflect the degree to which participants were willing to delay rewards with higher $k$-scores representing greater delay discounting rate.

Alcohol administration procedures and measures

IV alcohol administration was used to control for pharmacokinetic variability between individuals and to control for alcohol-related cues (Ramchandani et al., 2009). Participants completed two infusion sessions, one alcohol infusion and one saline control infusion in a single-blinded, randomized, counterbalanced and crossover design. There was a 1- to 2-week washout period between infusions with an average separation of 10.6 days. Participants registered a BrAC of 0.00 prior to experimental testing and regular smokers were permitted to smoke before alcohol administration. A nomogram which accounts for participants’ sex and body mass was used to intravenously administer alcohol (Ray and Hutchison, 2007). Participants received a 5% EtOH solution at a rate of 0.126-ml/min × weight, in kilograms, for females and 0.166-ml/min × weight for males. Participants were titrated to BrACs of 0.02, 0.04 and 0.06 g/dl whereupon infusion rates were reduced by half to maintain stable BrAC while testing. Testing lasted an average of 5 min at each target BrAC during which time participants completed alcohol administration measures. Participants were released from the laboratory when their BrAC dropped to 0.02 g/dl (or a BrAC = 0.00 g/dl if driving). Nursing supervision was available during the entire protocol and at discharge.

Measures administered at target BrACs included (a) The Bihapscopic Alcohol Effects Scale (BAES), a 14-item questionnaire scored on an 11-point Likert scale that captures participants’ subjective experiences of stimulation and sedation (Martin et al., 1994). (b) The Profile of Mood States (POMS) is a 40-item questionnaire that characterizes mood across four dimensions: negative mood, positive mood, tension and vigor (McNair et al., 1971). Both BAES and POMS have been validated in alcohol challenge studies (Ray et al., 2013).

Data analysis plan

Repeated-measures analysis of covariance (ANCOVA) was conducted using SPSS version 23. ANCOVA models consisted of Impulsivity (DDT $k$-score), a continuous between-subjects covariate, Condition, a two-level within-subjects factor (alcohol vs. saline, coded 0 and 1), Time, a four-level within-subjects factor (0 at baseline, 1 at BrAC = 0.02 g/dl or 18 min, 2 at BrAC = 0.04 g/dl or 43 min and 3 at BrAC = 0.06 g/dl or 75 min) and their interactions. Dependent variables included Stimulation and Sedation as measured by the BAES and Positive Mood, Negative Mood, Tension and Vigor as measured by the POMS, each tested in a separate model. OPRM1 genotype was included as a covariate because it was associated with SR in the parent study (Ray et al., 2013). Consideration was given to age, sex, monthly drinks (total drinks in the past 30 days), ethnicity (Caucasian vs. non-Caucasian), smoking status (smoker vs. non-smoker) as potential control variables, but their respective inclusion in the final model did not influence the results presented in Table 2. Post hoc analysis was conducted by testing Impulsivity × Time interactions at each level of Condition as well as Impulsivity × Condition interactions at each level of Time.

RESULTS

Demographics

Participants were predominantly males (73.8%) between ages 21 and 51 ($M = 29.14, SD = 9.48$). The sample was also ethnically

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**Table 1. Sample demographics**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD)</th>
</tr>
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<tbody>
<tr>
<td>Sex (% female)</td>
<td>26.2%</td>
</tr>
<tr>
<td>Race (% Caucasian)</td>
<td>69.0%</td>
</tr>
<tr>
<td>Age</td>
<td>29.1 (9.5)</td>
</tr>
<tr>
<td>Education</td>
<td>14.6 (3.4)</td>
</tr>
<tr>
<td>Smoking status (% smokers)*</td>
<td>61.9%</td>
</tr>
<tr>
<td>DSM-IV AD symptoms</td>
<td>4.8 (1.5)</td>
</tr>
<tr>
<td>ADS score</td>
<td>42.4 (5.5)</td>
</tr>
<tr>
<td>DrInC-2 R score</td>
<td>51.1 (24.8)</td>
</tr>
<tr>
<td>PACS score</td>
<td>20.1 (6.2)</td>
</tr>
<tr>
<td>CIWA-AR score</td>
<td>3.6 (4.4)</td>
</tr>
<tr>
<td>Binge drinking days (past 30 days)$\dagger$</td>
<td>14.9 (7.8)</td>
</tr>
<tr>
<td>Number of drinking days (past 30 days)</td>
<td>19.3 (7.6)</td>
</tr>
<tr>
<td>Drinks per drinking day (past 30 days)</td>
<td>7.1 (2.9)</td>
</tr>
</tbody>
</table>

*Participants were considered smokers if they reported smoking 100+ lifetime cigarettes.

$\dagger$ A binge drinking day was considered 5+ drinks in a day for males and 4+ drinks in a day for females.
Impulsivity and subjective stimulation and sedation

First, we tested whether impulsivity moderated the stimulating effects of alcohol over and above the effects of OPRM1 genotype. We observed a significant Impulsivity × Condition × Time interaction on subjective Stimulation (F(3, 114) = 2.99, P = 0.03) such that impulsive individuals reported greater alcohol-induced stimulation across rising BrAC, as compared to less impulsive individuals and as compared to placebo (Fig. 1). We also found a significant Impulsivity × Time interaction (F(3, 114) = 3.13, P = 0.03) wherein more impulsive participants experienced greater Stimulation over the course of the infusion. There was no main effect of Impulsivity on Stimulation (F(1, 38) = 0.19, P = 0.69).

We also examined whether impulsivity moderated the sedative effects of alcohol while controlling for OPRM1 genotype. We identified a significant Impulsivity × Time interaction (F(3, 114) = 7.59, P = 0.0001) such that highly impulsive individuals experienced increased Sedation across Condition over Time (Fig. 2). There was not a significant main effect of Impulsivity on subjective Sedation (F(1, 38) = 0.69, P = 0.41) nor were the Impulsivity × Condition (F(3, 114) = 0.02, P = 0.89) or Impulsivity × Condition × Time (F(3, 114) = 0.17, P = 0.92) interactions significant.

Table 2. F-statistics and P-values for main and interaction effects

<table>
<thead>
<tr>
<th></th>
<th>Stimulation</th>
<th>Sedation</th>
<th>Positive Mood</th>
<th>Negative Mood</th>
<th>Tension</th>
<th>Vigor</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F</td>
<td>P</td>
<td>F</td>
<td>P</td>
<td>F</td>
<td>P</td>
</tr>
<tr>
<td>OPRM1 genotype</td>
<td>3.50</td>
<td>0.07</td>
<td>0.32</td>
<td>0.57</td>
<td>1.17</td>
<td>0.29</td>
</tr>
<tr>
<td>Time</td>
<td>0.38</td>
<td>0.77</td>
<td>1.50</td>
<td>0.22</td>
<td>0.28</td>
<td>0.84</td>
</tr>
<tr>
<td>Condition</td>
<td>3.70</td>
<td>0.08</td>
<td>4.25</td>
<td>0.05</td>
<td>4.22</td>
<td>0.05</td>
</tr>
<tr>
<td>Impulsivity</td>
<td>0.19</td>
<td>0.69</td>
<td>0.69</td>
<td>0.41</td>
<td>1.17</td>
<td>0.29</td>
</tr>
<tr>
<td>Time × Condition</td>
<td>0.53</td>
<td>0.66</td>
<td>0.27</td>
<td>0.85</td>
<td>0.58</td>
<td>0.63</td>
</tr>
<tr>
<td>Time × Impulsivity</td>
<td>3.13</td>
<td>0.03</td>
<td>7.59</td>
<td>&lt;0.01</td>
<td>1.38</td>
<td>0.25</td>
</tr>
<tr>
<td>Condition × Impulsivity</td>
<td>0.07</td>
<td>0.79</td>
<td>0.02</td>
<td>0.89</td>
<td>0.76</td>
<td>0.39</td>
</tr>
<tr>
<td>Time × Condition × Impulsivity</td>
<td>2.99</td>
<td>0.03</td>
<td>0.17</td>
<td>0.92</td>
<td>2.64</td>
<td>0.05</td>
</tr>
</tbody>
</table>

Impulsivity and mood

Over and above the effects of OPRM1 genotype, we found a significant Impulsivity × Condition × Time interaction on Positive Mood (F(3, 114) = 2.64, P = 0.05), suggesting that more impulsive participants in the alcohol condition experienced greater Positive Mood along rising BrAC, as compared to less impulsive individuals and as compared to the placebo condition (Fig. 3). There was no main effect of Impulsivity on Positive Mood (F(1, 38) = 1.17, P = 0.29). Impulsivity × Condition (F(3, 114) = 0.76, P = 0.39) and Impulsivity × Time (F(3, 114) = 1.38, P = 0.25) interactions were not significant.

There were no significant main or interaction effects of Impulsivity on Negative Mood, Tension or Vigor accounting for OPRM1 genotype. Notably, the Impulsivity × Condition × Time interaction on Vigor (F(3, 114) = 2.31, P = 0.08) approached significance wherein more impulsive participants reported marginally greater Vigor across rising BrAC in the alcohol condition, as compared to the placebo. See Table 2 for all main effects and interactions.

Post hoc analysis

Post hoc testing was conducted by testing Impulsivity × Condition interactions at each level of Time and Impulsivity × Time interactions at each level of Condition, each controlling for OPRM1 genotype. Impulsivity × Condition interactions were not significant.
**DISCUSSION**

This secondary data analysis of an existing alcohol administration study in non-treatment-seeking individuals with AD (Ray et al., 2013) sought to elucidate the relationship between impulsivity and SR using highly controlled IV alcohol administration and a behavioral measure of impulsivity, the DDT. Results revealed that more impulsive participants experienced more subjective stimulation during alcohol infusion than less impulsive participants and as compared to the placebo condition. Additionally, greater impulsivity was associated with increases in positive mood over the course of rising BrAC and as compared to the placebo. Our findings extend the work of Leeman et al. (2014) by using a behavioral measure of impulsivity to demonstrate the association between impulsivity and SR in a sample of participants with AD. Furthermore, our results indicate that highly impulsive individuals with AD experience substantial increases in stimulation and hedonic reward during alcohol administration, suggesting that their alcohol consumption produces rewarding effects. By comparison, low impulsive individuals with AD may be less sensitive to the hedonic effects of alcohol, suggesting that alternative factors may influence the drinking behavior of these individuals.

Whereas Leeman et al. (2014) administered a self-report measure of impulsivity, the Barratt Impulsiveness Scale (Patton et al., 1995), we used the DDT, a behavioral measure of impulsivity that may be less vulnerable to self-report bias. Given the mixed evidence regarding associations between self-report and behavioral measures of impulsivity, the high level of consistency between these data and those reported by Leeman et al. (2014) suggests that the association between impulsivity and SR is generalizable and robust to protocol changes (i.e. from self-report to behavioral measure of impulsivity).
Interestingly, our findings revealed that in highly impulsive individuals, sedation increased over time regardless of condition (alcohol vs. placebo). Since alcohol, as compared to saline, did not cause significant increases in sedation it is possible that participants simply became more sedated as the experiment proceeded, and that this waning was more evidenced among more impulsive individuals. Additionally, this study did not assess sedation on the descending limb of BrAC when these effects are thought to be preponderant (Newlin and Thomson, 1990). Finally, we measured only the negative elements of sedation (i.e. down, slow thoughts, sluggish) even though some individuals may experience sedation as a positive state. The recently developed Subjective Effects of Alcohol Scale (Morean et al., 2013) has the potential to elucidate the relationship between impulsivity and sedation by capturing positively (i.e. calm, relaxed, secure) and negatively (i.e. dizzy, wobbly, woozy) valenced sedation items, which were not available in the present study.

Study strengths include the IV alcohol procedure which allowed us to control for individual differences in alcohol metabolism that can affect SR. Oral alcohol administration paradigms are unable to account for inter-individual differences in physiological functions such as gastric emptying, hepatic function and blood volume, that create significant variability in the time course of BrAC (Ramchandani et al., 2009). Friel et al. (1995) administered alcohol orally (0.51 g/kg for males and 0.43 g/kg for females) to social drinkers and recorded BrAC at 8 time points post-drink. They reported between-subject BrAC variations ranging from 39% at 14 min to 14% at 125 min. Our IV alcohol infusion method ensures that participants reach equivalent BrACs at each time point. Moreover, IV as compared to oral alcohol administration is not tied to ordinary alcohol cues—taste, smell, touch and appearance—which may increase craving and impact mood (Jones et al., 2013).

There are several limitations of the present study. While the IV alcohol technique offers a significant degree of experimental control, it lacks external validity as compared to oral alcohol administration studies. In this sample, OPRM1 G-allele carriers experienced increased alcohol-induced stimulation, positive mood and vigor in comparison to A-allele homozygotes (Ray et al., 2013). Therefore, we implemented statistical controls to account for potential biasing of our results.

CONCLUSION

This study is the first to investigate the relationship between impulsivity and SR in participants with AD. Our findings indicate that among individuals with AD, higher impulsivity was associated with greater stimulant and positive hedonic effects of alcohol along rising BrAC. While it is well established that high-risk social drinkers have impulsive tendencies (Verdejo-Garcia et al., 2008), our findings suggest that high impulsivity may characterize individuals with AD who drink for positive reinforcement. Future studies should explore individual differences that may explain the drinking behavior of comparatively less impulsive individuals with AD and seek to clarify associations between impulsivity and the sedative effects of alcohol across the progression of alcohol use and alcohol-related problems.

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CONFLICT OF INTEREST STATEMENT

None of the authors have any other conflicts of interest to disclose.

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