2010

Effects of alcohol on self-control: a psychophysiological approach

Sarah Lust
University of Missouri - Columbia

Bruce D. Bartholow
University of Missouri - Columbia

Effects of Alcohol on Self-Control: A Psychophysiological Approach

Sarah Lust & Bruce D. Bartholow

Department of Psychological Sciences, University of Missouri, Columbia, MO

This research was supported by grants R21-AA017282 (PI: Bartholow) and T32-AA013526 (PI: K. Sher) from the National Institute on Alcohol Abuse and Alcoholism.

Contact email (S. Lust): salxr6@mizzou.edu

Social Cognitive Neuroscience Lab

Bruce D. Bartholow, Lab Director

INTRODUCTION

- Considerable research indicates that alcohol intoxication can adversely affect behavior by impairing higher cognitive functions (e.g., Giancola, 2000), which can lead to increased risk-taking (Leigh, 1999).

- The primary purpose of this project was to assess the degree to which individual differences in interference control (i.e. the ability to focus attention and ignore distracting peripheral information) are associated with self-reported risk-taking behaviors.

- Another goal of this work was to test the extent to which the link between interference control and self-reported risk-taking is moderated by alcohol intoxication.

- Neural measures hypothesized to be associated with distinct aspects of cognitive control can predict decision making on a trial by trial basis (West & Alain, 1999; 2000).

- Two components of the event-related brain potential (ERP) thought to reflect evaluative and regulative aspects of cognitive control, the error-related negativity (ERN) and the negative slow wave (NSW), respectively, were measured in this study to permit tests of hypotheses concerning potential links between online regulation of cognitive control and self-reported risk-taking.

METHOD

- **Participants:** 96 social drinkers (48 men), 21-35 years old, with no major medical problems.

- **EEG data:** Recorded from 64 scalp locations (tin electrodes), sampled at 1000 Hz and filtered online at .05-40Hz (average mastoid reference derived offline). Impedance was kept below 8 KΩ. Response-locked ERP epochs of -400-600 ms were derived offline, and average waveforms were created according to participant and stimulus conditions.

- **Beverage administration and assessment of intoxication:** Ps were randomly assigned to Alcohol (0.80 g/kg ETOH), Placebo (0.04 g/kg ETOH) or Control beverage groups. Ps in the Alcohol and Placebo groups were told their beverage contained alcohol; Control Ps knew their drink did not.
- Subjective intoxication and breath alcohol concentration (BrAC) were assessed (in the alcohol and placebo groups) at baseline and at several intervals throughout the experiment.

Manipulation Checks: BrAC and Subjective Intoxication

- For Alcohol subjects, BrAC = .072 at task start and rose throughout the task, $F(3, 93) = 2.90$, $p = .03$.
- Alcohol participants reported greater subjective intoxication throughout the experiment than placebo participants, $F(1, 63) = 63.22$, $p < .001$. However, both groups reported a similar pattern of changes in "feeling drunk" over the course of the task $F(4, 252) = 0.44$, $p = .78$.
- The fact that placebo participants estimated having consumed at least 2 standard drinks indicates that our placebo manipulation was effective.

Eriksen Flanker Task (adapted from Ridderinkhof et al., 2002)

- Ps categorized the direction of the central arrow (the target) via button press.
- Arrow arrays appeared for 100 ms, followed by ITI varying between 1100-1500ms.
- Ps received feedback during 7 practice blocks (196 trials total) to ensure similar performance across beverage groups
- 10 experimental blocks were completed (no performance feedback ; 800 trials total)

Compatible	Incompatible
←←←←←	←←→←←

Overt Error Detection

- Following their response on each trial, Ps indicated their assessment of their performance on that trial by pressing 1 of 3 buttons (labeled *sure incorrect*, *don't know*, and *sure correct*).

RESULTS

1. Behavior: Did Alcohol Increase Typical Flanker Interference Effects?

- Participants responded more quickly, $F(1,89) = 37.45$, $p<.01$, and more accurately, $F(1,89) = 410.54$, $p< .01$, on compatible than on incompatible trials (**see Figure 1**).
  - There was no interaction with beverage group for reaction time.

2. There was a significant beverage group interaction for error rates such that placebo group participants made significantly fewer errors ($M = .09$) on compatible trials compared to participants in the alcohol ($M = .14$) and the control groups ($M = .13$), which did not differ from each other, $F(1, 90) = 4.36$, $p < .05$. (**see Figure 2**)
  - In contrast, follow-up analyses of errors on *incompatible* trials showed no significant linear or quadratic effects (Fs < 3.05, ps > .07).

3. Did Alcohol Impair Post-error Adjustment?

- The analysis showed a significant Beverage group x Previous trial interaction, $F(2, 85) = 3.62$, $p < .05$ (**see Figure 3**).
  - Ps in the alcohol and control groups showed no evidence of post-error adjustment (i.e., smaller interference effects on post-error vs. post-correct trials). However, participants in the placebo group showed significant post-error adjustment, seen as a smaller compatibility effect on post-error trials ($M = 41$ ms) than on post-correct trials ($M = 57.7$ ms), $F(1, 85) = 4.87$, $p < .05$.

4. ERN Amplitude: Was the ERN Reduced by Alcohol?

- Beverage group significantly affected ERN amplitude (**see Figure 4**), $F(2, 85) = 7.41$, $p < .001$ (see also Bartholow et al., 2009; Ridderinkhof et al., 2002).

5. NSW Amplitude: Was the NSW affected by Alcohol?

- Results showed a significant main effect of Compatibility $F(1, 90) = 10.34$, $p < .01$ such that NSW amplitudes were greater (more negative) for incompatible ($M = -2.6\mu V$) than compatible ($M = -1.8\ \mu V$) trials (**see Figure 5**). However, this effect was not moderated by beverage group.

6. Risk Score and the Compatibility Effect.

- The hypothesized relationship between flanker task behavioral performance (compatibility effects in accuracy) and risk score, including potential interactions with beverage group, was tested using separate general linear models including Beverage group and flanker performance variables as factors and executive function and quantity/frequency of alcohol use as covariates. Results showed:
  - Significant main effect of Beverage group, $F(2, 78) = 3.92$, $p < .05$
  - Significant Beverage group x Compatibility effect interaction, $F(2, 78) = 5.54$, $p < .01$. Separate partial correlations (controlling for covariates) between CE_acc and risk score for each beverage group (**see Figure 6**)showed that the relationship between the compatibility effect and risk score was significant only in the placebo condition.

7. Relations between Neural Indices of Self-Control and Risk-Taking.

- A set of regression equations tested whether neural indices of self-regulatory control and self-reports of risk-taking were associated, and whether any such relationships would be moderated by alcohol. Unfortunately, in each model tested none of the main effects or cross-product terms were significant predictors of risk scores (all βs < .13, ps > .23).

Figure 4. Beverage effects on the ERN

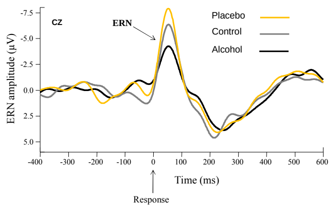


Figure 5. Beverage effects on the NSW

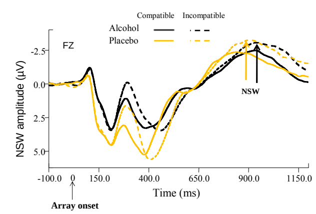


Figure 1. Compatibility effect in RT.

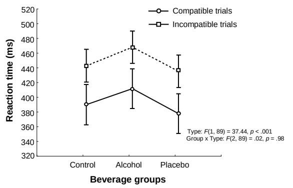


Figure 2. Compatibility effect in error rate

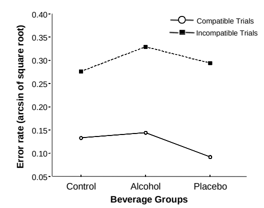


Figure 3. Post-error adjustment

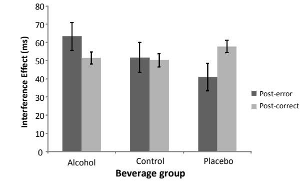
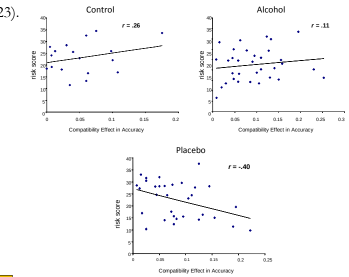


Figure 6. Correlations between Risk Score and CE_acc.



CONCLUSION

- Alcohol decreased the ERN and impaired post-error performance adjustment (see also Bartholow et al., 2009; Ridderinkhof et al., 2002). However, no relationship was found between neural indices of self-regulatory control and self-reports of risk-taking.

- An association between the size of the compatibility effect in accuracy and risk score emerged for Placebo participants. This finding suggests a potential compensatory effect among placebo subjects. That is, people that tend to take more risks while intoxicated may have a strong expectancy about alcohol's disinhibiting effects, and thus might try hard to compensate for anticipated impairment they expect alcohol to have on their cognitive functioning and task performance. This finding suggests that alcohol expectancy may actually lead to performance improvement for individuals that are prone to taking risks. Further analyses controlling for alcohol expectancies in this group could help elucidate this effect.

REFERENCES

Bartholow, B. D., Henry, E. A., Lust, S.A., & Saults, J. S. (2009). Alcohol impairs error processing in anterior cingulate cortex via reduced negative affect, not impaired error detection. *Manuscript in revision*.
Bartholow, B. D., Pearson, M. A., Dickter, C. L., Sher, K. J., Fabiani, M., et al. (2005). Strategic control and medial frontal negativity: Beyond errors and response conflict. *Psychophysiology,* 42, 33-42.
Yueng, N., Botvinick, M., Cohen, J. (2004). The neural basis of error detection: conflict monitoring and the error –related negativity. *Psychological Review,* 111, 931-959.
Ridderinkhof, K. R., et al. (2002). Alcohol consumption impairs detection of performance errors in mediofrontal cortex. *Science, 298,* 2209-2211.