Neurophysiological Markers of Problematic Drinking: Insights from a Twin Study
Preliminary Results

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BACKGROUND

Reduced P3 elicited during cognitive tasks and **enhanced P3 reactivity to alcohol cues** have been shown to be present among alcoholics and heavy drinkers.

- Reduced P3 is not a consequence of heavy drinking and alcohol abuse but instead a neurophysiological maker of risk for heavy drinking and AUDs (Begleiter et al., 1984; 1990; Carlson et al., 2002).
- Enhanced P3 to alcohol cues has been found among heavy drinkers or individuals with AUD. Substantial research demonstrates that enhanced P3 to alcohol cues predicts alcohol use prospectively (Bartholow et al., 2007; 2010).

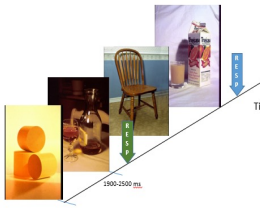
Aims:

- To decompose the genetic and environmental sources of variation of both P3 phenotypes (i.e., Go-P3 and Alcohol-P3 reactivity).
- To examine whether the P3 amplitude elicited during cognitive tasks (Go P3) and P3 reactivity to alcohol cues (Alcohol P3) interact in a way that places individuals at increased risk for heavy/problematic drinking.

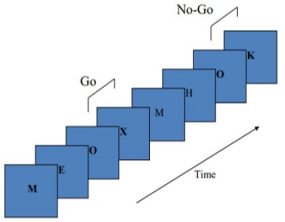
METHOD

The final sample included data from 173 MZ and DZ twins who completed either (or both) the Alcohol Image Task (AIT) and the Cognitive Performance Task (CPT) while EEG was recorded via ERPs.

Alcohol Image Task (AIT)



Cognitive Performance Task (CPT)



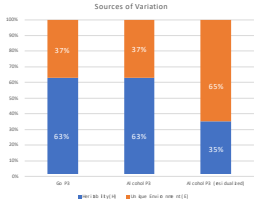
Participants completed Missouri Assessment of Genetics Interview for Children at age 12 through 17, and Semi-Structured Assessment for the Genetics of Alcoholism at age 18:

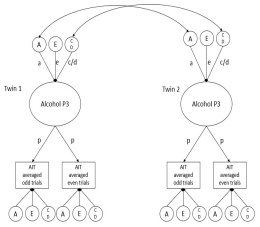
- **Age at drinking onset:** "How old were you when you had your very first full drink of alcohol?"
- **Maximum number of alcoholic drinks in 24 hours:** "What is the largest number of drinks you have ever had in any 24-hour period at any time in your life?"
- **Number of drinking occasions over time:** "In the past year, have you taken a drink on…" 1 = one or two occasions to 6 = forty or more occasions.

RESULTS

Biometric Genetic Analyses

Due to some incomplete twin pairs, models were estimated using a full-information ML estimator (MLR) in MPLUS.

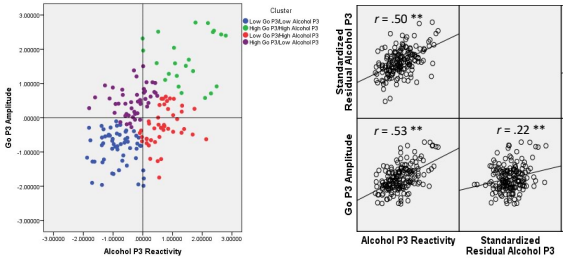




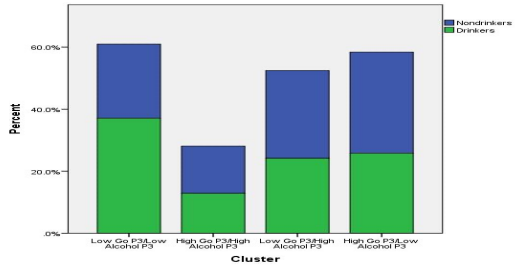
Alcohol P3 (residualized) was derived by regressing P3 elicited by nonalcohol cues on P3 elicited by alcohol cues and then saving the residuals. It is expected residualized measures to have lower heritability due to reduced reliability. A biometric model with latent variable (see diagram) can be informative in determining the extent to which alcohol-specific P3 is causally influenced by non-shared environmental influences.

Go-P3 ×Alcohol-P3.

K-means clustering forcing 4 clusters with centers (-2,-2) (2,2) (2,-2) (-2,2)



No statistically significant differences were found (all *p*s > .05).



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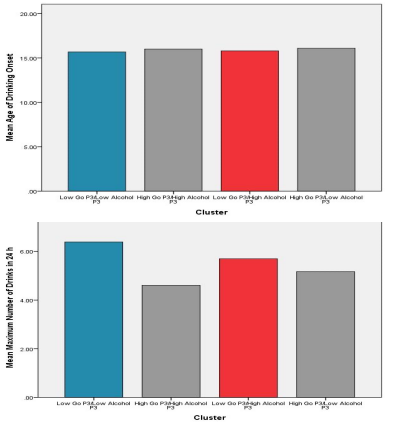

CONCLUSIONS

- Results suggested that both the P3 elicited by Go trials and the P3 reactivity to alcohol-related pictures are mostly genetically determined.
- Importantly, the effect of unique environmental experiences on both P3 phenotypes is difficult to quantify: The variance due to unique environmental experiences also contains residual variance.
- Even though both P3 phenotypes are likely generated by the same neurobiological structures, the positive correlation between these two neurophysiological markers is difficult to interpret.
- We found no evidence for interactive effects of the Go-P3 and Alcohol-P3 reactivity on drinking status, age of drinking onset, maximum number of drinks in 24 hours,
- Low Go-P3/High Alcohol-P3 cluster did not show the steeper increase in the number of drinking occasions over time from 12 to 17 years-old (not reported here due to space constrains).
- In sum, these data provided no evidence that individuals at the Low Go-P3/High Alcohol-P3 cluster are at higher risk for heavy/problematic drinking.

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