Genetic and environmental risks predicting patterns of alcohol use and misuse from adolescence through early adulthood

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Genetic and Environmental Risks Predicting Patterns of Alcohol Use and Misuse from Adolescence through Early Adulthood

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Study Background

Alcohol and substance use are primary public health concerns in the U.S. Initiation of alcohol use often begins during adolescence, and earlier use can be a predictor of later abuse and dependence. Research suggests that both genetic and environmental factors influence alcohol use and misuse. Genes in the dopamine system have been associated with a variety of impulsivity issues. DAT1 and MAOA specifically have been linked to alcohol use in some populations, and recent research suggests that cumulative effects of multiple polymorphisms across genes may be larger than the effects of individual genes.

In addition to genetic factors, environmental influences have been linked with the initiation and growth of alcohol use and misuse. Alcohol related social norms, or the normative behaviors of important others, have been linked to adolescent alcohol use and aspects of social control, such as parental knowledge and monitoring, have been associated with delayed alcohol use. Finally, stressful life experiences have been associated with increases in both alcohol use and misuse during adolescence.

Recent innovations in this field suggest that genetic and environmental factors are likely to have an interactive effect – with genetic risks for alcohol use and misuse in an individual being exacerbated by environmental risk factors. This study sought to assess the combined role of environmental and dopamine-related genetic correlates of early alcohol use and misuse by delineating the unique and interactive effects of four proposed contributors to youth alcohol use and misuse: genetic risks; social norms from parents and peers; social control from parents and schools; and stressful life experiences.

Data Source and Sample

Data were drawn from Add Health, a longitudinal survey of a nationally representative sample of adolescents in the U.S. Youth were interviewed four times starting in mid-adolescence (M age = 16) and continuing through early adulthood (M age = 29). Parents, peers, and school administrators were interviewed in wave 1, and genetic data were collected via cheek swabs from youth at Wave 4. Our analytic sample included 13,451 youth: 54% White, 19% African American, 15% Hispanic, 6% Asian, and 6% Multiracial/other.

Measures

Genetic Risks: Two dopamine polymorphisms were tested: DAT1 40 bp VNTR (low-activity alleles 2R, 3R, and 5R coded as risk) and MAOA 30 bp VNTR (10R alleles coded as risk for males; 9R alleles coded as risk for females). Risk alleles were summed to create genetic risk scores (GRS).

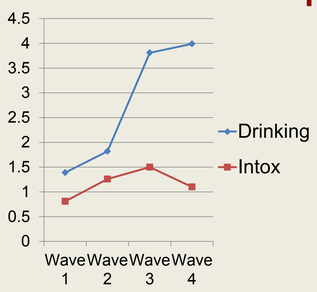
Environmental Risks/Protections (Wave 1):

Theory	Construct	Reporter & Measure
Social Norms	Parent Drinking	Parent report of days drank in prior month
	Friend Drinking	Youth report of number of 3 closest friends who drank alcohol >= 1/mo
Social Control	Parental Knowledge	Parent report of knowledge of adolescent's friends, activities (α = .67)
	School Punishment	Administrator report of school punishment for drinking on campus (α = .75)
Stress	Internal SLE	Youth report of internal stressful life events in prior year
	External SLE	Youth and parent report of external stressful life events in prior year

Youth Alcohol Use/Misuse: At each of the four survey waves, youth reported the number of days they (1) drank alcohol and (2) became intoxicated in the past month, coded as count variables.

Analytic Technique

Multilevel growth models examined trajectories of male and female drinking and intoxication from waves 1 through 4, using a negative binomial sampling distribution and assessing both linear and curvilinear growth. Models assessed main effects as well as interactions between genetic risks and each of the environmental variables (GxE). Missing data in the analytic sample were multiply imputed.



Results

For both sexes, growth in the frequency of drinking and intoxication was steepest over the first three waves (mid adolescence through transition to early adulthood), followed by slower growth in drinking and a decline in intoxication in the mid-late 20's.

Results (cont'd)

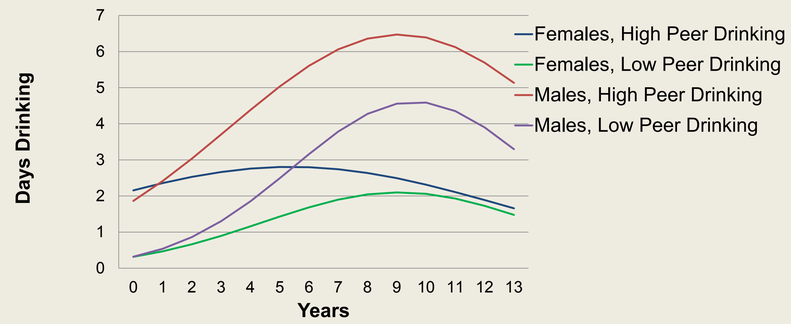
Genetic Risks:

- No significant main effects of the genetic risk score (GRS)
- No significant effects of gene X environment interactions (GxE)

Environmental Risks/Protections:

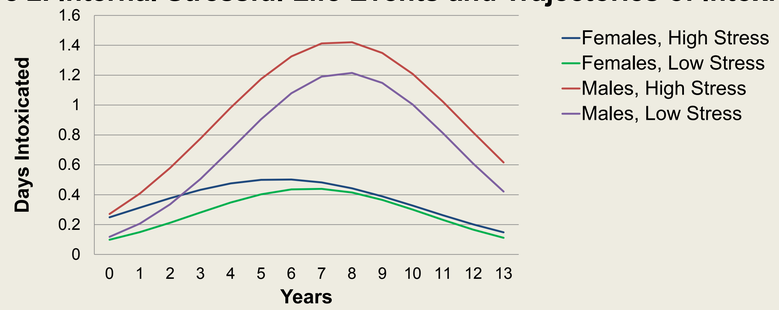
- Results found consistent associations with social norms and stressful life events, but few links between social control and youth drinking/intoxication
- Adolescents with parents who used more alcohol showed higher alcohol use but not intoxication
- Adolescents with more close friends who used alcohol showed higher alcohol use and intoxication, with differences converging over time for females (see Figure 1)

Figure 1. Peer Drinking and Trajectories of Alcohol Use



- Males with higher parental knowledge showed lower levels of drinking
- Adolescents who experienced more internal and external stressful life events (SLEs) showed higher levels of drinking and intoxication, with convergence over time, particularly for external SLEs (see Figure 2)

Figure 2. Internal Stressful Life Events and Trajectories of Intoxication



Study Conclusions & Implications

The lack of associations between dopamine signaling genes and alcohol use, as well as the lack of GxEs, concurs with other recent research failing to replicate genetic effects. This lack of effects may suggest that GxE interactions are less common than previously suggested or may reflect measurement limitations in AH data. Environmental contexts, particularly social norms and life stressors, were significant predictors of alcohol use and misuse, providing potential targets for prevention and intervention efforts.