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Genetic and Psychosocial Predictors of Alcohol Use Trajectories Among Disaster-Exposed Adolescents

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Abstract

Background and Objectives—Adolescent alcohol misuse is associated with numerous long-term adverse outcomes, so we examined predictors of alcohol use among disaster-exposed adolescents, a group at-risk for alcohol misuse.

Methods—The current study ($n=332$) examined severity of tornado-related exposure, posttraumatic stress disorder (PTSD) symptoms, emotional support, and a genetic risk sum score (GRSS) as predictors of alcohol use trajectories.

Results—Severity of exposure interacted with the GRSS to predict both intercept (12-month follow up quantity of alcohol use) and growth rate. Emotional support also interacted with adolescent PTSD symptoms to predict intercept and growth rate.

Discussion and Conclusions—Adolescents with greater severity of disaster exposure and high genetic risk comprise a high risk group, on which efforts to prevent alcohol use should be focused. Additionally, emotional support is essential in buffering the effects of PTSD symptoms on alcohol use outcomes among adolescents.

Scientific Significance—Toward the aim of reducing adolescent alcohol misuse following disaster exposure, there is utility in inserting immediate supports (e.g., basic resources) into communities/families that have experienced significant disaster-related severity, particularly among adolescents at high levels of genetic risk for alcohol use/misuse. Additionally, prevention

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Declaration of Interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this paper.

efforts aimed at improving emotional supports for adolescents with more PTSD symptoms may reduce propensity for alcohol misuse following disaster. This information can be easily incorporated into existing web-based interventions.

INTRODUCTION

Adolescence is the developmental period during which alcohol use is typically initiated, with predictable increases in quantity and frequency during this time.¹ Problematic drinking in adolescence is associated with numerous adverse outcomes (eg, neurocognitive impairments,² unprotected sex³), and increased risk for alcohol problems in adulthood.⁴ Therefore, researchers have attempted to identify factors that increase risk, particularly among vulnerable subpopulations.

Rates of alcohol use/misuse are higher in trauma-exposed adolescents compared to the general population.^{5–6} Although work has examined psychosocial predictors of drinking among individuals exposed to interpersonal violence,⁷ studies of disaster-exposed subjects may provide opportunities to understand the post-trauma context better. Specifically, in disaster-exposed samples, individuals have experienced a common stressor within a narrow time frame, allowing for isolation of key risk factors that may increase risk for substance use. Additionally, there is a natural range in exposure, allowing one to examine the impact of trauma severity.

Severity of Trauma Exposure

Although trauma may increase risk for adolescent alcohol use/misuse, this effect is not uniform. Little work has focused on the direct impact of increased trauma severity on alcohol use; however, some find that disaster-related sequelae (eg, increased parent withdrawal) impact adolescent mental health.⁸ Others find that increased conflict and poor parent-child relationships are associated with greater propensity for adolescent substance use.⁹ Caregivers within families that experience greater disaster-related exposure (eg, losing their home, car) may be less-well positioned to obtain information about adolescents' lives, precluding them from intervening to prevent alcohol use.¹⁰

The impact of severity of disaster exposure on adolescent alcohol use may be particularly strong for individuals at greater genetic propensity to drink alcohol. Work finds that environmental influences on alcohol use outcomes are stronger at higher levels of genetic risk.¹¹ Environments that provide less opportunity for substance use may suppress genetic risk, whereas environments that allow for more substance use amplify genetic risk.¹²

Posttraumatic Stress Disorder (PTSD) Symptoms

In addition to trauma severity, posttraumatic stress disorder (PTSD) symptoms may predict alcohol use/misuse. Prior work examining PTSD and alcohol use has focused on negative reinforcement models of substance use. Specifically, individuals with PTSD are motivated to use substances to avoid aversive internal states (eg, re-experiencing symptoms^{13,14}).

This “self-medication” model of substance use may be more salient for individuals without access to alternative, active coping tools. Specifically, the motivation to avoid distressing

symptoms by using substances may be weaker for individuals who perceive that they have alternative methods to cope (eg, if they have supportive others on whom they can depend). Among individuals exposed to interpersonal trauma, those with higher social support reported higher quality of life¹⁵ and/or lower risk for alcohol misuse.¹⁶

Other Influences on Adolescent Alcohol Use

Demographic and treatment-related factors may also predict alcohol use. Specifically, older adolescents,¹⁷ males, and Caucasians (compared to Asians and African-Americans¹⁸) show higher risk for alcohol misuse. Individuals exposed to trauma-focused interventions show improved mental health outcomes.^{19–20} Finally, number of traumatic events influences mental health and substance misuse.^{21–22} Adolescent gender, age, ancestry, intervention group, and number of prior traumatic events will be used as covariates.

The Current Study

To address these gaps in the literature, we examine how severity of disaster exposure, genetic risk, PTSD symptoms, and emotional support predict alcohol use within a sample of tornado-exposed youth followed over three time points during a 1-year period. Specifically, we hypothesize that the impact of severity of tornado exposure on intercept (quantity of alcohol use) and growth rate across adolescents age 12–17 will be stronger for those at higher levels of genetic risk for alcohol misuse. We also hypothesize that more PTSD symptoms will be associated with greater alcohol use and growth rate, particularly for those who report lower levels of emotional support. We will investigate these effects over and above adolescent gender, age, ancestry, intervention group, and number of prior traumatic events.

METHOD

Procedures

Families with adolescent offspring were recruited from areas affected by the spring 2011 Alabama and Missouri tornadoes. Recruitment procedures are described in detail elsewhere.^{23,24}

One caregiver and one randomly chosen adolescent between ages 12–17 in each family were interviewed via phone at each of three time points (baseline, 4-month follow-up, 12-month follow-up). During the baseline interview, families were asked whether they were interested in participating in another study component, involving adolescent provision of genetic data. Ruggiero et al.²⁴ reports additional information on genetic data collection.

Participants

Participants in this overall sample were 2,000 adolescents (mean age =14.5 years, SD =1.7; 51% females; 62.5% Caucasian) and their caregivers.

Procedures for Obtaining Genotypic Data

Of the 2,000 families involved in the larger study, 780 adolescents chose to provide genetic data. For these 780 interested in providing genotypic data, Oragene kits (DNA Genotek,

Ontario, Canada) were used for collection of saliva. Of the 780 participants who provided genetic data, 763 were successfully genotyped.

Current Study Participants

In addition to requiring successful genotyping, we also required adolescents to have provided genetic data that passed quality control standards, to have been present at the time of the tornado, and to have been interviewed at all three time points in order to be included in the current study ($n=332$). Comparisons of adolescents included in analyses to those excluded from analyses were performed (Table 1).

Measures

Covariates

Adolescent Sex: Girls (49.4%) were coded 0 and boys were coded 1.

Adolescent Age: Age was examined at baseline ($M=14.48$, $SD=1.75$).

Intervention Group—Parents and adolescents who were interviewed at baseline were asked to participate in activities within an online web portal. Following the baseline interview, families were randomly assigned to one of three web-based conditions: (1) *An evidence-based behaviorally focused intervention* (BI), which involved parents and adolescents receiving psychoeducation about the benefit of behaviorally-focused activities to alleviate PTSD symptoms; (2) *The behavioral intervention +parent self-help* (BI +self-help), which was identical to the behaviorally-focused intervention condition except that parents were provided an targeting behaviorally-focused skills to improve their own mental health symptoms; and (3) *Assessment only*, which involved parents and adolescents being asked questions about their mental health. Two dummy coded variables comparing these three groups (31.9% [$n=106$] Assessment, 31.3% [$n=104$] BI, 36.7% [$n=122$] BI +self-help) were used as covariates. There were no significant differences ($p<.05$) between these three groups on adolescent age or gender. However, there were differences in terms of race. Specifically, there were significantly more Caucasians in the BI group, in comparison to the BI +self-help or Assessment only groups.

Number of Prior Traumatic Events—Adolescents reported on whether they had experienced a number of different traumatic events (eg, physical assault, sexual assault, accident). On average, adolescents had experienced .98 traumatic event prior to the tornado ($SD=1.11$).

Ancestry Informative Proportions—Ancestral informative markers throughout the genome were analyzed as a quantitative means of classifying ancestry to account for population stratification, which can result in spurious findings due to differences in allele frequency across racial/ethnic groups. Specifically, a Principal Components analysis in the larger sample²⁴ was conducted on 9,827 single nucleotide polymorphisms (SNPs) known to differentiate Caucasians, Hispanics, African-Americans, and Asian-Americans. Borrowing this method from prior literature,²⁵ the two components with eigenvalues of 1 or higher were retained. These components differentiate Caucasians and African-Americans (current

subsample racial composition: 69.1% Caucasian, 26.8% African–American, 4% Other). These scores are referred to as Ancestry Principal Components (PC) 1 and 2.

Predictors

Adolescent PTSD Symptoms: The PTSD module from the National Survey on Adolescents²⁶ assessed DSM-IV PTSD symptoms since the tornado. On average adolescents reported 2.67 (SD =3.42) symptoms.

Severity of Tornado-Related Exposure: Parents reported whether they had experienced a range of nine events during the tornado (eg, physically injured, were concerned about the safety of others (range: 0–9). The average number of endorsed items within this subsample was 2.29 (SD =1.88).

Perceived Emotional Support: Five items from the Social Support Scale for Children²⁷ assessed perceptions of emotional support from family and peers. A mean of available items (with ranges of 0–2) was created to indicate emotional support ($M=1.13$, $SD=.41$).

Number of Drinks in the Past Month—Adolescents reported the number of alcoholic drinks consumed in the past month. The mean number of past month drinks ranged from .40 at baseline to .95 at follow-up. These rates are comparable to rates of adolescents living in Alabama and Missouri.^{28,29} However, these variables were highly skewed and kurtotic, and even after square root transformations, had skew and kurtosis values outside the acceptable range ($+/-2$, $+/-7$). Therefore, to protect against biased parameter estimates, bootstrapped standard errors will be used, as recommended.³⁰

Genetic Risk Sum Score (GRSS)—In the absence of large-scale genome-wide approaches (GWAS), candidate gene methods informed by knowledge of neurobiological risk for alcohol use disorders (AUDs) offer one promising approach.³¹ In this method, the use of a GRSS combines risk alleles from SNPs found in GWAS from prior literature into one polygenic score. Including SNPs from two or more GWAS resulted in 697 SNPs, six of which were available in the current dataset. An additional 18 overlapped with those in high linkage disequilibrium (LD; ie, $r^2 \geq 0.7$) with those from prior literature. After omitting those in high LD with one another, the remaining 21 SNPs (in Table 2) were coded using an additive model³² and comprised the GRSS.

Data Analytic Plan—All continuous predictors and covariates were centered prior to conducting analyses. Longitudinal growth model analyses were conducted using MPlus Version 7.³³ Missing data on endogenous variables were estimated as a function of the observed exogenous variables under the missingness at random assumption.³⁴

Severity, PTSD symptoms, GRSS, and emotional support, and interactions between severity and GRSS, and PTSD symptoms and emotional support, were predictors. All other interactions among these constructs were also tested, but trimmed when non-significant ($p < .05$). Sex, age, ancestry proportions, and experimental condition were main effect covariates.

Power Analyses—Prior research was consulted to determine that the current study ($n=332$) likely has sufficient power to detect medium and large effects. Specifically, within a longitudinal growth curve modeling framework, 150 people are needed to detect a medium effect ($d=.5$) and between 950 and 100 people are required to detect a small effect ($d=.2$) using three time points of data.³⁵

RESULTS

Zero-Order Correlations

Table 3 provides the zero-order Pearson (between two continuous variables), tetrachoric (two dichotomous variables), and biserial (dichotomous and continuous variables) correlations.

Main Study Analyses: Longitudinal Growth Model

Model-Building Approach to Determine Best Fitting Model—Using a model-building approach, no growth ($\chi^2=222.872$; 6 dfs) and linear growth models ($\chi^2=3.332$; 2 dfs) with month as the underlying metric were estimated, with the linear model fitting better than the no growth. Next, we attempted to estimate a full quadratic model (ie with random effects). However, this model would not produce a solution. Therefore, a quadratic model with no random effects was estimated ($\chi^2=3.132$; 1 dfs). This model did not fit better than the linear model. Thus, adolescent alcohol use increased in a linear fashion over the 1 year study period.

Main Model Results—Goodness of fit was determined by comparing results from the model with standards for acceptability.³⁶ Results suggest good fit to the data: RMSEA = .067, SRMR = .023 and CFI = .900. Results are presented in Table 4.

Some covariates were associated ($p<.01$) with intercept (ie, alcohol use at the 12-month follow-up). Specifically, older adolescents endorsed drinking .209 more drinks a month than younger adolescents ($B=.209$, $p<.001$). Additionally, males reported drinking .050 more drinks a month at the 12-month follow-up than females ($B=.050$, $p<.01$).

The interactions between genetic risk and severity of exposure and emotional support and PTSD symptoms were significant. The interaction between genetic risk and severity of exposure ($B=.347$, $p<.01$) suggests that for a one unit increase in severity, individuals one standard deviation above the mean on genetic risk increased their drinking by .279 drinks per month ($b=.279$, $p<.01$). Additionally, for a one unit increase in severity, individuals at the mean on genetic risk increased their drinking by .145 drinks per month ($b=.145$, $p<.05$). However, this relation was non-significant at one standard deviation below the mean on genetic risk ($b=.055$, NS). In other words, the detrimental impact of severity of disaster exposure on alcohol use was only significant for those at the mean and above the mean on genetic risk.

The interaction between emotional support and PTSD symptoms ($B=-.773$, $p<.001$) suggests that for those one standard deviation below the mean on emotional support, a one unit increase in PTSD symptoms was associated with an increase of .557 drinks per month ($b=.557$, $p<.001$). For those at the mean on emotional support, a one unit increase in PTSD

symptoms was associated with an increase in .222 drinks a month ($b = .222, p < .05$). This association was non-significant for those above the mean on emotional support ($b = -.113, NS$). Thus, the effect of adolescent PTSD symptoms on alcohol use was only significant for those at and below the mean on emotional support.

Some covariates exerted significant or marginally significant ($p < .1$) effects on growth rate. Specifically, males increased in the number of drinks consumed per month by .051 more than females ($B = .051, p < .01$). Older adolescents increased in their number of drinks consumed per month by .049 drinks more than younger adolescents ($B = .049, p = .07$). Additionally, those in the assessment group increased in their number of drinks consumed per month by .125 drinks more than those in the BI +parent self-help group ($B = .125, p < .05$).

The interactions between genetic risk and severity of exposure and between emotional support and PTSD symptoms were significant. For the interaction between genetic risk and severity ($B = .280, p < .001$), for a one unit increase in severity of exposure, individuals above the mean on genetic risk increased their growth rate in the number of drinks consumed by .247 in a month ($b = .247, p < .001$). For those at the mean on genetic risk, for a one unit increase in severity of exposure, individuals increased their growth rate in the number of drinks consumed by .139 in a month ($b = .139, p < .001$). There was no relation between severity and alcohol growth rate for those below the mean on genetic risk ($b = -.007, NS$). Thus, the adverse effect of severity of disaster exposure on alcohol use was only significant for those at the mean and above the mean on genetic risk.

In terms of the interaction between emotional support and PTSD symptoms ($B = -.590, p < .001$), for those one standard deviation below the mean and at the mean on emotional support, a one unit increase in PTSD symptoms was associated with an increase in growth rate by .378 drinks per month ($b = .378, p < .001$) and increase in .154 drinks per month ($b = .154, p < .001$), respectively. There was no association between PTSD symptoms and growth rate for those above the mean on emotional support ($b = -.025, NS$). In other words, the effect of adolescent PTSD symptoms on alcohol use was only significant for those at and below the mean on perceived emotional support.

Analyses Including Only Caucasian Participants—Although we attempted to control for population stratification via use of ancestry components, it is standard procedure in the genetics literature to confirm the overall pattern of findings in the largest sub-population that is more genetically homogeneous. Therefore, a model using the same predictors described above was estimated, including only Caucasian participants ($n = 188$). The interaction between PTSD symptoms and emotional support to predict intercept became marginally significant ($B = -.357, p < .1$), as did the interaction between genetic risk and severity to predict growth rate ($B = -.483, p < .1$). However, the interactions between genetic risk and severity to predict intercept ($B = .071, NS$) and the interaction PTSD symptoms and emotional support to predict growth rate ($B = -.020, NS$) became non-significant.

DISCUSSION

Hypothesized Effects

The current study had two hypotheses. We found support for the first hypothesis; that is, greater severity of disaster exposure was associated with alcohol use across a 1-year period, but only for individuals at high and medium levels of genetic risk. This finding is consistent with work indicating that environmental influences on alcohol use are stronger at higher levels of genetic risk for alcohol use/misuse.^{11,37} However, it adds to work by finding an interaction between severity of exposure and genetic risk within a disaster-exposed adolescent sample. It may be that following high levels of tornado-related exposure and the chaos of losing needed possessions, families experience increased conflict and adolescents experience more caregiver withdrawal. This environment may provide opportunities for adolescents with the genetic propensity and interest in using alcohol to do so. However, additional work is needed to elucidate the mechanisms by which severity of disaster exposure may influence alcohol use among adolescents following disaster.

We found support for the second hypothesis. Specifically, more PTSD symptoms was associated with alcohol use for those with less emotional support. Individuals who utilize more active coping strategies (eg, talking with a close other about a distressing topic) may be less likely to use alcohol to alleviate aversive symptoms. Teaching individuals alternative, active coping strategies for managing distress reduces risk for alcohol misuse.³⁸ Therefore, teaching adolescents to confide in a close other may prevent alcohol misuse.

Non-Hypothesized Effects

Although not hypothesized, age and sex predicted alcohol use outcomes. Specifically, older adolescents and males reported higher levels of alcohol use. The effects of age are consistent with prior work finding that alcohol use increases over adolescence.³⁹ Prior work suggests that adult men are more at risk than adult women, but this same gender difference is attenuated among adolescents.⁴⁰ Therefore, the differences between male and female adolescents in this study are stronger than would have been hypothesized. It may be that male adolescents exposed to disaster are more vulnerable to alcohol misuse, compared to females. The work examining sex differences in the relation between trauma and alcohol use is mixed, with some finding that males are more vulnerable⁴¹ and some finding the opposite.⁴² More work is needed to understand under which conditions males and females may be more at risk for alcohol misuse.

Although not hypothesized, there was one significant effect of the intervention. Specifically, those who received behavioral intervention and parent self-help showed less steep increases in growth rate, compared to those who received only the assessment. Although there is little work examining the impact of web-based interventions on adolescent alcohol use, web-based interventions for alcohol misuse are effective for college students.⁴³ It is important to note that in the current study, there was no significant effect of the BI only intervention, when compared to the assessment only. It may be that this combined intervention improved parent mental health, and this change allowed for improved monitoring of adolescent behavior (thus allowing parents to limit adolescent substance use) and/or allowed parents to

more easily provide support to adolescents, reducing the need for adolescents to use alcohol as a means of coping with distress. More research is needed to understand the mechanism(s) by which interventions targeting parent and adolescent mental health following a disaster may reduce adolescent risk.

Strengths and Limitations

Despite methodological strengths, including the measurement of both genetic and environmental constructs, limitations are important to note. Most importantly, as genetic influences exert moderate effects on alcohol misuse, with each SNP likely having a small effect size, large samples are typically necessary to detect significant effects.⁴⁴ Therefore, we may have been under-powered to detect effects involving the GRSS. Additionally, using an additive coding approach assumes that SNP effects are linear, that each SNP did not interact with others (ie, no epistatic effects), and that each SNP did not moderate main effects in different ways, which some have previously found.⁴⁵ Future research should examine non-linear effects of SNPs. In addition, this sample included some adolescents and their caregivers who were randomized to receive interventions. Thus, these findings may not generalize to non-treatment samples. These findings also only pertain to disaster-exposed youth. Future research is needed to clarify predictors of alcohol use trajectories among adolescents with different types of trauma exposure (eg, interpersonal trauma exposure) and/or those without trauma exposure.

Despite these limitations, the current study advances previous literature in several ways. First, this study identifies adolescents who experience high severity of exposure and high levels of genetic risk as being a particularly high risk group, on whom programs to prevent alcohol use should be focused. Second, this study finds that emotional support buffers against the adverse effects of more PTSD symptoms on risk for alcohol use. Therefore, following a natural disaster, prevention efforts aimed at improving emotional supports for adolescents should be implemented, prioritizing adolescents with more PTSD symptoms following the disaster. This information can be easily addressed with existing web-based interventions, and these findings may be used to guide future revisions of such interventions.

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TABLE 1

Comparing those included in and excluded from study analyses

	Included		Excluded		T (p-value)	Effect size Cohen's D (.1 =small, .3 =medium)
	N	Mean (SD)	N	Mean (SD)		
Ancestry PC 1	332	-.002 (.013)	375	.001 (.14)	2.183 ($p < .05$)	.222
Ancestry PC 2	332	-.014 (.013)	375	-.011 (.013)	1.665 (NS)	.230
Adolescent age	332	14.48 (1.75)	1668	14.48 (1.71)	.936 (NS)	.000
Number of prior traumatic events	332	.98 (1.11)	1668	1.05 (1.13)	1.029 (NS)	.062
Severity of exposure	329	2.29 (1.88)	1653	2.27 (1.86)	-.352 (NS)	.011
Genetic risk	332	22.36 (2.54)	375	22.34 (2.45)	-.091 (NS)	.008
Emotional support	332	1.13 (.41)	1666	1.18 (.40)	2.175 ($p < .05$)	.123
PTSD symptoms	332	2.67 (3.42)	1667	2.43 (3.35)	-1.237 (NS)	.071
	N	% of included	N	% of excluded	Chi-Square (p-value)	Cramer's V (.1 =small, .3 =moderate)
Adolescent sex	332	49.4% female	1668	49.4% female	.000 (NS)	.000
Adolescent race	272	69.1% white, 26.8% black, 4% other	1612	73% white, 23.9% black, 3.1% other	1.848 (NS)	.034
Experimental condition	332	31.9% assessment, 31.3% behavioral intervention, 36.7% BI +self help	1666	31.1% assessment, 34.3% behavioral intervention, 34.6% BI +self help	1.177 (NS)	.024

Ancestry PC 1 and 2: ancestry principal components 1 and 2 (used to control for population stratification).

TABLE 2

SNPs included in genetic risk score

	SNP	Gene	Citation*	In dataset?	Proxy SNP used, if applicable
1	rs6088519	DYNLRB1	Wang et al. 2013 ⁴⁷	Yes	N/A
2	rs12905964	SH3GL3	Kapoor et al. 2013 ⁴⁶	Yes	N/A
3	rs2061332	LOC100379224	Kapoor et al. 2013 ⁴⁶	Yes	N/A
4	rs3134954	TNXB	Kapoor et al. 2013 ⁴⁶	Yes	N/A
5	rs3130342	TNXB	Kapoor et al. 2013 ⁴⁶	Yes	N/A
6	rs2066702	ADH1B	Gelemtier et al. 2014	Yes	N/A
7	rs4758317	LMO1	Kapoor et al. 2013 ⁴⁶	No	rs110419
8	rs2517521	LOC729792	Kapoor et al. 2013 ⁴⁶	No	rs2844670
9	rs10141811	C15orf53	Kapoor et al. 2013 ⁴⁶	No	rs12899449
10	rs262978	C15orf53	Wang et al. 2013 ⁴⁷	No	rs7165988
11	rs13231387	EXOC5	Wang et al. 2013 ⁴⁷	No	rs10150771
12	rs3096695	SLC26A3	Kapoor et al. 2013 ⁴⁶	No	rs60147601
13	rs3746321	ACSS2	Wang et al. 2013 ⁴⁷	No	rs6120757
14	rs10133300	MIRN186	Wang et al. 2013 ⁴⁷	No	rs6695355
15	rs1564104	ZNF224	Wang et al. 2013 ⁴⁷	No	rs2068061
16	rs3746319	EXOC5	Kapoor et al. 2013 ⁴⁶	No	rs3742578
17	rs6493109	PKD2L2	Kapoor et al. 2013 ⁴⁶	No	rs1880458
18	rs1032339	TRIM69	Kapoor et al. 2013 ⁴⁶	No	rs3100139
19	rs12705421	SLC7A7	Kapoor et al. 2013 ⁴⁶	No	rs1061040
20	rs12903120	ZNF222	Kapoor et al. 2013 ⁴⁶	No	rs7258517
21	rs262970	YEATS2	Kapoor et al. 2013 ⁴⁶	No	rs262993

* Gelemtier et al. (2014), Kapoor et al. ⁴⁶, and Wang et al. ⁴⁷ utilized at least two unique samples to examine relations between SNPs and phenotypes.

TABLE 3

Correlations between study variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Adolescent Sex	–												
2. Adolescent Age	–.010	–											
3. Ancestry PC 1	–.072	.000	–										
4. Ancestry PC 2	–.064	–.051	.911***	–									
5. Severity of tornado exposure	–.088	–.037	.180***	.198***	–								
6. Experimental condition: dummy code 1	.043	–.012	–.003	–.027	–.064	–							
7. Experimental condition: dummy code 2	–.034	–.028	.044	.022	.054	–.522***	–						
8. Number of Prior traumatic events	.103*	.043	.118*	.093	–.038	–.061	.050	–					
9. Genetic risk	–.074	.064	.182***	.228***	.088	–.029	.027	.093	–				
10. Adolescent PTSD symptoms	–.093	–.018	.114*	.076	.090	–.036	–.084	.394***	.094	–			
11. Emotional Support	.062	.092	–.131**	–.159**	.074	–.031	–.024	–.112*	–.108*	–.118*	–		
12. Baseline past month drinks	–.026	.189***	.007	.017	–.016	–.015	.081	.060	–.013	.082	–.111*	–	
13. 4-Month follow-Up past month drinks	–.017	.110*	.057	.076	–.047	.029	–.030	.153***	.089	.163**	–.159**	.513***	–
14. 12-month follow-up past month drinks	–.020	.080	.069	.065	.125*	.066	–.016	–.039	.054	.103*	–.133**	.339***	.161**

n = 332.* *p* < .05.** *p* < .01.*** *p* < .001.

TABLE 4

Results of linear growth model

Predictor	Intercept		Slope	
	<i>B</i>	SE	<i>B</i>	SE
Age	.209 ***	.100	.049 ($p=.07$)	.005
Sex	.050 **	.092	.051 **	.010
Ancestry PC 1	-.135	.109	-.008	.369
Ancestry PC 2	.175	.149	.050	.316
Dummy code 1: comparing assessment to behavioral intervention (BI)	.014	.600	-.011	.051
Dummy code 2: comparing assessment to BI +parent self-help	-.041	.449	-.125 *	.029
Number of prior traumatic events	-.034	.075	-.048	.018
Severity of exposure	.145 *	.111	.139 ***	.005
GRSS	.106	.717	.136	.064
PTSD symptoms	.222 *	.001	.154 ***	.002
Emotional support	.137 **	.080	.088 ***	.000
Emotional support X PTSD symptoms	-.773 ***	.061	-.590 ***	.001
GRSS X severity	.347 **	.163	.280 ***	.007

B =Standardized regression coefficient. SE =standard error. $n=332$;*
 $p<.05$,**
 $p<.01$,***
 $p<.001$.