

Adolescent health brief

# Paternal Alcoholism and Youth Substance Abuse: The Indirect Effects of Negative Affect, Conduct Problems, and Risk Taking

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## Abstract

This longitudinal study followed 200 adolescents into early adulthood to explore the potential mediating roles that hostility, sadness, conduct problems, and risk-taking play in the relationship between paternal alcoholism and substance abuse. Results indicated that paternal alcoholism predicted hostility; in turn, hostility predicted risk taking, which predicted substance abuse. © 2008 Society for Adolescent Medicine. All rights reserved.

## Keywords:

Adolescence; Substance abuse; Parental alcoholism/alcohol dependence; Children of alcoholic parents; Negative affect; Risk taking; Conduct disorder

Negative affect may play an important mediating role in the relationship between parental alcoholism and offspring (COA) substance abuse. To date, most studies that have examined the relationship between negative affect and COA substance abuse have examined a general indicator of negative affect. However, recent research suggests that certain components of negative affect may be more strongly related to substance abuse than others [1]. Therefore, one goal of this study was to comparatively examine the potential mediating roles that hostility and sadness play in the relationship between parental alcoholism and substance abuse. Because theory (e.g., the deviance proneness model of vulnerability) [2] and research also suggest that conduct problems and risk taking may play intermediate roles in the relationship between parental alcoholism and COA substance abuse, another goal was to examine whether they further mediate this relationship.

## Methods

### Participants

The participants were drawn from a larger longitudinal study (The RISK project) [3] that was designed to follow

offspring of alcohol and drug-dependent fathers over time. At Time 1, the sample included 200 15–19-year-old adolescents (68% Caucasian; 62% girls) and their biological fathers (56% alcohol dependent). The mean age of the adolescents was 16.76 (SD = 1.35). All of the adolescents and their fathers were followed up 5 years later (Time 2).

### Measures

The adolescents and their fathers were administered a psychiatric interview. The youth also completed questionnaires. Apart from the Semistructured Assessment for the Genetics of Alcoholism (SSAGA), all of the measures were given only to the adolescents and were administered at Time 1, except for the substance abuse measures (which were given at Time 2). These measures consistently have been found to have good psychometric properties.

The SSAGA [4] was administered to assess lifetime alcohol dependence, major depression, and antisocial personality disorder (ASPD) in the fathers. Paternal depression and ASPD were included as covariates because they frequently are comorbid with alcohol dependence [5]. The adolescents completed the sadness and hostility scales from the Positive and Negative Affect Schedule [6] to assess negative affect, the 51-item Risk-Taking Questionnaire [7] to measure adolescent risk taking, and the SSAGA to assess conduct problems (20 items were summed to create a scale score). The Michigan Alcoholism Screening Test (MAST)

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[8] was used to measure youth substance abuse. Youth also were asked how often (in the past 6 months) they drank enough to get drunk or high. The response scale for these variables ranged from 1 = “never” to 8 = “nearly every day or more often.”

*Procedures*

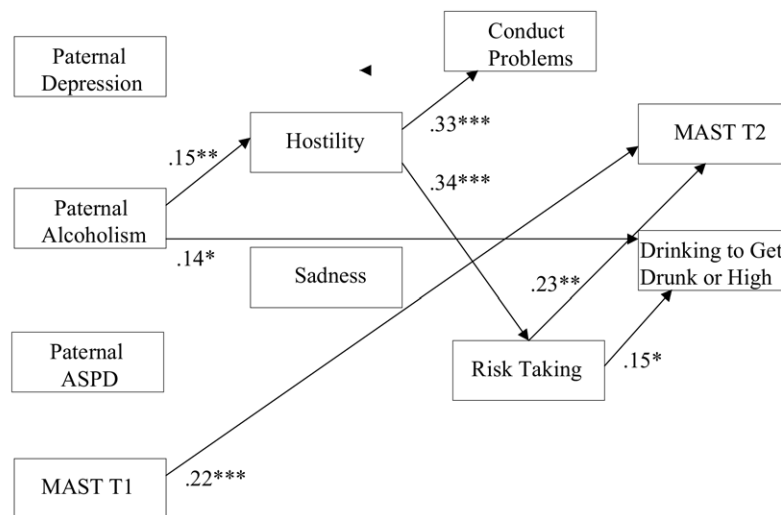
The research protocol was approved by the institutional review board at the University of Connecticut Health Center. Recruitment for the study took place through the community (e.g., YMCA/YWCA) and through alcohol/drug treatment programs and support groups. At both times of measurement, individuals who indicated that they were interested in participating were asked to call a research assistant for information and screening. If they were still interested after the phone call, they were invited to come to the university to provide informed consent and complete the measures previously discussed. All of the participants agreed to being contacted for a follow-up interview and additional testing 5 years after the initial testing. At Time 2, all participants were administered the SSAGA again. To compensate them for their time, the youth and their fathers each received a payment of \$100 at Time 1 and \$150 at Time 2. The attrition rate between Time 1 and Time 2 was 15%. Individuals who did not participate at Time 2 did not significantly differ from those who did participate at Time 2 on any of the demographic variables (e.g., gender, age) or substance use variables.

**Results**

Structural equation modeling was used to examine the underlying relations involved in the relationship between paternal alcoholism and substance abuse. In all models,

paternal depression, paternal ASPD, and youth problem drinking (the Time 1 MAST score) were included as co-variables. The first model tested a fully saturated model. This model did not fit the data well,  $\chi^2 (19) = 291.86, p = .00$ ; comparable fit index (CFI) = .88; root mean square error of approximation (RMSEA) = .27. The second model was identical to the first model, with the exception that nonsignificant direct paths were deleted. The overall fit of this model was similar to the first model,  $\chi^2 (24) = 297.11, p = .00$ ; CFI = .88; RMSEA = .24. In the third model, the nonsignificant specified paths from the previous model were deleted. This model also did not fit the data well,  $\chi^2 (35) = 307.88, p = .00$ ; CFI = .88; RMSEA = .20.

Examination of modification indices indicated that the disturbance terms between the endogenous variables should be allowed to be correlated. Therefore, a fourth model was conducted. In this model, the disturbance terms between the negative affect scales, conduct problems, and risk taking, and the substance abuse indicators were allowed to correlate with each other, respectively. The data fit this model well, ( $\chi^2 (32) = 74.35, p = .00$ ; CFI = .98; RMSEA = .08). Of note, paternal depression and ASPD did not predict any of the endogenous variables. However, paternal alcoholism significantly predicted the frequency of drinking to get drunk or high (beta = .14,  $p < .05$ ). As shown in Figure 1, significant indirect paths between paternal alcoholism and substance abuse also were observed. More specifically, paternal alcoholism significantly predicted youth hostility (beta = .15,  $p < .01$ ); hostility subsequently predicted risk taking (beta = .34,  $p < .001$ ); and risk taking, in turn, significantly predicted the MAST (beta = .23,  $p < .01$ ) and frequency of drinking to get drunk or high (beta = .15,  $p < .05$ ). Sobel tests confirmed that risk taking significantly mediated the relations between hostility and the MAST (critical



*Note.* For ease of interpretation, only significant standardized regression coefficients and their paths are shown.

Figure. Final standardized trimmed model.

Table 1  
Means and standard deviations of study variables by COA status

Scale	COAs		Non-COAs	
	Mean	SD	Mean	SD
Hostility*	13.87	4.50	12.53	3.87
Sadness	11.10	3.96	11.05	4.29
Conduct problems	19.39	8.81	17.53	7.53
Risk taking†	14.84	8.85	12.33	8.33
MAST*	3.52	6.64	1.91	3.65
Drinking to get drunk/high**	3.26	1.79	2.54	1.47

Note: n = 200.

†  $p < .10$ ; \*  $p < .05$ ; \*\*  $p < .01$ .

ratio = 2.74,  $p < .01$ ) and between hostility and frequency of drinking to get drunk or high (critical ratio = 1.97,  $p < .05$ ). Of note, sadness was not involved in any of the indirect paths between paternal alcoholism and the substance abuse indicators.

## Discussion

In this study, COAs had higher levels of hostility than non-COAs (Table 1). In addition, consistent with the literature [9,10], hostility significantly predicted risk taking; which in turn, significantly predicted substance abuse. Importantly, this study extended the literature by *simultaneously* examining these indirect relations over time as adolescents transitioned into early adulthood, a critical period for the development of substance abuse problems.

In contrast to the results for hostility, sadness did not play a significant role. This finding conflicts with the literature; however, it should be noted that many depression measures include items relating to aspects of depression other than sadness (e.g., irritability). Therefore, it may be that sadness alone is not related to substance abuse.

Although the present investigation extends the current literature, caveats should be noted. As noted, the sample assessed was a high-risk sample. Caution should be used when generalizing the results to community samples. Mothers also did not participate in the study. Therefore, possible

distinctions between paternal and maternal alcoholism could not be addressed.

Nonetheless, this study underscores the importance of longitudinally examining the underlying relations involved in the relationship between paternal alcoholism and youth substance abuse. Moreover, it highlights the importance of assessing distinct components of negative affect when examining the role that negative affect plays in the development of substance abuse problems.

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