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General and substance-specific predictors of young adult nicotine dependence, alcohol use disorder, and problem behavior: Replication in two samples*

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Abstract

Background—This paper presents two replications of a heuristic model for measuring environment in studies of gene-environment interplay in the etiology of young adult problem behaviors.

Methods—Data were drawn from two longitudinal, U.S. studies of the etiology of substance use and related behaviors: the Raising Healthy Children study (RHC; N = 1,040, 47% female) and the Minnesota Twin Family Study (MTFS; N = 1,512, 50% female). RHC included a Pacific Northwest, school-based, community sample. MTFS included twins identified from state birth records in Minnesota. Both studies included commensurate measures of general family environment and family substance-specific environments in adolescence (RHC ages 10–18; MTFS age 18), as well as young adult nicotine dependence, alcohol and illicit drug use disorders, HIV sexual risk behavior, and antisocial behavior (RHC ages 24, 25; MTFS age 25).

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Contributors

All authors were involved in study conceptualization. Drs. Samek, Hicks, and Bailey conducted the statistical analyses with consultation from the other authors. Drs. Samek and Bailey conducted literature searches, summarized prior work, and wrote the first draft of the manuscript. All authors contributed to and have approved the final manuscript.

Conflict of interest

Dr. Catalano serves on the board of Channing Bete Company, which owns portions of the Raising Healthy Children intervention mentioned here. All other authors declare that they have no conflict of interest.

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Results—Results from the two samples were highly consistent and largely supported the heuristic model proposed by Bailey et al. (2011). Adolescent general family environment, family smoking environment, and family drinking environment predicted shared variance in problem behaviors in young adulthood. Family smoking environment predicted unique variance in young adult nicotine dependence. Family drinking environment did not appear to predict unique variance in young adult alcohol use disorder.

Conclusions—Organizing environmental predictors and outcomes into general and substance-specific measures provides a useful way forward in modeling complex environments and phenotypes. Results suggest that programs aimed at preventing young adult problem behaviors should target general family environment and family smoking and drinking environments in adolescence.

Keywords

replication; problem behavior; nicotine dependence; alcohol misuse; measuring environment for GxE

1. INTRODUCTION

Tobacco dependence and alcohol use disorders carry serious health and social consequences with significant personal and economic costs. Tobacco and alcohol problems often co-occur with each other (Hawkins et al., 2002; Hughes et al., 2000; Jackson et al., 2005) and with other problem behaviors, particularly criminal or antisocial behaviors (Krueger et al., 2002; McGue et al., 2006), illicit drug use (Bailey et al., 2006; Tsuang et al., 2001; Young et al., 2006), and risky sexual behavior (Bailey et al., 2011; Iacono et al., 1999; Kanh et al., 2012). Research suggests that the development of tobacco and alcohol dependence and comorbid problems is influenced by both environmental and genetic factors (Kreek et al., 2005; Rutter et al., 2006), including environmental and genetic main effects, gene-environment interactions (GxE), and gene-environment correlations (rGE). Understanding the etiology of adult tobacco and alcohol misuse requires measuring and defining these phenotypes and modeling a complex array of interacting genetic and environmental risk and protective factors.

To reduce the complexity of analyses of gene-environment interplay in understanding problem behavior, some authors suggest simplifying strategies for measuring environment. One common strategy is to pick a single, powerful environmental exposure, like childhood abuse (Moffitt et al., 2006). A second strategy involves combining multiple environmental pathogens into an index of environmental risk (Blomeyer et al., 2006). Strengths of these methods include their parsimony in measuring environment and, given well-chosen environmental exposures, their likelihood of identifying GxE if it is present. These methods, however, do not reflect the state of knowledge about the complex, multidimensional nature of environmental influences on problem behavior. Nor do they provide clear implications for treatment or prevention.

1.1. Model to be replicated

Bailey et al. (2011) presented an alternative model for simplifying complex, multidimensional measures of the environment for studies of gene-environment interplay in the etiology of problem behavior. They advocated using a *general* versus *substance-specific* framework to conceptualize substance use phenotypes and environmental predictors. Using latent variable modeling, they partitioned variance in nicotine dependence (ND), alcohol use disorder (AUD), and illicit drug use disorder (IDUD) symptom counts; crime; and HIV sexual risk behavior (SRB) into shared and substance-specific, or unique residual, variance. Similarly, latent variables organized environmental risk in adolescence into positive general family environment (e.g., good family management, strong bonding) and substance-specific family environment (e.g., parent and sibling use, parent attitudes) factors. This framework allowed the simultaneous examination of processes related to co-occurring problem behaviors and processes uniquely related to a particular substance or behavior. It also allowed the distillation of multiple measures of complex environments into a small number of simplified yet multidimensional measures.

Bailey et al. (2011) found support for the proposed framework using data from the Seattle Social Development Project (SSDP; Hawkins et al., 2005, 2008). Specifically, good general adolescent family environment and general problem behavior in young adulthood (age 24) were negatively related. Adolescent family smoking and drinking environments predicted unique variance in young adult ND and AUD, respectively. Neither substance-specific family environment measure was related to unique variance in the other young adult problem behaviors. An extension of this model to problem behavior among SSDP participants at age 33 largely replicated the pattern of findings (Epstein et al., 2013). Although supported in both studies, the model may have been sample dependent.

1.2. A “test and replicate” model of collaboration

Reproducibility of findings is a foundation of scientific inquiry, but concerns abound regarding publication bias and the lack of systematic replication of findings in psychology and other social sciences (Bakker et al., 2012; Makel et al., 2012; Pashler and Wagenmakers, 2012). Many argue that, to address these issues, more authors should attempt to replicate new findings using well-powered studies (Bakker et al., 2012). In general, the suggested strategies for replication have focused on and are most clearly applicable to cross-sectional or short-term, pre-post experimental designs. Long-term longitudinal and etiological studies are extremely difficult to replicate closely, given their scale and complexity.

For those who wish to examine genetic as well as environmental influences on substance use using longitudinal data, options for replication are limited. In 2011, our two research groups, the Social Development Research Group at the University of Washington, and the Minnesota Center for Twin and Family Research at the University of Minnesota, began a collaborative investigation of gene-environment interplay in the etiology of substance use and general problem behavior using several datasets available at the two sites. Our collaborative model involves a test and replicate strategy, where predictive models are developed and tested in one sample, and then replicated in the remaining samples.

This paper presents two replications, in independent samples, of the Bailey et al. (2011) conceptual model of general and substance-specific environmental influences. Data are drawn from the Raising Healthy Children study (RHC) and the Minnesota Twin Family Study (MTFS).

2. METHOD

2.1. Participants

2.1.1. RHC—RHC is a longitudinal study of students attending 10 public schools in a suburban Pacific Northwest U.S. school district. It is a study of the etiology of positive and problem behaviors with a randomized test of a preventive intervention nested within it (see Brown et al., 2005; Haggerty et al., 2006 for a detailed overview). It was designed as a replication and extension of the SSDP study. The sample includes 1,040 individuals (76% of those eligible; 47% female) who were age 5 (younger cohort) or 6 (older cohort) in 1993. Annual data collection was conducted in the spring through 2011, when participants were age 24/25. The sample was 75% White, 7% Asian or Pacific Islander, 4% African American, 2% Native American, and 12% mixed race; 9% of participants were Hispanic/Latino(a). Unlike MTFS, which is a twin study (see below), RHC did not target twins or siblings. Thus, the majority of youth were unrelated; however, the sample did include 102 siblings (full, half, step siblings; see section 2.4 Analysis).

Retention rates ranged from 85% to 94% in the years from which age 10–18 family environment data were drawn. A total of 923 individuals (89% of the original sample) participated in 2010 (younger cohort age 23, older cohort age 24) and 917 (88%) participated in 2011, the years from which outcome data were drawn. Retention was not related to gender, age, or race. Participants in the intervention group (89% versus 93% of controls) or in the older cohort (86% versus 97% in the younger cohort) were less likely to be retained in young adulthood.

2.1.2. MTFS—MTFS is a study of the development and etiology of substance use and related behaviors (see Iacono et al., 1999 for a detailed overview). Twins born in the state of Minnesota between 1977 and 1984 and their parents were recruited to participate the year the twins turned 11 years old. Families were identified using birth certificates, which are publicly available in Minnesota. Approximately 90% of twin families were successfully located and contacted to determine study eligibility. Eligible participants lived within a day's drive of the lab. Exclusion criteria included any mental or physical handicap and adoption of the twins by nonrelatives (determined by a prescreen telephone interview with one parent, usually the mother). About 84% of eligible families agreed to participate. Most twins (95%) were Caucasian (2% Native American, 1% Asian/Pacific Islander, < 1% Black, Hispanic/Latino(a), Other/Mixed), matching the region from which they were sampled in the relevant birth years (Iacono et al., 1999). Out of 756 pairs of twins, 270 were dizygotic (DZ) and 486 were monozygotic (MZ). All twin pairs were same sex.

A total of 1,512 participants completed the first assessment (756 sets of twins 49.7% male; *M* age = 11.7, *SD* = .43). Retention was 87% and 88%, respectively, at the two interviews from which the present data were drawn: the second follow-up (FU2; *M* age = 18.2, *SD* = .

70) and the fourth follow-up (FU4; *M* age = 25.3, *SD* = .74). Attrition was not related to child sex or race/ethnicity, except males were less likely than females to participate at FU4.

2.2 Procedures

2.2.1. RHC—Study procedures were approved by the University of Washington Institutional Review Board. Parents gave written permission for their minor child to participate and minor children assented each year. Parents and young adults over age 18 gave consent at each survey time point. Through 2006 (younger cohort age 19, older cohort age 20), surveys were administered in person. Questions about sexual behavior were self-administered. After 2006, about two thirds of the sample completed the survey over the Internet and one third was interviewed in person. A randomized trial of this multimode survey administration indicated no differences in rates of reported sexual activity or risk behavior by survey mode (McMorris et al., 2009).

2.2.2. MTFS—Procedures were approved by the University of Minnesota Institutional Review Board. Twins and their parents provided written consent to participate at each assessment; parents provided written permission for children under the age of 18. A variety of measures were completed at each time point, including diagnostic interviews, self-report surveys, and computerized assessments. If twins could not be scheduled for an in-person follow-up visit (21% at FU2, 16% at FU4), they were interviewed by phone and completed all assessments.

2.3 Measures

Online supplementary materials[1] describe items used, scale reliabilities, ages of administration, and correspondence with the Bailey et al. (2011) study measures. Some items differed across studies; however, congeneric measures of key constructs were available. Family environment measures in RHC were assessed yearly when target youth were ages 10 to 18 and averaged across adolescence. In MTFS, family environment measures were obtained when twins were age 18.

2.3.1. General family environment—Indicators of general family environment included bonding to family members, management, conflict, and positive involvement. In RHC, these constructs were reported by youth. In MTFS, relevant items were administered to the twins and drawn from the Parent Environment Questionnaire (PEQ; Elkins et al., 1997) and the Family Adaptability and Cohesion Evaluation Scales (FACES III; Olson et al., 1985). Child ratings of mother and father from the PEQ were averaged.

2.3.2. Family drinking environment—Indicators of family drinking environment in RHC included parent drinking attitudes, parent and sibling drinking, and involvement of the youth in family member drinking (e.g., getting or opening a drink for a family member). Family drinking environment measures were parent reported except for sibling drinking, which was reported by the target youth. In MTFS, measures of family drinking environment included self-reported co-twin and parent drinking and were assessed using the Substance

[1]See Supplementary Table 1, by accessing the online version of this paper at http://dx.doi.org and by entering doi:…

Abuse Module (SAM; Robins et al., 1987), an expanded version of the CIDI (Robins et al., 1988). Items measuring parent attitudes about drinking were not available; however an item indexing perceived pressure to drink from the co-twin was included in the measure of sibling drinking. Maternal and paternal alcohol use was averaged.

2.3.3. Family smoking environment—Parent smoking, sibling smoking, and involvement of youth in family member smoking (getting or lighting cigarettes for family members) were indicators of family smoking environment in RHC. One parent per family reported their own and their partner's smoking and any involvement of the target child in family member smoking. Sibling smoking was reported by target youth. MTFS measures of self-reported co-twin and parent smoking were obtained using the SAM (Robins et al., 1987). An item indexing perceived pressure to smoke from the co-twin was included in the sibling smoking measure. Maternal and paternal reports of smoking were averaged.

2.3.4. Problem behaviors at age 24/25—In both studies, problem behaviors included ND, AUD, and IDUD DSM-IV symptom counts (American Psychiatric Association, 1994); antisocial behavior; and HIV SRB. In RHC, ND symptoms were measured using the Diagnostic Interview Schedule (Robins et al., 1981; 1989) in 2011 when the younger cohort was age 24 and the older cohort was age 25. All other problem behaviors were measured at age 24 for both cohorts. AUD and IDUD symptom counts were measured using the World Health Organization International Composite Diagnostic Interview (CIDI; World Health Organization, 1990). HIV SRB measures assessed having three or more sex partners in the past year, using condoms inconsistently with the last three sexual partners, being involved in prostitution, and having used alcohol or other drugs before having sex half of the time or more often. Scores represented a count of the number of sex risk behaviors reported (possible range 0–4). Antisocial behavior was operationalized as past-year involvement in crime, and was measured by self-report of engagement in a series of minor delinquency, property crime, drug crime, and violent crime acts in the past year. The number of crimes reported was summed.

In MTFS, ND, AUD, and IDUD symptoms were assessed using a modified version of the SAM (Robins et al., 1987). Antisocial behavior was operationalized as DSM-IV Antisocial Personality Disorder Criterion A (AAB) symptoms, and was assessed using a version of the Structured Clinical Interview for Personality Disorders (Spitzer et al., 1987) modified to cover DSM-IV symptoms. Structured diagnostic interviews were conducted by trained clinical interviewers. The measures of HIV SRB assessed past-year engagement in SRB under the influence of drugs and alcohol and number of sexual partners (casual and committed, lifetime and last 12 months). These two measures (sex under the influence and number of lifetime sexual partners) were standardized and summed to create a single measure of risky sex.

Both studies used community samples. As would be expected, few respondents in either study reported high numbers of ND, AUD, or IDUD symptoms or criminal/antisocial behaviors. For example, only 5% to 13% of RHC participants and 1% to 5% of MTFS participants reported more than four symptoms for any of the DSM-IV disorders measured.

For this reason and to mirror procedures used in Bailey et al. (2011), all symptom counts were capped at four.

2.3.5. Control variables—Control variables included child sex, race/ethnicity, and socioeconomic status (SES) for the family of origin. The RHC race/ethnicity measure combined parent reports, school records, and repeated self-reports; the MTFS race/ethnicity measure was a summation of repeated self-reports. Family of origin SES was measured in RHC by parent-reported eligibility for the National School Lunch/School Breakfast program between grades 5–7 and in MTFS by a composite of parent reports of mother's and father's education level, highest occupational prestige rating, and income.

2.4. Analysis

Correlation coefficients and structural equation models were estimated using Mplus version 6 (MTFS) and 7 (RHC; Muthén and Muthén, 1998–2011). Categorical variables were specified as such for both types of analysis. We used the Weighted Least Squares Means Variances (WLSMV) estimator and Theta parameterization, which allows for the estimation of residuals for categorical variables (Muthén and Muthén, 1998–2011). Full Information Maximum Likelihood estimation was used to minimize bias due to missing data. In RHC, the DIFFTEST option and multiple-group modeling were used to check the appropriateness of pooling treatment and control groups and older and younger cohorts for analysis. We found no differences in model parameters between treatment and control groups or between younger and older cohorts; all RHC participants were pooled for analysis. The analyses presented here included the 102 RHC siblings. Supplementary analyses including only one child per family (not shown) tested for differences due to sibling nesting within family. Patterns of significance were identical and parameter estimates were highly similar; most differed slightly at the second or third decimal place. Analyses of MTFS data accounted for clustering of twins within families using the COMPLEX specification.

3. RESULTS

Descriptive information and correlations are presented in Table 1 (RHC) and Table 2 (MTFS). In RHC, 35% of participants reported at least one ND symptom, 48% reported at least one AUD symptom, 39% reported at least one IDUD symptom, 19% reported at least one criminal activity, and 58% reported at least one SRB in young adulthood. In MTFS, 36% of participants reported at least one ND symptom, 35% reported at least one AUD symptom, 18% reported at least one IDUD symptom, and 57% reported at least one AAB symptom.

Structural equation models testing the fit of the heuristic model in each sample showed acceptable fit [RHC: χ^2 = 481.36 on 150 *df*, RMSEA = .046 (90% Confidence Interval = .042, .051), CFI = .93, WRMR = 1.26; MTFS: χ^2 = 237.64 on 82 *df*, RMSEA = .035 (90% Confidence Interval = .030, .041), CFI = .96; WRMR = 1.01]. Table 3 (RHC) and Table 4 (MTFS) display factor loadings and standard errors for each latent factor in the two samples. All indicators loaded significantly on their respective factor in both studies.

Figure 1 (RHC) and Figure 2 (MTFS) show the significant structural paths in the two samples, respectively. In both samples, adolescent general family environment, family smoking environment, and family drinking environment were all significantly related to the shared variance among young adult problem behaviors. Additionally, family smoking environment predicted unique variance in young adult ND symptoms in both samples. In the RHC sample, there was a significant, negative association between adolescent family smoking environment and unique variance in AUD symptoms at age 24; however, we believe this to be a suppressor effect given the positive zero-order correlation coefficients linking family smoking variables and young adult AUD symptoms (see Table 1). In the MTFS sample, there was no substance-specific association between family drinking or smoking and the unique variance in AUD symptoms. In both RHC and MFTS, family drinking and smoking environments were positively related to each other, and both were negatively related to general family environment. Males in both studies reported higher levels of general problem behavior in young adulthood. Neither study suggested associations between race/ethnicity and young adult problem behavior. SES was unrelated to young adult problem behavior among RHC youth, but those from higher SES backgrounds in MTFS reported more adult problem behavior. The positive SES/problem behavior link was interpreted as suppressor effect; SES was negatively related to problem behavior indicators at the zero order.

In the RHC sample, the model explained 25% of the variance in the general problem behavior factor, 50% of the variance in nicotine dependence symptoms, and 36% of the variance in AUD symptoms (all p's < .001). In the MTFS sample, the model explained 41% of the variance in the general problem behavior factor, 57% of the variance in ND symptoms, and 61% of the variance in AUD symptoms (all p's < .001). Unstandardized estimates for both RHC and MTFS are available in online supplementary materials[2].

4. DISCUSSION

This study aimed to replicate the heuristic model and findings from Bailey et al. (2011) in samples from RHC and MTFS, two independent, well-powered, longitudinal studies. There was strong consistency between the RHC and MTFS results that largely showed support for the methodological approach of partitioning environmental influence and phenotype into general and drug-specific components. In fact, patterns of significance in the RHC and MTFS samples were identical, with the exception of two likely suppressor effects, one inversely linking family smoking environment and unique variance in AUD symptoms in RHC and one linking higher SES with increased young adult problem behavior in MTFS.

As in the Bailey et al. (2011) paper, indicators of adolescent general family environment, family smoking environment, family drinking environment, and young adult general problem behavior formed usable latent factors. The general family environment to general problem behavior link found in the original study was replicated in both samples. Taken together with the Bailey et al. (2011) and Epstein et al. (2013) studies, the current results suggest that the quality of the general family environment in adolescence is predictive of a

[2]See Supplementary Tables 2 and 3 by accessing the online version of this paper at http://dx.doi.org and by entering doi:…

range of problem behaviors in adulthood and constitutes an important target for preventive interventions. These results are consistent with a large body of literature that underscores the importance of positive adolescent family environment in preventing young adult problem behaviors (e.g., Bailey et al., 2006; Chassin et al., 1998; Epstein et al., 2013) and support the use of the general/specific modeling framework for future gene-environment interplay studies.

Adolescent family smoking environment was consistently related to unique variance in adult ND in both samples studied here and in the SSDP data (Bailey et al., 2011; Epstein et al., 2013). Other researchers also have found substance-specific links between family smoking environment and smoking among young adults (Chassin et al., 1998; Keyes et al., 2008). This emerging pattern of results suggests an important and unique role of family smoking environments in young adult ND. Preventive interventions aimed at reducing young adult smoking need to target the smoking behavior and attitudes of family members during childhood and adolescence (see also Stone et al., 2012). General family environment, however, also contributed indirectly to adult ND in both samples, suggesting that smoking prevention efforts also should target general family characteristics like monitoring, bonding, conflict, and involvement.

Neither this study nor Epstein et al. (2013) found a link between family drinking environment and unique variance in AUD symptoms, as was reported by Bailey et al. (2011). Notably, the alcohol environment to unique alcohol variance link was small in the original study (standardized estimate = .11, $p = .04$). In the two studies reported here, it appears that family drinking environment may operate indirectly on young adult AUD via its link to general problem behavior. This finding goes against some prior literature that suggests alcohol-specific heritability (Kendler et al., 2003; Hicks et al., 2004), but is consistent with studies demonstrating heritability or intergenerational transmission of a general tendency toward antisocial behavior, of which alcohol use is a part (Bailey et al., 2006; McGue et al., 2006; Dick et al., 2010). More research is needed to determine whether adolescent family drinking environment contributes to unique variance in young adult AUD that is not shared with other problem behaviors and to understand the association between family drinking environment and young adult problem behavior.

RHC and MTFS data showed associations between family smoking and drinking environments and young adult general problem behavior that were not found in Bailey et al. (2011) or Epstein et al. (2013). Sample differences between the prior studies and the studies reported here likely explain this difference in findings. The SSDP sample was largely urban, sampled respondents from a mix of high-risk and lower risk neighborhoods, and was racially/ethnically diverse, including nearly 50% non-White participants. In contrast, both samples used here were mostly White (75% in RHC, 95% in MTFS), less urban, and not oversampled for high-risk individuals. In these lower risk samples, family smoking and drinking may have been more indicative of deviance, and therefore more closely tied to young adult general problem behavior than in the higher risk sample used in the first two studies. Future replication attempts should test the generalizability of findings with respect to race/ethnicity, urban/suburban location, neighborhood risk, and socioeconomic status.

Study strengths include the longitudinal data, the relatively large samples, and the congeneric measures across studies. Some limitations should be considered when drawing conclusions from the current findings. The samples used here are geographically restricted (although not to the same region), which may limit generalizability. Exact replication of long-term, longitudinal studies is extremely difficult; the replications reported here are conceptual rather than exact. Some authors have argued that exact replications are to be preferred, and that replications should be independent, not including authors of the original study(ies) (Makel et al., 2012). We enthusiastically encourage further replications of the proposed heuristic model. Although both studies were longitudinal, the current procedures did not investigate potential changes in the roles of family general, smoking, and drinking environments across adolescent development.

Understanding the etiology of adult problem behavior requires measuring and defining complex phenotypes and modeling a broad array of interacting genetic and environmental risk and protective factors. The proposed simplifying model offers several advantages for those studying GxE and *r*GE in problem behavior. It yields a small number of parsimonious measures of environment that reflect current knowledge about the multidimensional nature of environmental influence and have clear implications for treatment and prevention. Although it has yet to be tested with molecular genetic data, we believe the model will facilitate the inclusion of stronger and more sophisticated measures of environment in GxE studies.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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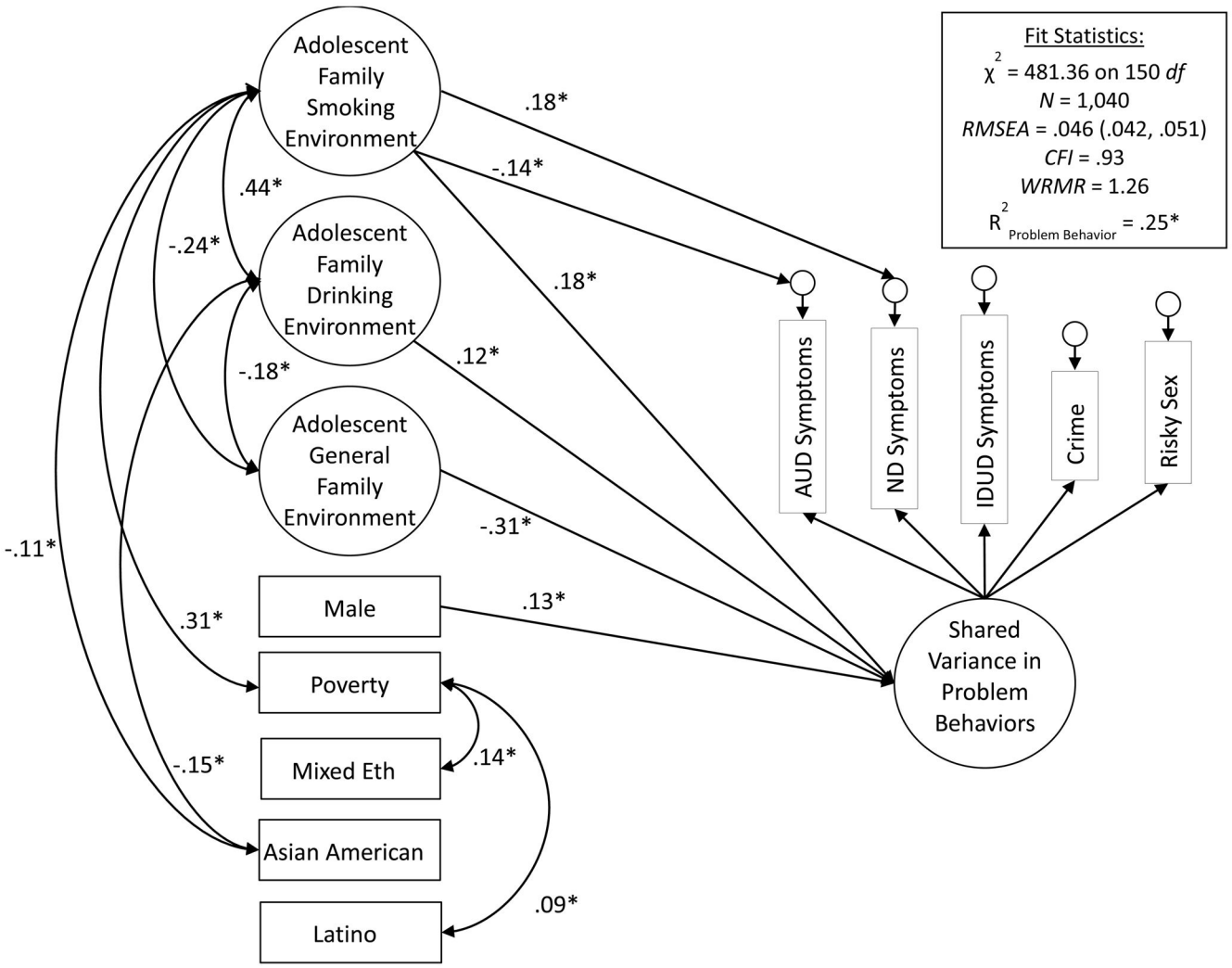

Figure 1.

Structural equation modeling results for RHC sample: standardized coefficients. Nonsignificant paths are not shown for readability. ND = nicotine dependence, AUD = alcohol use disorder, IDUD = illicit drug use disorder.

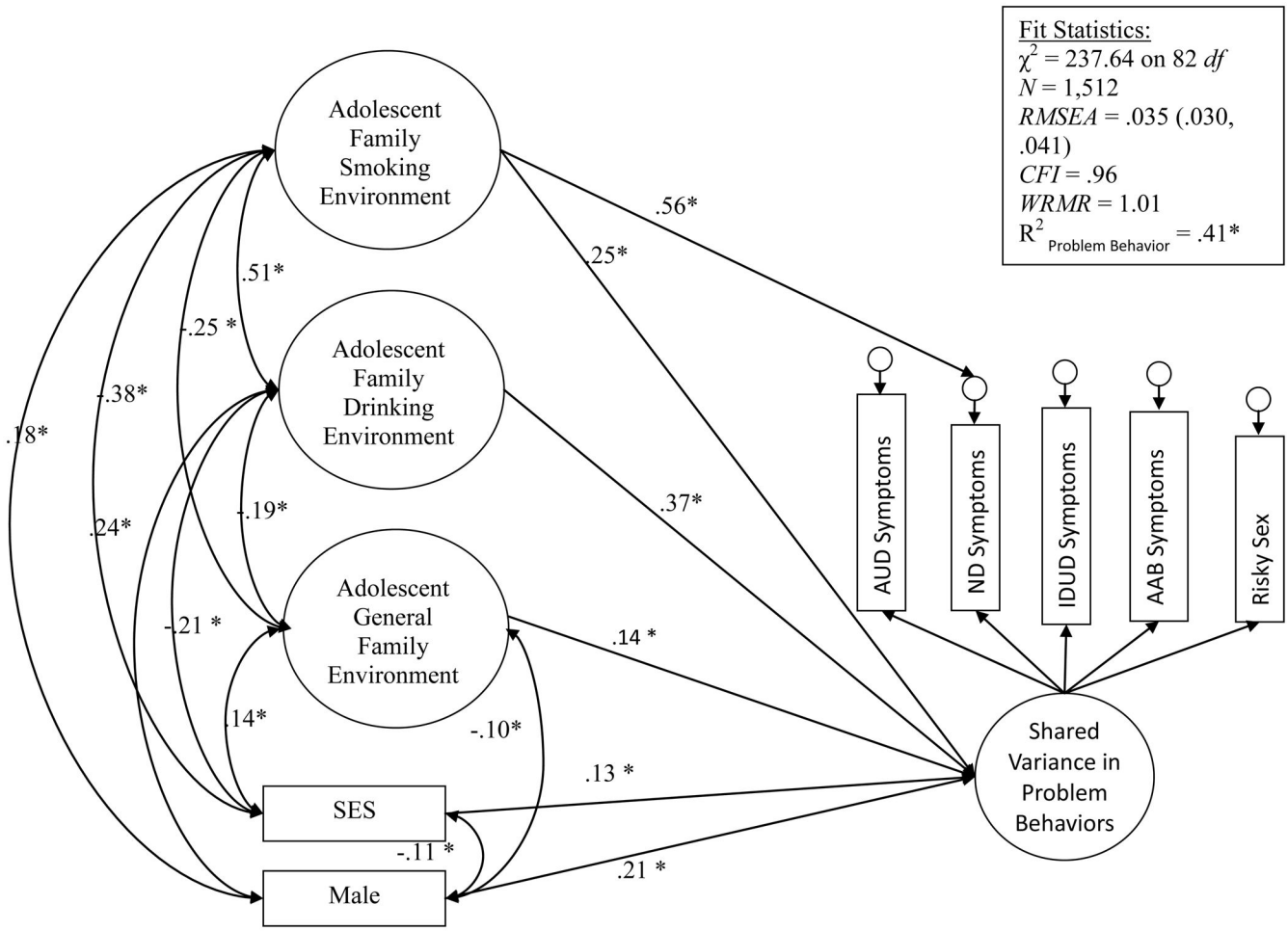

Figure 2.

Structural equation modeling results for MTFS sample: standardized coefficients. Nonsignificant paths are not shown for readability. Co-twin smoking and drinking were correlated, as were parent smoking and drinking; these paths were omitted to improve readability of the figure. ND = nicotine dependence, AUD = alcohol use disorder, IDUD = illicit drug use disorder, AAB = adult antisocial behavior.

Table 1

Correlations and descriptive statistics for variables in RHC.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Fam management	-															
2. Fam conflict	−.65*	-														
3. Involvement	.73*	−.54*	-													
4. Bonding	.73*	−.60*	−.72*	-												
5. Sib Smoking	−.20*	.16*	−.19*	−.19*	-											
6. Par Smoking	−.07*	.13*	−.17*	−.15*	.23*	-										
7. Involvement in par smoking	−.03	.05*	−.06*	−.03	.16*	−.28*	-									
8. Sib drinking	−.25*	.19*	−.22*	−.19*	.73*	.16*	.05	-								
9. Par drinking	−.06	.10*	−.06	−.07*	.11*	.27*	.09*	.15*	-							
10. Pro drink attitudes	−.07*	.11*	−.07*	−.07*	.04	.22*	.17*	.06	.46*	-						
11. Involvement in par drinking	−.03	.06*	.00	−.01	−.00	.06	.41*	−.00	.30*	.22*	-					
12. Risky sex	−.10*	.09*	−.08	−.07	.05	.02	.02	.10*	.02	.02	.03	-				
13. Crime	−.18*	.24*	−.21*	−.17*	.13*	.14*	.15*	.19*	.11*	.05	.08*	.33*	-			
14. ND symptoms	−.17*	.26*	−.21*	−.19*	.27*	.23*	.10*	.22*	.10*	.07	.06	.10*	.42*	-		
15. AUD symptoms	−.17*	.15*	−.16*	−.16*	.19*	.08	.00	.19*	.11*	.13*	.02	.28*	.46*	.36*	-	
16. IDUD symptoms	−.27*	.30*	−.28*	−.26*	.24*	−17*	.07	.27*	.17*	.11*	.06	.16*	.57*	.64*	.45*	-
M	3.29	2.06	2.82	3.01	.21	.03	1.10	.22	.01	1.71	1.15	.70	.30	.88	1.21	1.06
SD	.37	.48	.50	.46	.30	.60	.25	.27	.50	.44	.26	.70	.76	1.36	1.52	1.55

* $p < .05$

NOTE: Fam = family, Sib = sibling, Par = parent, ND = nicotine dependence, AUD = alcohol use disorder, IDUD = illicit drug use disorder.

Table 2

Correlations and descriptive statistics for variables in MTFS.

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Fam management	--												
2. Fam conflict	−.55*	--											
3. Involvement	.43*	−.38*	--										
4. Bonding	.70*	−.63*	.48*	--									
5. Co-twin smoking	−.19*	.12*	−.20*	−.08*	--								
6. Par smoking	−.10*	.04	−.08	.00	.23*	--							
7. Co-twin drinking	−.18*	.12*	−.13*	−.06	.59*	.09*	--						
8. Par drinking	−.03	−.01	.01	.06	.14*	.31*	.21*	--					
9. Risky sex	−.16*	.24*	−.11*	−.15*	.21*	.05	.30*	.11*	--				
10. AAB symptoms	−.20*	.20*	−.11*	−.16*	.27*	.14*	.33*	.13*	.50*	--			
11. ND Symptoms	−.20*	.20*	−.14*	−.08*	.48*	.23*	.35*	.13*	.32*	.48*	--		
12. AUD symptoms	−.16*	.22*	−.13*	−.13*	.23*	.05	.30*	.14*	.49*	.67*	.46*	--	
13. IDUD symptoms	−.11*	.17*	−.02	−.08	.25*	.15*	.27*	.07	.41*	.66*	.49*	.56*	--
M	.00	−.00	−.00	.00	.01	.06	.06	−.04	.05	.82	.88	.75	.45
SD	.68	.74	.68	.75	2.43	3.47	3.23	4.24	1.70	.92	1.35	1.26	1.11

* $p < .05$

NOTE: Fam = family, Par = parent, AAB = Adult Antisocial Behavior, ND = nicotine dependence, AUD = alcohol use disorder, IDUD = illicit drug use disorder.

Table 3

Factor loadings for latent variables in RHC.

	Standardized Estimate (SE)
Family general environment	
Family management	.86 (.02)*
Conflict	−.75 (.02)*
Involvement	.82 (.02)*
Bonding	.82 (.02)*
Family drinking environment	
Pro drinking attitudes	.58 (.04)*
Parent drinking	.73 (.04)*
Sibling drinking	.33 (.04)*
Involvement in parent drinking	.37 (.02)*
Family smoking environment	
Parent smoking	.72 (.04)*
Sibling smoking	.47 (.05)*
Involvement in parent smoking	.36 (.02)*
Shared variance in problem behaviors	
Alcohol use disorder symptoms	.62 (.05)*
Nicotine dependence symptoms	.66 (.05)*
Illicit drug use disorder symptoms	.86 (.04)*
Crime	.69 (.04)*
Sex risk behavior	.28 (.04)*

* $p < .05$

Table 4

Factor loadings for latent variables in MTFS.

	Standardized Estimate (SE)
Family general environment	
Family management	.82 (.02)*
Conflict	−.73 (.02)*
Involvement	.56 (.03)*
Bonding	.79 (.02)*
Family drinking environment	
Parent drinking	.31 (.05)*
Co-twin drinking	.70 (.05)*
Family smoking environment	
Parent smoking	.27 (.05)*
Co-twin smoking	.77 (.08)*
Shared variance in problem behaviors	
Alcohol use disorder symptoms	.80 (.04)*
Nicotine dependence symptoms	.32 (.05)*
Illicit drug use disorder symptoms	.73 (.03)*
Adult antisocial behavior	.86 (.02)*
Sex risk behavior	.61 (.02)*

* $p < .05$

NOTE: SE = Standard Error