HHS Public Access

Author manuscript

Alcohol Clin Exp Res. Author manuscript; available in PMC 2019 September 01.

Published in final edited form as:

Alcohol Clin Exp Res. 2018 September ; 42(9): 1783–1794. doi:10.1111/acer.13827.

Influence of Parental Alcohol Dependence Symptoms and Parenting on Adolescent Risky Drinking and Conduct Problems: A Family Systems Perspective

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Abstract

Background—Parental alcohol problems are associated with adverse adolescent outcomes such as risky drinking and conduct problems. Important questions remain about the unique roles of fathers' and mothers' alcohol problems and differences and/or similarities in pathways of risk across ethnicity and gender. In this study, we used a family systems approach to consider spillover and crossover effects of fathers' and mothers' alcohol problems (number of alcohol dependence symptoms) and parenting behaviors in relation to adolescents' risky drinking and conduct problems.

Methods—The sample included 1,282 adolescents (aged 12–17) and their parents from the Collaborative Study on the Genetics of Alcoholism. Parents completed the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) and adolescents completed an adolescent-version of SSAGA. Data were analyzed using multivariate structural equation modeling.

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Results—Fathers' alcohol dependence symptom count was associated with higher adolescent risky drinking and conduct problems indirectly via disruption to fathers' and mothers' positive parenting behaviors, whereas mothers' alcohol dependence symptom count was not associated with adolescents' risky drinking and conduct problems directly or indirectly via positive parenting behaviors. No differences in these associations were found across ethnic background and offspring gender.

Conclusions—Findings highlight the importance of considering the unique roles of fathers' and mothers' alcohol dependence symptoms in influencing family processes and adolescent outcomes.

Keywords

alcohol dependence; parenting; risky drinking; externalizing; conduct problems

Introduction

It has been estimated that 7.5 million, or 10.5% of children younger than the age of 18 in the United States, live with a parent who had an alcohol use disorder in the past year (SAMHSA, 2012). This is a major public health concern given the substantial evidence that offspring of parents with alcohol problems are at an elevated risk for a host of psychosocial problems (Park & Schepp, 2015). Here, we took a family systems perspective to examine parenting as a mechanism linking parental alcohol dependence symptoms (ADS) to adolescents' risky drinking and conduct problems. Expanding upon previous research, we considered how fathers' and mothers' ADS might influence their own (spillover effect) as well as the other parent's parenting behaviors (crossover effect), in relation to adolescent outcomes. We further examined whether and how these associations may vary across adolescent ethnicity and gender.

Direct and Indirect Effect of Parental Alcohol Dependence Symptoms via Parenting Behaviors: A Family Systems Perspective

Parental alcohol problems, both concurrent parental drinking problems and family/parental history of alcoholism, have been shown to be associated with a range of offspring maladaptive outcomes from childhood to adulthood (e.g., Conway et al., 2004; Schepis et al., 2008). Among adolescents, parental alcohol dependence is associated with higher risk for early initiation of alcohol use (Waldron et al., 2014), higher levels of alcohol consumption and heavy episodic drinking (Lieb et al., 2002), more externalizing problems, such as aggression (Hussong et al., 2010) and conduct disorders (Kuperman et al., 1999), and more depressive and anxiety symptoms (Waldron et al., 2009). In this study, we focus on adolescents' risky drinking, because adolescence is the developmental period when many individuals initiate alcohol use and risky drinking during adolescence is a precursor for alcohol problems later in life (Marshall, 2014). We take a multivariate approach to also consider adolescents' conduct problems as they are prevalent (Merikangas et al., 2009) and often co-occur with alcohol use and misuse among adolescents (Armstrong & Costello, 2002).

Parents serve as a primary socialization agent for adolescents and play an important role in influencing adolescent outcomes such as substance use and deviant behaviors through parenting and parent-child interactions (Oetting & Donnermeyer, 1998). Thus, in addition to direct associations (due to social learning or modeling), parental alcohol dependence may influence adolescent adjustment indirectly by disrupting positive parenting behaviors. Positive parenting includes parental behaviors that express and convey support, warmth, affection, and acceptance to their offspring (Oppenheimer, Hankin, Jenness, Young, & Smolen, 2013). The extent to which parents exhibit positive parenting toward their children is associated with a variety of developmental outcomes, including problematic alcohol use and externalizing problems. For example, low levels of positive parenting such as low parental involvement and support (Barnes et al., 2000; Shelton & van den Bree, 2010), low parental warmth (Klevens & Hall, 2014), poor parent-child communication (Luk et al., 2010), and low parent-child closeness (Shorey et al., 2013), have been associated with more alcohol use and externalizing problems among adolescents. Parental alcohol problems consume parents' psychosocial and financial resources. Thus, parents with concurrent or past (through legacy or cascading effects) alcohol problems may be impaired in their ability to maintain good home environments and provide positive parenting behaviors, which in turn negatively influence adolescent outcomes. Indeed, parental alcohol dependence is associated with decreased parental monitoring (Chassin et al., 1993; Shorey et al., 2013), lower parental warmth and involvement (Kachadourian et al., 2009), poorer parent-child communication (Ohannessian, 2012), and lower parent-child closeness (Shorey et al., 2013), which in turn are related to higher adolescent risky drinking and conduct problems.

Many studies on the effects of parental alcohol dependence have focused on fathers' alcohol dependence or operationalized parental alcohol dependence broadly (e.g., having a parent with alcohol dependence or not) (e.g., Chassin et al., 2004; Elkins et al., 2004; Kachadourian et al., 2009). Thus, there is a limited understanding of the unique roles of fathers' and mothers' alcohol dependence in influencing adolescent outcomes. This is important to note, given that the effects of parental alcohol dependence may differ between fathers and mothers. Ohannessian and colleagues (2005), for example, found that fathers' alcohol dependence, but not mothers' alcohol dependence, was related to drinking behaviors among adolescents. It was also found that fathers' and mothers' problem drinking differentially affect substance use for boys and girls such that the association between parental problem drinking and adolescent substance use was stronger in same-sex parent-adolescent dyads (e.g., mother-daughter; Ohannessian, 2012). Furthermore, some studies suggest that fathers' and mothers' problem drinking may influence adolescents' substance use and externalizing problems via different mediating pathways (Finan et al., 2015; Shorey et al., 2013).

Moreover, even fewer studies have considered alcohol problems and parenting behaviors from both fathers *and* mothers as separate constructs and analyzed them simultaneously in the same model to examine whether and how one parent's alcohol problems may influence the other parent's parenting behaviors, which in turn can affect adolescent outcomes. This is a noteworthy gap, given that researchers have called for using a family systems perspective to better understand individual development (Cox & Paley, 2003). Family systems theory posits that individuals or subsystems (e.g., the parental or the parent-child subsystem) in the family are interdependent and influence one another through processes such as spillover

and/or crossover effects (White & Kline, 2002). Spillover refers to the transfer of affect or behavior within one person across subsystems (e.g., father's ADS influences father's parenting behavior), whereas crossover refers to the transfer of affect or behavior between people in the family (e.g., father's ADS influences mother's parenting behavior). Here, we considered the unique contributions of fathers and mothers and the spillover and crossover effects between fathers' and mothers' ADS and parenting behaviors in relation to adolescent outcomes.

Ethnic Differences

Rates of adolescents' drinking and conduct problems vary across ethnicity. Compared to European American (EA) adolescents, African American (AA) adolescents are less likely to consume alcohol (Johnston et al., 2015) but have more conduct problems (McLaughlin et al., 2007). In addition, the influence of risk and protective factors, including those from the family context, on adolescent outcomes may differ across ethnic groups (Su & Supple, 2014). EA and AA families often differ in their neighborhood environments, socioeconomic status, and cultural norms, which may lead to differences in parenting behaviors and their effects on child development (Amato & Fowler, 2002; Hill, 2006). For example, a high level of parental control may be benign or protective among AAs (compared to EAs), because it is relatively normative in AA communities, particularly among those who live in at-risk neighborhoods (Deater-Deckard & Dodge, 1997; Mason et al., 2004). Indeed, an authoritarian parenting style characterized by low acceptance and high control was associated with lower increase in heavy episodic drinking over time for AA adolescents but not for EA adolescents (Clark et al., 2015). Deutsch and colleagues found that maternal support was more strongly associated with lower delinquent behaviors among EA adolescents than for AA adolescents (Deutsch et al., 2012). Other studies found no ethnic differences in the associations between parental problem drinking, parenting, and adolescent drinking and related outcomes (Shorey et al., 2013; Skinner et al., 2011). There is a clear need for more research to examine potential differences in pathways of risk/protection to alcohol use and related outcomes across ethnic groups.

Gender Differences

In addition to gender differences in adolescents' drinking patterns (Johnston et al., 2015) and prevalence of conduct problems (Hicks et al., 2007), researchers have suggested that there may be gender differences in physiological, psychological, and social factors influencing alcohol use and related outcomes (Schulte et al., 2009). There are mixed results from prior research on whether parental alcohol dependence/problems and parenting behaviors are associated with alcohol use and externalizing problems differentially in male and female adolescents (e.g., Elkins et al., 2004; Luk et al., 2010; Morgan et al., 2010; Ohannessian et al., 2005). For example, some studies showed that parental closeness (Kelly et al., 2011) and parental support (Choquet et al., 2008) have a stronger effect on alcohol and substance use for adolescent girls than for boys. Other studies found parental monitoring to be more protective in reducing substance use and externalizing behaviors among adolescent boys than girls (Borawski et al., 2003; Tebes et al., 2011). Yet, others found no differences in the relations between parental alcohol problems, parenting, and adolescent outcomes (Shorey et al., 2013). These mixed findings could be in part due to differences in sample characteristics

(i.e., age of the adolescents) and research methodology (i.e., measurement of parenting behaviors) across studies. Furthermore, few studies have examined these offspring gender differences when considering the unique influences of fathers and mothers (Ohannessian, 2012; Shorey et al., 2013).

The Current Study

The primary goal of this study was to consider the potentially unique roles of fathers' and mothers' ADS in influencing adolescent risky drinking and conduct problems, both directly and indirectly via parenting behaviors. A secondary goal was to examine potential differences in these pathways of risk based on adolescents' ethnicity and gender. Grounded in family systems theory (Cox & Paley, 2003), we considered ADS and parenting behaviors from both fathers and mothers simultaneously to examine their unique roles, as well as the spillover and crossover effects between fathers' and mothers' ADS and parenting behaviors, in influencing adolescent outcomes. We hypothesized that, in addition to direct associations, fathers' and mothers' ADS would have indirect effects on adolescents' risky drinking and conduct problems via a pathway marked by disruptions to their own (spillover effect) and the other parent's (crossover effect) positive parenting behaviors. We did not have specific hypotheses regarding differences in these risk pathways across ethnicity and adolescent gender given the lack of consistent evidence from prior research.

Materials and Methods

Sample

Participants came from the Collaborative Study on the Genetics of Alcoholism (COGA; Begleiter et al., 1995), a multi-site study that was designed to identify genes involved in alcohol dependence and related phenotypes. Probands were identified through alcohol treatment programs at six U.S. sites and were invited to participate if they had a sufficiently large family (usually sibships of more than three with parents available) with two or more members in the COGA catchment areas. Population-based comparison families were also recruited. Data collection for COGA started in 1991 when adults in the target extended families were invited to complete the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA), a comprehensive interview that assesses alcohol use disorders and other psychiatric phenotypes such as major depressive disorder and conduct disorder (Bucholz et al., 1994). Approximately 5 years later, adults in the COGA extended families were invited again to complete a SSAGA interview (regardless of whether they completed interviews in the first wave); thus some adults had one interview while others completed two SSAGA interviews.

In 2004, COGA launched the Prospective Study that aims to examine how genetic risks unfold across development and in conjunction with the environment. Specifically, offspring of adults in the COGA extended families who were aged between 12 and 22 and had at least one parent who completed SSAGA in the original COGA adult interviews were recruited for the COGA Prospective Sample (Bucholz et al., 2017). These offspring participants complete SSAGA, or an adolescent version of SSAGA (i.e., CSSAGA) if they were younger than 18 years old, at enrollment and are re-interviewed at about 2-year intervals. For those COGA

Prospective participants where only one parent had completed SSAGA, the other parent was invited for a SSAGA interview when his/her offspring enrolled in the COGA Prospective Study.

For the purpose of the current study, we linked data from the COGA adult interviews and the Prospective Study. We only included adolescents who 1) completed their baseline assessment at enrollment, and their first follow-up assessment (approximately two years after the baseline), 2) were aged between 12 and 17 at their first follow-up assessment, and 3) self-identified as European American (EA) or African American (AA) — the two largest ethnic groups in COGA. This strategy resulted in an analytic sample of 1,282 adolescents from 497 COGA extended families (50.0% female, 69.1% EA). This is a relatively early adolescent sample, as 47.4% of the sample was aged 12 at their baseline assessment (M_{age} = 12.99, *SD* = 1.13). Fathers' age ranged from 28 to 70 (M_{age} = 44.39, *SD* = 6.17) and mothers' age ranged from 27 to 58 (M_{age} = 41.74, *SD* = 5.82) at the time of their adolescents' baseline assessment.

Measures

Parental alcohol dependence symptoms (ADS)—Data on parental ADS were drawn from parents' SSAGA interviews. 425 (33.2%) fathers and 662 (51.6%) mothers completed SSAGA in the first wave of COGA; 648 (50.5%) fathers and 1011 (78.9%) mothers completed SSAGA in the 5-year follow up wave. Only a small subset of parents (93 fathers and 111 mothers) completed SSAGA when their adolescents enrolled in the COGA Prospective Sample, as many of them already completed SSAGA earlier and thus were not invited for the interview again. Some parents refused to participate and others were lost to contact (e.g., because of parental separation and/or not living with the offspring). For parents (373 fathers and 626 mothers) who completed SSAGA twice, we used data from the interview in which they endorsed the greatest number of ADS to index their ADS. Parental ADS was operationalized as the number of alcohol dependence criteria endorsed according to the DSM-IV criteria (American Psychiatric Association, 1994). Our rationale for using ADS data from a single interview (vs. calculating average scores across multiple assessments when available) was that in high-risk sample such as COGA we wanted to measure parents' greatest expression of their predisposition/risk to alcohol dependence on which the sample was originally ascertained.

Of the fathers who had data on ADS (n= 805), 31.1% endorsed zero ADS and 40.9% endorsed 3 or more ADS, which met the clinical diagnosis for DSM-IV alcohol dependence. Of the mothers who provided data on ADS (n = 1,177), 46.3% endorsed zero ADS, and 25.7% endorsed 3 or more ADS. We used ADS rather than alcohol dependence diagnosis in order to increase power for analysis. Of the families where ADS data are available both for father and mother (n = 706), 25.2%, 8.2%, and 13.6% had only father, only mother, and both parents met diagnosis for alcohol dependence, respectively.

Positive parenting—Data on parenting behaviors were drawn from the Home Environment section of adolescents' baseline CSSAGA assessment. Items accessing parenting behaviors were adapted from the Home Environment Interview for Children

(Reich, Earls, & Powell, 1988). We conducted an exploratory factor analysis (EFA) with items that we considered broadly tap onto the positive parenting construct, which yielded three subscales that we labeled as parental involvement, parent-child communication, and parent-child closeness. In view of the previous findings that multiple dimensions of parenting better account for their influence on adolescent behaviors than a single dimension (Ryan et al., 2010), we used these three subscales to index positive parenting behaviors as a latent construct. The parental involvement scale asked adolescents 5 questions about whether or not their father/mother figure helped them with schoolwork, chores, fun activities, shopping, and making plans. Responses were coded as 0 (*no*) and 1 (*yes*). Scores were summed to create variables for *maternal involvement* and *paternal involvement.* The parent-child communication scale contained 3 items asking adolescents whether or not they and their father/mother figure talked about news, their problems, or other things like movies, friends or anything else. Response options were 0 (*no*) and 1 (*yes*) for each item. Sum scores were used to create variables for *mother-child communication* and *father-child communication.* The parent-child closeness scale included 2 questions that asked participants how well they got along with their father/mother figure most of the time and how close they felt to their father/mother figure. Response options ranged from 1 (*poor*) to 4 (*excellent*), and from 1 (*not at all close*) to 3 (*very close*) for the two questions, respectively. Scores were standardized and averaged across the two items to create variables representing *father-child closeness* and *mother-child closeness.* Correlations between the three scales ranged from .39 to .49 (mean $r = .42$) and from .50 to .57 (mean $r = .54$) for fathers and mothers, respectively. Some adolescents reported parenting behaviors for their non-biological father (n = 370) or mother (n = 82) figures. Because we only had data on alcohol dependence symptoms from biological parents, adolescent-reported parenting behaviors of non-biological parents was coded as missing.

Adolescent risky drinking—Data on adolescent risky drinking, as indexed by engagement in heavy episodic drinking in the past year, came from adolescents' first follow-up assessment. Adolescents responded to one question: "how often did you have 5 or more drinks in 24 hours during the last 12 months". Responses ranged from " 0 = *never*" to "12 = *every day*". Adolescents who reported never initiating alcohol use were assigned a score of zero. Because 85.8% of adolescents reported no heavy episodic drinking in the past year, we created a dichotomous variable to indicate engagement in risky drinking.

Adolescent conduct problems—Data on adolescent conduct problems, as indexed by conduct disorder criterion counts (CDSX), also came from adolescents' first follow-up assessment. CDSX was operationalized as the number of conduct disorder symptoms (e.g., often bullies, threatens, or intimidates others) ever endorsed according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) and had a possible range of 0–15. In the current sample, CDSX ranged from 0 to 11. In total, 66.0% of the adolescents endorsed 0 CDSX, 18.3% endorsed 1 CDSX, 8.8% endorsed 2 CDSX, and 6.9% endorsed 3 or more CDSX. Because endorsing 3 or more CDSX meets the clinical diagnostic criteria for conduct disorder, we grouped adolescents into four categories to indicate their conduct problems (0 = no CDSX, 1 = one CDSX, 2= two CDSX, 3 = three or more CDSX).

Covariates—We included adolescents' age, gender (1 = *male*, 0 = *female*), and ethnicity (1 = *European American*, 0 = *African American*) as covariates given prior evidence that they are related to adolescent alcohol use and conduct problems (Johnston et al., 2015). Because family structure may impact parental influences and family processes (Brown & Rinelli, 2010), we also considered family structure as a covariate in this study. *Family structure* was coded as 1 if adolescents indicated that they lived with both biological mother and biological father; all other family living arrangements (e.g., one parent, step-parent) were coded as 0. Given that family socioeconomic status has been associated with adolescent alcohol use and conduct problems and may confound the effect of ethnicity (Piotrowska et al., 2015; Goodman & Huang, 2002), we included parental education, indexing family socioeconomic status, as a covariate in this study. Parents reported their highest level of education as part of SSAGA, and scores were converted to the number of years typically required to complete that level of education. *Parental education* was calculated as the average of maternal and paternal education. In cases where information was only available for one parent, the available score was used to index parental education. Because alcohol use disorders often co-occur with internalizing and/or externalizing psychopathology (Dawson et al., 2010), we also included paternal and maternal antisocial behavior and internalizing problems as covariates. *Paternal/maternal antisocial behavior* was operationalized as the number of DSM-IV Antisocial Personality Disorder criteria endorsed at the SSAGA assessment when fathers/mothers indorsed their maximum number of alcohol dependence symptoms. *Paternal/maternal internalizing problems* was measured using a 7-item Internalizing Symptom Scale recently developed by COGA researchers based on the SSAGA (Acion et al., 2017). This scale assessed symptoms related to agoraphobia, panic disorder, social phobia, obsessive compulsive disorder, post-traumatic stress disorder, major depression, and suicidality that took place in the context of depression; the scale has a possible range between 0 and 21.

Analytic Strategy

We began with preliminary analyses to examine the amount and patterns of missingness of our data, in order to determine the appropriate approach to handle missing data in subsequent analysis, followed by examination of descriptive statistics and correlations among study variables. We then examined the associations between parental ADS, parenting behaviors, and adolescent risky drinking and conduct problems by conducting structural equation models (SEM) using Mplus version 7.31 (Muthén & Muthén, 1998–2012). We conducted a multivariate SEM model to consider adolescents' risky drinking and conduct problems simultaneously given that they are related constructs (see Figure 1). Fathers' positive parenting was modeled as a latent variable indicated by paternal involvement, father-adolescent communication, and father-adolescent closeness. Mothers' positive parenting was modeled as a latent variable indicated by maternal involvement, mother-adolescent communication, and mother-adolescent closeness. To account for method effects, and to take into account the shared variances between fathers' and mothers' parenting (so that unique effects of fathers' and mothers' parenting can be examined), errors terms were correlated between fathers' and mothers' parenting subscales, as were the residual errors for mothers' and fathers' positive parenting latent factors. The residual errors for adolescent risky drinking and conduct problems were also allowed to be correlated. Standardized

loadings for each indicator and correlated error terms for the latent constructs are presented in Table 3. Risky drinking and conduct problems were specified as categorical variables in the SEM model, and thus the robust weight least square estimator (WLSMV) was used by default in Mplus.

In the path model, fathers' and mothers' ADS were specified as predictors of their own and the other parent's parenting behaviors, as well as adolescents' risky drinking and conduct problems. Fathers' ADS and mothers' ADS were specified as correlated. Fathers' and mothers' parenting behaviors were specified as predictors of adolescent risky drinking and conduct problems. Adolescents' age, gender, and ethnicity, family structure, parental education, paternal/maternal antisocial behavior and internalizing problems were included as covariates to predict adolescent risky drinking and conduct problems. To evaluate indirect effects (e.g., father ADS → father parenting → adolescent risky drinking), we used the MODEL CONSTRAINT command in Mplus to specify new parameters that represent the multiplication of coefficients for the two specific paths (e.g., father → father parenting (*a*), and father parenting → adolescent risky drinking (*b*)) that comprise the indirect pathway. These new parameters (*ab*), which were tested along with other path coefficients in the model, provided a test of indirect effects. MODEL CONSTRAINT is the recommended method for estimating indirect effects in Mplus in the context of multiple imputation (Muthén & Muthén, 1998–2012). The estimation of indirect effects via the MODEL CONSTRAINT command uses the delta method (MacKinnon, 2008) that takes into account the covariance between the *a* and *b* estimates and provides estimates of standard errors for the indirect effects.

To test for potential differences in the associations between parental ADS, parenting behaviors, and adolescent outcomes across ethnicity and adolescent gender, we first conducted multigroup confirmatory factor analyses (MGCFA) to consider measurement invariance for the paternal and maternal positive parenting latent factors across the comparison groups (i.e., EA vs AA; Male vs Female), followed by multigroup SEM analysis with Wald chi-square test of parameter equalities. MGCFA compared two alternative models, one with factor loadings for the latent constructs freely estimated across groups and another model where factor loadings were constrained to equality across groups. To the extent that the two alternative models do not differ significantly in model fit, as indicated by a non-significant chi-square difference test and/or difference in Comparative Fit Index (ΔCFI) smaller than .01 in absolute value (Cheung & Rensvold 2002), there is evidence of metric measurement invariance, which is sufficient for group comparisons in structural path coefficients (Byrne & Watkins, 2003; Steenkamp & Baumgartner, 1998). To test for potential differences in path coefficients between males and females, multigroup analysis was conducted by removing gender from the SEM model and then comparing a model with all remaining paths constrained to equality with one that had all paths freely estimated across male and female adolescents. Similarly, to test for potential differences in path coefficients across ethnic groups, multigroup analysis was conducted by removing ethnicity from the SEM model and then comparing a model with all remaining paths constrained to equality with one that had all paths freely estimated across EA and AA adolescents. We accounted for interdependence within family in all analyses using the CLUSTER command in Mplus.

Results

Missing Data and Imputation

Of the whole sample, 477 (37.2%) adolescents were missing data for fathers' ADS, and 105 (8.2%) were missing data for mothers' ADS. Adolescents who had missing data on their fathers' and/or mothers' ADS did not differ in age, gender, risky drinking, conduct problems, and their self-reported parenting behaviors, compared to adolescents who were not missing any data for their fathers' and mothers' ADS. African American (AA) adolescents (64.1%) were more likely to have missing data on parental ADS than European American (EA, 36.3%) adolescents ($\chi^2 = 85.42$, $df = 1$, $p < .001$). 373 (29.1%) adolescents were missing data on fathers' parenting and 83 (6.5%) were missing data for mothers' parenting. AA adolescents (49.7%) were more likely to have missing data on fathers' parenting than EA adolescents (19.9%) ($\chi^2 = 118.47$, $df = 1$, $p < .001$).

We used multiple imputation to account for missing responses, given that multiple imputation is more flexible to handle missing data for a mixture of categorical and continuous variables (Enders, 2010). Multiple imputation has also been shown to be robust and provide unbiased results even for high rates of missing data (Graham et al., 2003). Specifically, we used maximum likelihood estimation method to create ten imputed datasets in Mplus. The data imputation model included all of the observed study variables included in the SEM model. We conducted subsequent analyses with the imputed datasets and the final parameter estimates, standard errors, and goodness-of-fit statistics of the SEM model (representing the average results across 10 imputed datasets) were obtained with the automatic aggregation procedure implemented in Mplus (Rubin, 1987).

Descriptive Statistics and Zero-order Correlations

Table 1 presents sample sizes and descriptive statistics of the study variables by adolescent ethnicity and gender, along with results from independent samples T tests for mean differences across ethnic and gender groups. Parental ADS and positive parenting behaviors were similar between EA and AA adolescents. However, compared to EA adolescents, AA adolescents were less likely to be living with both biological parents and to be engaged in risky drinking, had lower parental education, higher paternal and maternal antisocial behaviors, and more conduct disorder symptoms. Family structure, parental education, and parental ADS were similar between male and female adolescents. However, compared to males, female adolescents reported higher levels of maternal involvement and mother-child communication and lower levels of mother-child closeness. Female adolescents also reported lower rates of heavy episodic drinking drinking and fewer conduct disorder symptoms. Table 2 shows bivariate correlations between variables considered in this study from analysis based on the imputed datasets.

Predicting Adolescent Risky Drinking and Conduct Problems

Results from the multivariate SEM model predicting adolescent risky drinking and conduct problems are presented in Figure 1. The model demonstrated mediocre fit to the data (χ^2 (105) = 720.54; $p < .001$; CFI = .77; RMSEA = .07). Although the chi-square value was statistically significant (which is common for models with large sample size) and the CFI

below typically accepted standards of "acceptable" fit (i.e., .90), the RMSEA was indicative of acceptable model fit (Kline, 2010). We note that relatively low values of the CFI tend to occur in complex models where there are small correlations among variables. Given the small magnitude of several associations in the model (e.g., mothers' ADS were not related to fathers' and their own parenting), a somewhat low CFI is not unexpected. Kenny (2014) suggests that since the CFI is an incremental fit index, CFI may not be a useful indicator of model fit in cases where a null model (no associations specified among the study variables) produces RMSEA values < .158; ours was .102. As such, despite the relatively low CFI, the overall model fit statistics suggest acceptable fit.

Standardized coefficients for the structural paths are presented in Figure 1. Consistent with the hypothesis, fathers' ADS were negatively associated with fathers' and mothers' positive parenting behaviors. Mothers' positive parenting, in turn, was negatively associated with adolescents' risky drinking whereas fathers' positive parenting was negatively associated with adolescents' conduct problems. Contrary to prediction, neither fathers' nor mothers' ADS were directly associated with adolescents' risky drinking and conduct problems. However, the indirect effect of fathers' ADS on adolescents' conduct problems via fathers' positive parenting was statistically significant (ab = .021, SE = .008, p = .008). There was also an indirect effect (albeit marginally significant) of fathers' ADS on adolescents' risky drinking via mothers' positive parenting (ab = .010, SE = .006, p = .067). Mothers' ADS was not associated with their own or fathers' positive parenting behaviors. Thus, there was no direct or indirect association (via mothers' or fathers' positive parenting) between mothers' ADS and adolescents' risky drinking and conduct problems.

Ethnic Differences in Pathways of Risk

Results from MGCFA suggested the measurement model allowing factor loadings to be freely estimated across racial groups (χ^2 = 31.66, df = 10, CFI = .986) and the model constraining factor loadings to equality across racial groups (χ^2 = 33.91, df = 14, CFI = .987) were not significantly different in terms of model fit ($\Delta\chi^2$ = 2.25, Δdf = 4, p = .69, ΔCFI = .001), suggesting that factor loadings were invariant (i.e., metric invariance) across the EA and AA groups in the measurement model assessing fathers' and mothers' positive parenting. The Wald test for simultaneously constraining all of the key path coefficients in the model to equality across ethnic groups was not significant ($\Delta\chi^2$ = 14.69, Δdf = 12, p = .26), suggesting that there were no ethnic differences in these path coefficients.

Gender Differences

Results from MGCFA indicated the measurement model allowing factor loadings to be freely estimated across gender groups (χ^2 = 36.57, df = 10, CFI = .983) and the model constraining factor loadings to equality across gender groups (χ^2 = 48.26, df = 14, CFI = .979) were not significantly different in terms of model fit ($\Delta\chi^2$ = 12.01, Δdf = 4, p = .02, ΔCFI = −.004), suggesting that factor loadings were also invariant across the gender groups in the measurement model assessing fathers' and mothers' positive parenting. Multi-group SEM analysis indicated no significant differences in path coefficients across gender ($\Delta\chi^2$ = 9.00, Δdf = 12, p = .70). This suggested that the patterns of associations among the various constructs in the model were similar between adolescent males and females.

Follow-up Analysis

We conducted follow-up analyses to check the robustness of our results. As noted above, there was a relatively large amount of missing data for parental assessments of ADS in our sample. We conducted additional analysis with the subsample (n = 706) of adolescents having complete data on both parents' ADS, in order to examine whether the patterns of association are consistent or biased due to missing data. The overall pattern of associations was similar to results from analysis using the imputed datasets for the whole sample. Given that family processes may vary as a function of family structure/living arrangement, we also ran analysis with the subsample (n = 929) of families where adolescents lived with both biological parents to check whether the patterns of association were consistent. The overall pattern of associations was similar to those from the whole sample. Results from these follow-up analyses are not presented but available upon request.

Discussion

Grounded in family systems theory (Cox & Paley, 2003), the present study examined the unique roles and spillover and crossover effects of fathers' and mothers' ADS and parenting behaviors in relation to adolescent risky drinking and conduct problems, measured approximately 2 years after the parenting behaviors were assessed. We further tested for differences in these relationships by adolescents' ethnicity and gender. Findings suggested that fathers' ADS were associated with adolescent outcomes indirectly by disrupting their own (spillover effect) and mothers' (crossover effect) positive parenting behaviors, whereas mothers' ADS were not associated with adolescent outcomes either directly or indirectly via parenting behaviors. We found no ethnic or gender differences in these associations.

Consistent with hypothesis and prior research (e.g., Chassin et al., 1993, Ohannessian, 2012), our findings suggest that parenting behaviors serve as a mediating pathway for the influence of fathers' ADS on adolescent risky drinking and conduct problems. Our findings revealed unique patterns of spillover and crossover effects for fathers and mothers. Consistent with the family systems perspective, fathers' ADS not only undermine their own positive parenting behaviors but also disrupt mothers' parenting behaviors. Perhaps fathers' ADS create high levels of strain and emotional tension in the home and/or impair the quality of relationship with the adolescents' mother (Kachadourian et al., 2009; Leonard, & Eiden, 2007), which in turn was associated with higher adolescent risky drinking and conduct problems. It is also likely the case that when the father exhibited ADS, the mother would need to compensate by increasing her responsibilities and duties (e.g., doing some of the fathers' duties), which may result in additional stress and time involvement that could potentially undermine mothers' positive parenting. In contrast, mothers' ADS did not negatively influence their own or fathers' parenting behaviors. Perhaps mothers are more likely to maintain positive parenting behaviors such as involvement and communication, even in the context of ADS, given that most mothers have primary caregiving roles in the family.

Consistent with expectations, our bivariate correlations analyses indicated that both fathers' and mothers' ADS were correlated with higher likelihood of adolescent risky drinking and more conduct problems. However, results from our multivariate SEM model indicated that

neither fathers' nor mothers' ADS were directly associated with these adolescent outcomes. These findings suggest that parental ADS may influence adolescent outcomes indirectly rather than directly, reiterating the importance of understanding the mediating pathways through which parental ADS exert an effect on adolescent adjustment. Findings from this study suggest parenting behaviors as an important mechanism through which fathers' ADS play a role in adolescents' risky drinking and conduct problems. Although our findings indicate no indirect effect of mothers' ADS on these adolescent outcomes via parenting behaviors, we note the possibility that mothers' ADS may influence adolescent outcomes via other mechanisms such as increased adolescents' environmental stress and negative affect (Chassin et al., 1993) and more family conflict (Loukas et al., 2001). It is also likely that mothers' ADS influence other aspects of parenting such as monitoring, discipline, or negative parenting practices that were not considered in our model, which may in turn affect adolescent outcomes. For example, Chassin and colleagues (1996) showed that mothers' alcohol dependence was associated with lower maternal and paternal monitoring, which in turn was associated with higher adolescent substance use. Additional research is needed to further understand the pathways by which mothers' alcohol problems may impact adolescent outcomes.

Interestingly, results from our multivariate SEM model indicated that fathers' but not mothers' positive parenting was associated with fewer adolescent conduct problems, whereas mothers' but not fathers' positive parenting was associated with adolescents' lower likelihood of engaging in risky drinking. This is unexpected, given that both paternal and maternal parenting behaviors have been shown to be associated with risky drinking and externalizing problems in adolescents (Leidy et al., 2011; Ryan et al., 2010). In our study, when paternal and maternal parenting behaviors were analyzed in separate models (post-hoc analysis, see supplemental Figures 1 and 2), both were indeed significantly associated with adolescent risky drinking and conduct problems, as also indicated by the bivariate correlations between these variables. Our model where fathers' and mothers' parenting analyzed simultaneously took into account the variances shared between fathers and mothers and provided an examination of the unique effects of fathers' and mothers' parenting. It is interesting that results from the fathers and mothers combined model and those from the analyses conducted separately for fathers and mothers were somewhat different. This suggests that while some of the effects of fathers' and mothers' parenting on adolescents' risky drinking and conduct problems exert through processes shared between fathers and mothers within the family, mothers' parenting seems to have an extra unique effect on adolescents' risky drinking while fathers' parenting has an extra unique effect on adolescents' conduct problems. Future research is needed to replicate these findings.

Pathways of risk did not vary across European American and African American adolescents, suggesting the important role of fathers' ADS and its spillover and crossover effects between ADS and parenting behaviors across ethnic groups. This is consistent with some prior studies showing no ethnic differences in the associations between parental problem drinking, parenting, and adolescent outcomes (e.g., Shorey et al., 2013), but contradicts other studies that found the associations between parenting behaviors and adolescent outcomes to vary across ethnicity (e.g., Clark et al., 2015). Overall, the path coefficients linking parental ADS and parenting behaviors to adolescent risky drinking and conduct problems also did not vary

across adolescent gender. These findings are consistent with some prior studies that found no difference in the associations between fathers' and mothers' alcohol dependence or problem drinking and adolescent psychopathology for sons and daughters (Lieb et al., 2002; Ohannessian et al., 2005), but contradict others that suggested stronger influence of fathers' alcoholism on alcohol abuse for daughters than for sons (Morgan et al., 2010), or stronger associations between same-sex parent-adolescent dyads (Ohannessian et al., 2012). Future research is needed to further understand the role of ethnicity and gender in the association between parental alcohol problems and adolescent outcomes.

Limitations

Our findings need to be interpreted in light of several limitations. First, the assessment of parental ADS generally occurred years prior to the adolescent interview, and there is considerable variability in adolescents' age at the time of assessment of parental ADS (from 1 to 15 years, $M = 7.56$, $SD = 3.13$). For the majority of the sample, we did not have data on parental ADS at the time when parenting and adolescent outcomes variables were measured, and thus did not know whether or not parental ADS were still present at the assessment of parenting behaviors and adolescent outcomes. Thus, it was not possible to determine whether or how long adolescents were exposed to parental ADS. We note, however, that both concurrent and history of parental alcohol problems have been shown to be associated with negative parenting behaviors and psychosocial outcomes in offspring (Conway et al., 2004; Schepis et al., 2008). Prior research also suggests that parental alcohol dependence and related externalizing disorders have long-term negative influence on offspring alcohol problems, regardless of timing of parental exposure (Edwards et al., 2017). Parental alcohol dependence (either past or concurrent), in part, represent parents' genetic predispositions toward risky and problematic behaviors and/or stable individual characteristics (e.g., personality traits), which can play an important role in influencing their parenting behaviors and child outcomes. In addition, adult alcohol dependence tends to be relatively stable across time (Chassin et al., 2004), and our post hoc analyses indicated that adding adolescents' age at the assessment of parental ADS as a covariate did not change the pattern of results.

Second, we only focused on positive parenting because our measures of negative parenting were weak (only included a few items assessing strictness and spanking), only available for a subset of the sample, and lacked adequate reliability. However, parenting is a multidimensional construct, and positive and negative parenting may have differential effects on offspring outcomes (Dallaire et al., 2006). Thus, future studies should consider additional dimensions of parenting to further understand the effect of parental ADS on adolescent outcomes via parenting. In addition, although we used a latent variable approach to measure positive parenting that capitalized on multiple dimensions of positive parenting behaviors and helped account for measurement error, we acknowledge that some of the factor loadings were low (e.g., .57 for maternal involvement). This limitation regarding the measurement of positive parenting may have attenuated the effects observed in our analysis.

The large amount of missing data for fathers is another limitation. We note, however, that there was no significant difference in key variables between adolescents who had missing

data for fathers and those without missing data for fathers. Our follow-up analysis indicated that the patterns of associations are largely similar between analysis with the subsample of adolescents having complete parental data and analysis with the whole sample. Thus, our findings were unlikely to be biased due to missing data. Despite the degree of missingness, which we accounted for using multiple imputation, the availability of data for both fathers and mothers allowed us to examine spillover and crossover effects within the family in the context of parental ADS, which also contributes to the relatively small literature on the role of fathers in adolescent development.

Another limitation is that we only included EA and AA adolescents in this study. Future research needs to examine whether the pattern of associations observed here are generalizable to other ethnic groups. Because our sample is highly enriched both for alcohol dependence and larger families, replication of our findings in community or nationally representative samples will be important. Further, we only examined the role of parental ADS on adolescent risky drinking and conduct problems. Since parental alcohol problems affect a range of offspring outcomes, future research is warranted to extend the current investigation to other adolescent outcomes such as internalizing problems. Finally, we did not consider the role of prenatal exposure to alcohol because relevant data was only available to a small subset of adolescents in our sample. Given that prenatal exposure to alcohol has implications for later development (e.g., Riley et al., 2011), future research is needed to examine whether or not the pattern of association between parental ADS, parenting, and adolescent outcomes may differ for prenatally alcohol exposed and non-exposed adolescents.

Conclusion

Despite several limitations, this study has many strengths, including the clinical assessment of ADS from fathers and mothers, the family systems approach to consider the spillover and crossover effects of fathers' and mothers' ADS and parenting behaviors, prospectively measured parenting behaviors and adolescent outcomes, rigorous tests of ethnic and gender differences in pathways of risk, and careful consideration of a number of important covariates. Our results suggest that fathers' ADS put adolescents at risk for risky drinking and conduct problems, above and beyond the effects of parenting, parental education, parental antisocial personality disorder and internalizing problems, family structure, and adolescents' age, gender, and ethnicity. Disrupted fathers' and mothers' parenting behaviors such as lower involvement and poor parent-child communication may serve as indirect pathways of risk associated with fathers' ADS. Our findings emphasize the importance of considering the unique roles of fathers' and mothers' ADS in influencing family processes and adolescent outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The Collaborative Study on the Genetics of Alcoholism (COGA), Principal Investigators B. Porjesz, V. Hesselbrock, H. Edenberg, L. Bierut, includes eleven different centers: University of Connecticut (V. Hesselbrock); Indiana University (H.J. Edenberg, J. Nurnberger Jr., T. Foroud); University of Iowa (S. Kuperman, J. Kramer); SUNY Downstate (B. Porjesz); Washington University in St. Louis (L. Bierut, J. Rice, K. Bucholz, A. Agrawal); University of California at San Diego (M. Schuckit); Rutgers University (J. Tischfield, A. Brooks); Department of Biomedical and Health Informatics, The Children's Hospital of Philadelphia; Department of Genetics, Perelman School of Medicine, University of Pennsylvania, Philadelphia PA (L. Almasy), Virginia Commonwealth University (D. Dick), Icahn School of Medicine at Mount Sinai (A. Goate), and Howard University (R. Taylor). Other COGA collaborators include: L. Bauer (University of Connecticut); J. McClintick, L. Wetherill, X. Xuei, Y. Liu, D. Lai, S. O'Connor, M. Plawecki, S. Lourens (Indiana University); G. Chan (University of Iowa; University of Connecticut); J. Meyers, D. Chorlian, C. Kamarajan, A. Pandey, J. Zhang (SUNY Downstate); J.-C. Wang, M. Kapoor, S. Bertelsen (Icahn School of Medicine at Mount Sinai); A. Anokhin, V. McCutcheon, S. Saccone (Washington University); J. Salvatore, F. Aliev, B. Cho (Virginia Commonwealth University); and Mark Kos (University of Texas Rio Grande Valley). A. Parsian and M. Reilly are the NIAAA Staff Collaborators. We continue to be inspired by our memories of Henri Begleiter and Theodore Reich, founding PI and Co-PI of COGA, and also owe a debt of gratitude to other past organizers of COGA, including Ting-Kai Li, P. Michael Conneally, Raymond Crowe, and Wendy Reich, for their critical contributions. This national collaborative study is supported by NIH Grant U10AA008401 from the National Institute on Alcohol Abuse and Alcoholism (NIAAA) and the National Institute on Drug Abuse (NIDA). This research was also support in part by National Institute of Health grants K02AA018755 (PI: Dick) and K01AA024152 (PI: Salvatore)

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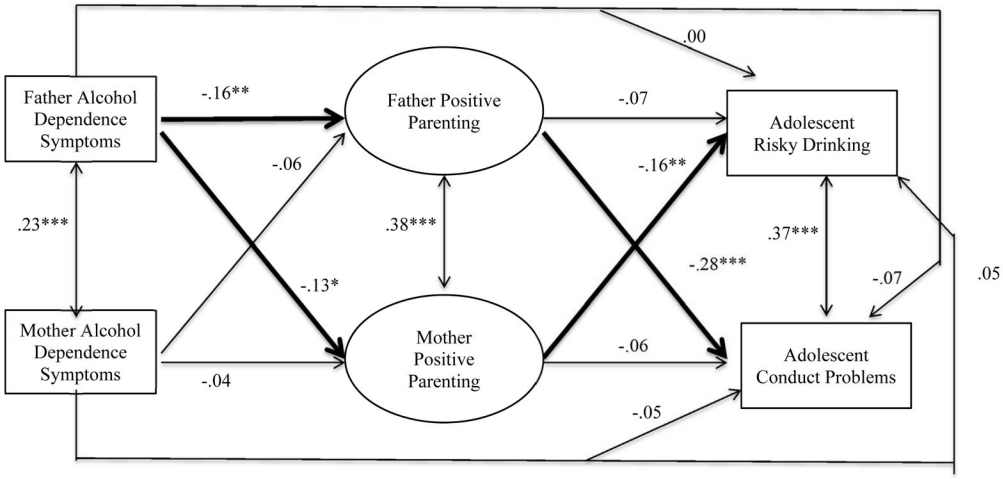

Figure 1.

Predicting Adolescent Risky Drinking and Conduct Problems from Parental Alcohol Dependence Symptoms and Parenting Behaviors

Note. Standardized path coefficients are presented. Statistically significant paths are bolded. Adolescents' age, gender, ethnicity, family structure, parental education, and paternal and maternal antisocial behaviors and internalizing problems were included as covariates for adolescent risky drinking and conduct problems. * $p < .05$, ** $p < .01$, *** $p < .001$. N = 1282.

Table 1

Descriptive Statistics of the Study Variables by Adolescent Ethnicity and Gender

	Ethnicity								Gender							
	European American			African American					Male			Female				
	N	Mean	*SD*	N	Mean	*SD*	t/χ^2 (*df*)	*p*	N	Mean	SD	N	Mean	*SD*	t/χ^2 (*df*)	*p*
A Age	886	13.00	1.14	396	12.97	1.11	−.38	.71	639	13.01	1.14	643	12.97	1.12	−.52	.60
Family structure [a]	885	.79	--	396	.57	--	66.67 (1)	< .01	639	.73	--	642	.72	--	.04 (1)	.84
Parental education	864	13.75	1.96	340	11.96	1.73	−14.67	< .01	600	13.36	2.08	604	13.13	2.05	−1.87	.06
F ASB	629	2.43	1.93	175	3.19	2.04	4.57	< .01	404	2.71	1.98	400	2.47	1.97	−1.71	.09
M ASB	819	1.37	1.53	360	2.20	1.93	7.31	< .01	581	1.67	1.71	598	1.58	1.70	−.93	.35
F INT	567	1.43	2.30	130	1.61	2.42	.79	.43	347	1.51	2.38	350	1.42	2.26	−.51	.61
M INT	759	2.17	2.55	276	2.17	2.41	.02	.98	509	2.37	2.60	526	1.97	2.41	−2.56	.01
F AD symptoms	630	2.47	2.43	175	2.42	2.45	−.24	.81	403	2.60	2.45	402	2.32	2.41	−1.65	.10
M AD symptoms	819	1.61	2.02	358	1.57	2.20	−.31	.76	583	1.63	2.13	594	1.56	2.02	−.60	.55
F involvement	710	3.55	1.34	199	3.50	1.36	−.51	.61	464	3.62	1.33	445	3.46	1.36	−1.85	.07
M involvement	848	4.26	1.05	351	4.24	1.01	−.33	.74	595	4.16	1.07	604	4.37	1.00	3.31	< .01
F-A communicate	710	2.07	1.02	199	2.05	1.01	−.30	.77	464	2.08	1.04	445	2.06	1.00	−.32	.75
M-A communicate	848	2.30	.92	351	2.38	.87	1.36	.17	595	2.17	.97	604	2.47	.81	5.77	< .01
F-A closeness	710	.15	.75	199	.06	.97	−1.19	.23	464	.15	.80	445	.12	.80	−.44	.66
M-A closeness	848	.09	.77	351	.06	.82	−.56	.58	595	.14	.69	604	.03	.86	−2.36	.02
A HED [b]	868	.18	--	391	.06	--	34.32 (1)	< .01	627	.16	--	632	.12	--	5.00 (1)	.03
A CDSX	885	.48	.86	396	.75	1.00	4.60	< .01	639	.69	1.00	642	.44	.80	−4.80	< .01

Note. A = Adolescent, F = Father, M = Mother, ASB = antisocial behavior, INT = internalizing problems, AD = alcohol dependence, F-A = father-adolescent, M-A = mother-adolescent, HED = heavy episodic drinking, CDSX = Conduct Disorder Symptoms. Family structure was coded 1 = intact family, 0 = non-intact family. Mean values for gender and family structure represent the proportions of adolescents who were male and who came from intact families, respectively. Independent sample t-tests were performed to test for ethnic/gender differences in mean levels of quantitative variables.

[a] Chi-square tests were performed to test for ethnic/gender differences in categorical variables.

[b] Proportions of adolescents who engaged in heavy episodic drinking in the past year are reported (instead of mean).

Table 2

Bivariate Correlations Between Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. A Age	--																	
2. A Gender	.02	--																
3. Ethnicity	.01	.02	--															
4. Fam struc	−.05	.01	.23	--														
5. Par Educ	−.05	.05	**.40**	.26	--													
6. F ASB	.02	.05	**−.22**	−.31	**−.39**	--												
7. M ASB	.00	.02	**−.22**	−.19	**−.35**	**.42**	--											
8. F INT	.01	.01	−.07	−.21	**−.14**	**.43**	**.23**	--										
9. M INT	−.01	**.08**	.00	−.12	−.08	**.22**	**.34**	**.20**	--									
10. F ADS	**.08**	.06	−.01	−.17	**−.25**	**.61**	**.30**	**.34**	**.14**	--								
11. M ADS	.02	.02	.01	−.12	**−.23**	**.26**	**.51**	**.11**	**.27**	**.31**	--							
12. F involv	**−.10**	.07	.02	−.03	.06	**−.10**	−.07	−.04	−.05	−.06	−.05	--						
13. M involv	**−.10**	**−.10**	.01	.09	**.08**	**−.10**	**−.10**	−.03	−.03	−.07	**−.10**	**.39**	--					
14. F-A com	−.01	.01	.05	.11	**.16**	**−.13**	−.07	−.04	−.06	**−.10**	−.06	**.43**	**.21**	--				
15. M-A com	−.02	**−.16**	−.04	.02	**.08**	**−.09**	−.04	−.02	−.03	−.07	−.01	**.19**	**.36**	**.56**	--			
16. F-A close	**−.17**	.03	**.10**	.05	**.12**	**−.18**	**−.16**	**−.10**	−.07	**−.15**	**−.09**	**.45**	**.09**	**.39**	**.14**	--		
17. M-Aclose	**−.14**	**.07**	.02	.06	**.10**	**−.13**	**−.09**	**−.14**	**−.07**	**−.13**	−.07	**.17**	**.35**	**.15**	**.34**	**.42**	--	
18. A HED	**.36**	**.10**	**.30**	−.03	**−.10**	**.16**	**.10**	**.12**	.06	**.20**	**.15**	**−.13**	**−.21**	−.09	**−.15**	**−.16**	**−.14**	--
19. A CDSX	−.02	**.15**	**−.16**	−.17	**−.27**	**.25**	**.26**	**.16**	**.14**	**.15**	**.12**	**−.18**	**−.12**	**−.17**	**−.11**	**−.27**	**−.15**	**.34**

Note. Coefficients from bivariate correlations analysis with imputed datasets (average results across 10 imputed datasets) are presented. Statistically significant coefficients ($p < .05$) are bolded. A = Adolescent, fam struc = family structure, Par Educ = parental education, F = Father, M = Mother, ASB = antisocial behaviors, INT = internalizing problems, ADS = alcohol dependence symptoms, involve = involvement, com = communication, close = closeness, HED = heavy episodic drinking, CDSX = Conduct disorder symptoms. Gender was coded 1 = male, 0 = female; ethnicity was coded 1 = European American, 0 = African American; family structure was coded 1 = intact family, 0 = non-intact family.

Table 3

Standardized Factor Loadings and Residual Correlations for Measurement Model By Ethnicity and Gender

Construct	Indicator	Whole sample	EA	AA	Male	Female
Fathers' positive parenting	→ Paternal involvement	.68	.70	.70	.61	.75
	→ Father-child communication	.59	.57	.61	.54	.61
	→ Father-child closeness	.66	.65	.68	.72	.64
Mothers' positive parenting	→ Maternal involvement	.57	.60	.52	.40	.69
	→ Mother-child communication	.58	.56	.52	.50	.60
	→ Mother-child closeness	.62	.63	.66	.61	.71
Residual correlations	Paternal with maternal involvement	.40	.38	.35	.42	.35
	Father-child with Mother-child communication	.66	.64	.55	.69	.52
	Father-child with mother-child closeness	.41	.49	.44	.42	.53
	Fathers' with mothers' positive parenting	.38	.40	.42	.45	.37

Note. Standardized factor loadings from confirmatory factor analysis are presented. For the purpose of model identification, (unstandardized) factor loadings for paternal and maternal involvement were fixed to 1. EA = European American; AA = African American. Sample sizes are 886, 396, 639, and 643 for the EA, AA, male, and female subsamples, respectively.

$p < .05$ for all factor loadings

Alcohol Clin Exp Res. Author manuscript; available in PMC 2019 September 01.