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Gender differences in the association between family conflict and adolescent substance use disorders

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Abstract

Purpose—The objectives of this study were: 1) to examine whether the association between childhood family conflict and the risk of substance use disorders (SUD) in adolescence differs by gender, and 2) to determine if anxious/depressive symptoms and conduct problems explain this association among adolescent males and females.

Methods—Data came from 1,421 children aged 10 through 16 when enrolled in the Project on Human Development in Chicago Neighborhoods. We assessed gender differences in the association between childhood family conflict and adolescent SUDs by fitting a logistic regression model that included the interaction of gender and family conflict. We also investigated whether conduct problems and anxious/depressive symptoms explained the association between family conflict and SUDs differently for males and females through gender-specific mediation analyses.

Results—The association between childhood family conflict and SUDs in adolescence differed by gender ($p=0.04$). Family conflict was significantly associated with SUDs among females (OR: 1.61; CI: 1.20, 2.15), but not among males (OR: 1.00; CI: 0.76, 1.32). The elevated risk of SUDs among females exposed to family conflict was partly explained by girls' conduct problems but not anxious/depressive symptoms.

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Conclusions—Females living in families with elevated levels of conflict were more likely to engage in acting out behaviors, which was associated with the development of substance use disorders. Future epidemiologic research is needed to help determine when this exposure is most problematic with respect to subsequent mental health outcomes and the most crucial time to intervene.

Keywords

SUDs; adolescents; family conflict; gender differences; conduct problems; prospective study

INTRODUCTION

Family adversity has been consistently associated with an increased risk for substance use disorders (SUD).(1, 2) However, an unresolved question is whether or not males and females respond differently to this adversity. Gender-specific patterns of responses to family adversity suggest at least two possibilities: 1) the strength of the association between family conflict and SUDs differs by gender; and 2) the psychological mechanisms linking family conflict to SUDs, which can develop as a result of family conflict (3–5) and can increase the risk of substance-related problems,(6) differ between males and females. Empirically, the first possibility is one of effect modification, wherein we would expect to observe a significant interaction between gender and family conflict in prediction models for SUDs. The second possibility concerns mediation of the association between family conflict and SUDs, and suggests that mediation analyses with mental health problems would yield different results between males and females. The current study investigates both of these possibilities.

Scant research has examined the differential impact of familial discord on substance use problems by gender. Furthermore, the limited research on this subject has produced discrepant results as to how males and females ultimately respond to adverse home environments. Some studies report that family conflict is a risk factor for substance-related problems for both males and females,(4, 5) others report this for females only,(3) and yet others report this for males only.(7)

Regarding the hypothesis that different psychological pathways underlie the association between family conflict and SUDs for males and females, there is corroborating evidence in the general stress literature. First, females are at greater risk for internalizing symptoms (e.g., anxiety depression, withdrawal) following exposure to familial stressors such as low socioeconomic status (SES), parental psychopathology, and marital conflict. In contrast, males have an elevated risk of externalizing symptoms (e.g., aggression, delinquency, hyperactivity) following exposure to these same types of stressors.(8–10) However, it is unclear whether the results of these studies generalize to all types of stressors, and specifically to family conflict. Some research has demonstrated that females exposed to family conflict are at greater risk for internalizing problems, whereas males are at greater risk for externalizing problems.(11–15) Alternatively, other research suggests that adverse family environments are associated with conduct problems among females (3) and depression among males.(13)

Second, there is evidence for gender-specific pathways to SUDs. For example, prior studies have shown that SUDs develop following internalizing problems among females and externalizing problems among males.(16) But other research (6) has found that the association between externalizing problems and SUDs are much stronger for females than males.

In a previous publication, we found that exposure to family conflict during childhood was associated with a 22% higher risk of SUDs in late adolescence, and that approximately one-third of this association was explained by higher levels of externalizing problems following exposure to family conflict.⁽¹⁾ We now extend this work by investigating: 1) gender differences in the association between family conflict and SUDs; and 2) gender differences in the pathways underlying the association between family conflict and SUDs. We hypothesize that the association between family conflict and SUDs will be stronger for females compared to males, based on evidence that females are more sensitive to stressors related to the family environment than males,^(17, 18) and that the association between family conflict and SUDs will be explained in part by elevated anxious/depressive symptoms for females and conduct problems for males.

METHODS

Participants and Data

Data come from the Project on Human Development in Chicago Neighborhoods (PHDCN), a large-scale study that examined the influence of families and neighborhoods on youth development.^(1, 19) The PHDCN used a three-stage design to identify and enroll participants in a longitudinal study (Wave I: 1994–95; Wave II: 1997–99; and Wave III: 2000–01), as described elsewhere.⁽¹⁹⁾ The current study included participants from age groups 12 and 15 (those closest in age to 12 or 15 years old). These participants were ages 10–16 at Wave I, 13–19 at Wave II, and 16–22 at Wave III ($n=1,517$). These age groups were chosen because this age range represents children and young adolescents who were living at home at the time of the baseline interview and were at risk for SUDs at Wave III.

Measures

Measures were drawn from all three waves of the Longitudinal Cohort Study of the PHDCN.⁽²⁰⁾ Self-report assessments were administered to both the youth participants and to primary caregivers. Family conflict was assessed at Wave I (baseline), mental health problems were assessed at Wave II, and SUDs were assessed at Wave III.

The dependent variable was any past-year SUD, which included alcohol dependence or marijuana abuse or dependence. In Wave III of the PHDCN, participants in age groups 12 and 15 were administered substance use interviews, which included a series of questions about their past year substance use behaviors. The interviews were adapted from the 1991 National Household Survey on Drug Abuse (NHSDA). The versions differed slightly for the two age groups. The interview administered to those in age group 12 included questions on alcohol and associated problems. Alcohol use disorders were assessed for age group 15 through the Alcohol Use Follow-Up questionnaire, which was adapted from the Diagnostic Interview Schedule (DIS-IV) Alcohol Module. DSM-IV diagnoses of alcohol and marijuana use disorders were formulated in a similar fashion as other published studies.^(21, 22) Individuals who met diagnostic criteria for an SUD at Wave I were excluded from all analyses ($n=96$).

Family conflict in Wave I was assessed with the Conflict subscale of the Family Environment Scale (FES),⁽²³⁾ which was completed by primary caregivers and includes nine dichotomous questions that provide evidence of family conflict. This subscale has been shown to have good internal consistency reliability (Cronbach's $\alpha = 0.72$),⁽²⁴⁾ which was true for the present sample (Cronbach's $\alpha = 0.70$). Each statement that endorsed conflict was given a score of 1, for a total score ranging from 0–9. We standardized this score to a mean of 0 and standard deviation of 1 and used it as an index of family conflict with higher scores reflecting higher levels of conflict.

Hypothesized mediating variables included in the analyses were conduct problems and anxious/depressive symptoms at Wave II measured by the Aggressive Behavior (Cronbach's $\alpha = 0.77$) and Anxious/Depressed (Cronbach's $\alpha = 0.85$) subscales of the Achenbach Youth Self-Report (YSR).(25) Reliability and validity of the YSR have been well established.(25) These measures were standardized to have a mean of 0 and a standard deviation of 1, with higher scores reflecting higher levels of symptoms.

The following variables measured at Wave I were included in the analyses as potential confounders because of their association with either family conflict or the hypothesized mediators and SUDs: history of parental substance use problems, history of parental depression, parental criminal record, physical abuse/punishment, primary caregiver marital status, family structure, and family SES. Age group and race/ethnicity were also controlled for in the analyses.

Analysis

First we examined the demographic, parental, and family characteristics of males and females in the sample separately. Gender-specific bivariate associations were evaluated between SUDs at Wave III and all independent variables using t-tests for continuous variables and chi-square tests of independence for categorical variables.

Next, we fitted a logistic regression model in which the dependent variable was a Wave III SUD and our primary covariate of interest was an interaction term between the family conflict index and gender (males coded as '0' and females as '1'), where both the main effects of family conflict and gender were included in the model. All potential confounders were included in the final model. The gender-specific odds ratios quantifying the association between family conflict and SUDs were obtained from the logistic regression coefficients in the model with the gender x conflict interaction.

We then proceeded with the mediation analyses for males and females. Meditational analyses were conducted using a "multiple mediator models approach".(26) This method assesses the relative effect sizes of aggressive behavior and anxious/depressive symptoms as mediators. This was done by fitting the following models: 1) a linear regression model of Wave II conduct problems on Wave I family conflict; 2) a linear regression model of Wave II anxious/depressive symptoms on Wave I family conflict; and 3) a logistic regression model of Wave III SUDs on Wave I family conflict, which also included Wave II conduct problems and Wave II anxious/depressive symptoms. Conduct problems and anxious/depressive symptoms at Wave I were also included in Models 1–3 to control for prior onset of these problems. Given that Models 1 and 2 are linear regressions and Model 3 is a logistic regression, standardized coefficients from Models 1, 2, and 3 were used in the tests of mediation.(26) All potential confounders were included in each of these models.

To test for mediation, we conducted a product of coefficients test for each hypothesized mediator.(26) The mediated effect for conduct problems is given by the product of the coefficients for family conflict in Model 1 and conduct problems in Model 3. Similarly, the mediated effect for anxious/depressive symptoms is given by the product of the coefficients for family conflict in Model 2 and anxious/depressive symptoms in Model 3.

Statistical significance of the mediated effect was assessed by constructing confidence intervals around each effect, which were calculated by taking the mediated effect and adding/subtracting 1.96 (the Z-score corresponding to a Type I error rate of 5%) times its standard error.(27) Evidence of mediation was supported if the confidence interval did not cover zero or, alternatively, if the mediated effect divided by its standard error was greater than 1.96.(27)

We estimated the relative size of the mediation effect as the proportion of the total effect that was attributable to the mediated effect. This quantity was computed by dividing the mediated effect (i.e., product of coefficients) by the total effect.

Due to attrition between Waves I and III and skipped answers at Wave III, 432 participants did not have complete data at Wave III. Therefore, to decrease the potential for bias that could result from using a complete case analysis,(28) we utilized the Markov Chain Monte Carlo (MCMC) method of multiple imputation.(29) Using the MI procedure in SAS, we generated five multiply imputed datasets, which were analyzed separately, and the regression models were combined with the MIANALYZE procedure to produce a single set of coefficients for each set of models. All covariates in the analyses were included in the multiple imputation computation. For all analyses, including the tests for effect modification and mediation, we used generalized estimating equations (30) to account for the clustering of individuals within neighborhoods, implemented in SAS version 9.1 (SAS Institute Inc, Cary, NC).

RESULTS

The demographic, parental, and family characteristics of males and females in the sample are presented separately in Table 1 for individuals with and without an SUD at Wave III. Additionally, gender-specific bivariate associations between those with and without SUDs at Wave III for each of the characteristics are presented. Compared to females without SUDs at Wave III, females with SUDs had a higher mean family conflict score ($p=0.01$); however compared to males without an SUD at Wave III, males with an SUD did not have a statistically different mean family conflict score ($p=0.22$).

As reported previously,(1) when males and females are analyzed together, higher levels of family conflict at Wave I are associated with an increased risk of SUDs at Wave III: for each one-standard deviation increase in family conflict, the odds of an SUD were 1.22 times higher (95% CI: 1.02, 1.43). When we estimated the main effects of family conflict and gender on the presence of a diagnosis of an SUD at Wave III, we did observe a significant interaction between family conflict and gender ($p=0.04$), and determined that the association between family conflict in childhood and SUDs in adolescence was statistically significant among females (OR: 1.61; CI: 1.20, 2.15), but not among males (OR: 1.00; CI: 0.76, 1.32).

The results of the mediation analyses are presented in Table 2. Among females, conduct disorders partly appeared to mediate the association between childhood family conflict and SUDs in adolescence. Family conflict at Wave I was significantly associated with an increased risk for conduct problems at Wave II ($\beta=0.56$; 95% CI: 0.25–0.88); conduct problems at Wave II were significantly associated with SUDs at Wave III (OR: 1.46; CI: 1.16–1.86); and the relation between family conflict and SUDs was substantially lower in the model that included the mediators (OR: 1.18; CI: 0.90–1.82). Finally, the product of coefficients test was statistically significant (2.54; $p<0.05$). The mediation effect was 0.21 (CI: 0.05–0.37), and the proportion of the overall association between family conflict and SUDs that is mediated by conduct problems was 0.47.

In contrast, there was no evidence for mediation by anxious/depressive symptoms among females. Family conflict in Wave I was not significantly associated with anxious/depressive symptoms at Wave II ($\beta=-0.06$; CI: $-0.47, 0.35$; $p=0.76$), anxious/depressive symptoms at Wave II were not significantly associated with SUDs at Wave III (OR: 1.19; CI: 0.90, 1.57), and the proportion of the overall association mediated by anxious/depressive symptoms was 0.04.

Among males, we did not observe evidence that either conduct problems or anxious/depressive symptoms mediated the relationship between family conflict in childhood and SUDs in adolescence (Table 2). Family conflict at Wave I was not associated with conduct problems at Wave II ($\beta=0.17$; 95% CI: $-0.13, 0.47$; $p=0.83$) or with anxious/depressive symptoms at Wave II ($\beta=-0.05$; 95% CI: $-0.42-0.31$; $p=0.77$).

DISCUSSION

The goals of this study were first to examine whether the association between family conflict in childhood and SUDs in adolescence differed by gender, and then to conduct mediation analyses to test two possible explanations for any observed association between family conflict and SUDs. Our first hypothesis that the effect of family conflict on the development of SUDs in adolescence would be stronger for females compared to males was substantiated. In fact this association was observed for females only.

Our hypothesis that the relationship between family conflict and SUDs would manifest through anxious/depressive symptoms for females and through conduct problems for males was not supported in the current study. Among males, living in families with elevated levels of conflict was not found to be a risk factor for either conduct problems or anxious/depressive symptoms, and furthermore, anxious/depressive symptoms were not associated with later SUDs. Interestingly, while there was a significant association between conduct problems at Wave II and SUDs at Wave III, conduct problems were unrelated to conflict in the home environment.

While anxious/depressive symptoms were not found to mediate the association between family conflict and SUDs among females, conduct problems were. Females living in families with elevated levels of conflict were at an increased risk of conduct problems, which was associated with an increased risk of developing SUDs in adolescence. The relationship between family conflict and conduct problems, as well as the relationship between conduct problems and SUDs, has been well documented.(31, 32) However, these relationships have most often been examined in the context of acting out behaviors among males, and relatively little attention in this same capacity has been applied to females. It is more common that these relationships are explained by internalizing problems, such as depression or anxiety, among females,(8, 10, 33) and our finding is therefore contrary to the expected. One possible explanation for why females but not males exhibit conduct problems in response to family conflict is that there may be differences in how females and males cope with conflict in the home. There are established differences in coping styles between adolescent females and males, where females are more likely to respond to stress by directly avoiding problems (avoidant coping style) compared to males.(17) Accordingly, in response to exposure to violence, avoidant coping styles have been shown to increase the risk of delinquent behavior among female adolescents, but decrease this behavior among males.(17)

A second explanation could be related to gender differences in perceptions of conflict-related stress, in that females and males may be attuned to different stressors. It has been demonstrated that compared to males, females are more sensitive to stressors related to their families,(18) which could contribute to their acting out behaviors. The current study was not able to examine styles of coping or perceptions of conflict, and therefore, further research using measures of coping, such as the Adolescent Coping Process Interview,(34) should be conducted to test this hypothesis.

We found no association between anxious/depressive symptoms and SUDs and therefore no mediation effect of anxious/depressive symptoms among either males or females in the sample. This contrasts with previous research that has demonstrated an association between

anxious/depressive symptoms and SUDs among adolescent females.(8, 16) It could be that anxious/depressive symptoms and SUDs are in fact associated among females in the sample, but not in the direction that we hypothesized, as it is possible for SUDs to precede or develop after anxiety and depression, particularly among females,(35) which was not the focus of the current study. Another possible reason that we did not find an association between anxious/depressive symptoms and SUDs among females could be because co-occurrence at Wave I was controlled for, and mental health symptoms were only measured in this study at Wave II. Alternatively, it is possible that the lack of evidence of the hypothesized mediating pathway was a result of the dichotomous outcome of presence of any SUD. The use of different measures, such as quantity of past month use, could explain the discrepancy between this study and previous research.

The study's findings should be considered within the context of its limitations. First, while the prospective assessment of the associations allowed us to establish the temporality between Wave I exposures, Wave II mediators, and Wave III outcomes, uncontrolled confounding remains an issue. This is particularly important for mediation analyses, which require confounders of the exposure and the mediator to be controlled in order to infer the presence of causal mediation.(36) Second, the PHDCN took place in the city of Chicago, and therefore the ability to generalize to other adolescents may be limited. Third, while the process of multiple imputation that we employed helped to safeguard against selection bias associated with the attrition from Wave I to Wave III, it is possible that participants dropped out for important reasons that were unrelated to the observed data, and therefore could have biased the results of our study. Fourth, a limitation to the analysis is that while the sample was large (n=1,421), the clustered sampling design of the PHDCN and the inclusion of many covariates reduce the effective sample size. Additionally, the power to detect interaction effects is lower than the power to detect main effects, and the mediation analyses were conducted with stratified samples, reducing the sample size even further. Fifth, the family conflict score measures evidence of current conflict in the home, but if the conflict is chronic, which is not indicated by the data, the effect of this exposure on subsequent mental health and SUD outcomes could be underestimated. Another possible limitation is the period when the data were collected (between approximately 1994 – 2001), as rates of substance use and mental health problems between male and female youth have changed over time, (37) as have external influences that have been shown to differentially predict these problems by gender, such as the increased use of technology (e.g., social networking websites, online gaming).(38) Finally, this paper examined only two potential mediators of the association between family conflict in childhood and SUDs in adolescence; however, various other possible mechanisms that could help explain gender differences, including for example, differences in vulnerability or resilience, were not examined.

In the context of these limitations, this research helps to identify a potential pathway underlying the association between family conflict and adolescent SUDs among females. First, among a large group of adolescents who were sampled from an urban population, we found that females but not males living in families characterized by elevated levels of conflict in childhood were more likely to engage in acting out behaviors, which was associated with the development of SUDs in adolescence. This could be due to differential coping mechanisms employed by males and females related to family conflict, as females may be more susceptible to vulnerabilities at home whereas males may be more susceptible to vulnerabilities within their peer groups.(39) Second, among females, we found no mediating effect of anxious/depressive symptoms, which is contrary to what we had expected. However, this may be related to the timing of the problems (e.g., if SUDs occur prior to the onset of anxious/depressive symptoms).(6)

The results of this study could help identify important points of intervention and future research. Practitioners who are working with children and adolescents whose families are characterized by conflict, particularly females, should consider focusing on coping skills, as prior studies have demonstrated that adaptive coping skills have been effective in mitigating the effect of family conflict,(40) which could prevent or decrease conduct problems, and ultimately substance use disorders. Future research is needed to determine whether there are critical and/or sensitive periods for the exposure of family conflict, which would indicate 1) when this exposure is most problematic with respect to subsequent mental health outcomes, and 2) the most crucial time to intervene.

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Sample characteristics, stratified by gender and substance use disorders (SUD) – 1,421 participants in the Project on Human Development in Chicago Neighborhoods.

Table 1

	Females (n=725) n (%) or Mean (SD)		Males (n=696) n (%) or Mean (SD)		p
	No SUD (n=616)	SUD (n=109)	No SUD (n=522)	SUD (n=174)	
Wave I Demographics					
Race/Ethnicity					0.11
Hispanic	271 (44.0)	46 (42.2)	245 (46.9)	73 (42.0)	0.72
Black	242 (39.3)	41 (37.7)	183 (35.1)	66 (37.9)	
White	78 (12.7)	16 (14.7)	74 (14.2)	31 (17.8)	
Other	25 (4.0)	6 (5.5)	20 (3.8)	4 (2.3)	
Age Group					0.26
12	353 (57.3)	59 (54.1)	314 (60.2)	85 (48.9)	<0.01
15	263 (42.7)	50 (45.9)	208 (39.8)	89 (51.1)	
Wave I Parental and Family Characteristics					
Familial conflict					
Mean: Continuous measure ¹	2.59 (1.9)	3.15 (2.1)	2.54 (1.9)	2.75 (1.9)	0.22
Family structure					0.23
Two parent household	396 (64.3)	72 (66.0)	323 (61.9)	114 (65.6)	
One parent household	176 (28.6)	33 (30.3)	168 (32.2)	49 (28.2)	
Living with guardian (not parent)	44 (7.1)	4 (3.7)	31 (5.9)	9 (5.2)	
Caregiver marital status					0.52
Married	335 (54.4)	62 (56.9)	300 (57.5)	88 (50.6)	
Single	210 (34.0)	34 (31.2)	172 (33.0)	58 (33.3)	
Partnered	71 (11.6)	13 (11.9)	50 (9.5)	28 (16.1)	
Average family SES score ²	-0.27 (1.4)	0.05 (1.3)	-0.18 (1.4)	0.18 (1.4)	<0.01
Parental history of substance use					0.29
No	520 (84.4)	87 (79.8)	451 (86.4)	141 (81.0)	0.86
Yes	96 (15.6)	22 (20.2)	71 (13.6)	33 (19.0)	
Parental history of depression					0.36
No	530 (86.0)	98 (89.9)	466 (89.3)	138 (79.3)	0.38

	Females (n=725) n (%) or Mean (SD)			Males (n=696) n (%) or Mean (SD)		
	No SUD (n=616)	SUD (n=109)	p	No SUD (n=522)	SUD (n=174)	p
Yes	86 (14.0)	11 (10.1)		56 (10.7)	36 (20.7)	
Parental criminal record			0.67			0.45
No	540 (87.7)	97 (89.0)		466 (89.3)	153 (87.9)	
Yes	76 (12.3)	12 (11.0)		56 (10.7)	21 (12.1)	
History of parent-child physical abuse (Wave I)			0.82			0.15
No	386 (62.7)	68 (62.4)		314 (60.2)	111 (63.8)	
Yes	230 (37.3)	41 (37.6)		208 (39.8)	63 (36.2)	
<u>Participant Mental Health</u>						
Mean aggressive behavior score ^I (SD)	5.93 (4.7)	6.54 (5.2)	<0.001	5.06 (3.3)	6.05 (8.9)	<0.01
Mean anxious/depressive symptoms score ^I (SD)	5.17 (3.5)	7.04 (4.6)	0.14	4.48 (3.80)	4.85 (3.82)	0.26

^I Score prior to standardization

² Family SES is a standardized composite of parental education, income, and occupation, derived through principal components analysis, with a mean of 0 and a standard deviation of 1.

Results of mediation analyses of the association between familial conflict in childhood and substance use disorders in adolescence among females and males.

Table 2

Mediator	Linear regression models of the association between family conflict at Wave I and conduct problems and anxious/depressive symptoms at Wave II ^a		Logistic regression models of the association between conduct problems and anxious/depressive symptoms at Wave II and substance use disorders at Wave III ^b	
	β (95% CI)	SE	OR (95% CI)	SE
Females				
Conduct Problems	0.56 (0.25–0.88) **	0.16	1.46 (1.16, 1.86) **	0.12
Anxious/Depressive Symptoms	−0.06 (−0.47, 0.35)	0.21	1.19 (0.90, 1.57)	0.13
Males				
Conduct Problems	0.17 (−0.13, 0.47)	0.15	1.48 (1.13, 1.93) **	0.13
Anxious/Depressive Symptoms	−0.05 (−0.42, 0.31)	0.18	0.98 (0.76, 1.23)	0.12

* $p < 0.05$;
 ** $p < 0.01$

β indicates linear regression coefficient; OR, odds ratio; CI, confidence interval; SE, standard error

^a Results of four linear regression models are shown, in which the association between family conflict and each hypothesized mediator is estimated, separately for females and males. All models controlled for the covariates listed in Table 1.

^b Results of two logistic regression models are shown, in which the independent associations between the hypothesized mediators and substance use disorders are estimated in the same model; separate models were fitted for females and males. Both models controlled for the covariates listed in Table 1.