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A Prospective Study of Familial Conflict, Psychological Stress, and the Development of Substance Use Disorders in Adolescence

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Abstract

Background—Exposure to adverse family environments in childhood can influence the risk trajectory for developing substance use disorders in adolescence. Evidence for this is largely based on cross-sectional studies which have been unable to establish the temporality of this association and investigate underlying pathways.

Methods—The sample consisted of 1,421 adolescents from the Project on Human Development in Chicago Neighborhoods, a three-wave longitudinal study conducted between 1994 and 2001 that followed children from ages 10 through 22. Logistic regression analyses with multiple imputation were conducted to examine the relation between familial conflict in childhood and substance use disorders in late adolescence and emerging adulthood. We conducted mediational analyses to determine if internalizing and externalizing problems explain this relationship, and we investigated whether external social support mitigates the adverse effects of familial conflict on the development of substance use disorders.

Results—Familial conflict was significantly associated with the risk of substance use disorders during adolescence (Odds Ratio: 1.23; 95% CI: 1.02–1.47), and 30% of this effect was due to higher levels of externalizing problems (but not internalizing problems). External social support in childhood did not buffer the effects of familial conflict on substance use disorders during adolescence.

Conclusion—Exposure to familial conflict early in life increases the risk of substance use disorders during late adolescence and emerging adulthood, due partly to higher levels of externalizing problems, but not internalizing problems. Future research is needed to identify additional pathways underlying this association, and the extent to which these pathways are modifiable.

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Keywords

Substance use disorders; Adolescent; Familial conflict; Psychological stress; Social support; Prospective study

1. Introduction

The adverse consequences of adolescent substance use disorders (SUD) include an increased likelihood of sexual risk-taking (i.e., earlier sexual debut, multiple partners, and unprotected sex), driving while intoxicated, and delinquency (Cook et al., 2006; Dembo et al., 1991). Moreover, given the high rate of persistence of SUDs into adulthood (Bauman and Phongsavan, 1999; Grant and Dawson, 1997; Riala et al., 2004), the health effects of alcohol and marijuana use, including liver, respiratory, and lung function problems become cumulatively more harmful over the course of many years (Meyerhoff et al., 2005; Moore et al., 2005; Taylor et al., 2002). Identifying points of intervention to reduce risk of onset would therefore have both immediate and long term public health benefits.

A child's family environment has been recognized as one of the most significant influences on a variety of subsequent behavioral outcomes, including substance use problems in adolescence (Bray et al., 2001; Johnson and Pandina, 1991; Nation and Heflinger, 2006). Family environments with high levels of adversity, including violence, stress, parental drug use, ineffective communication and discipline, and poor sibling relationships, have been linked to adolescent drug use (Vakalahi, 2001; Madu and Matla, 2003). In one of the largest scientific research studies examining the relationship between adversity in childhood and behavioral outcomes later in life, the Adverse Childhood Experiences (ACE) study investigated the effects of abuse, neglect, and household dysfunction on substance use problems in adolescence and adulthood. Using retrospective reports, findings indicated that exposure to such adverse family environments was significantly associated with alcohol use initiation in early adolescence (prior to age 14) and in mid-adolescence (between ages 15 and 17) (Dube et al., 2006), alcohol abuse in adulthood (Dube et al., 2002) and illicit drug use problems in adolescence (Dube et al., 2003). These studies also found a graded relationship between adverse childhood experiences and substance use problems, in that a higher number of adverse childhood experiences was associated with an increasing risk for alcohol and illicit drug problems.

These key studies have elucidated the importance of investigating the long-term effects of adverse family environments on subsequent substance use pathology; however, in these studies, both reports of childhood adversity and adolescent substance use were obtained retrospectively, which could, as a result of possible recall bias, limit the accuracy of the results. This highlights the need for these relationships to be investigated prospectively.

The ACE studies suggest that a possible mechanism for the relationship between adverse experiences in childhood and later substance use is that adolescents use substances as a maladaptive means of coping with stress induced by adverse family environments in childhood. In one of the first studies testing the social stress model of substance abuse among a sample of urban adolescents, Rhodes and Jason (1990) demonstrated that poor family environments (i.e., poor parental relationships, a high degree of family problems) were significantly associated with a higher level of drug use. This prompted the argument that examining stress is a critical component in the development of SUDs and that future research should examine this relationship prospectively. Few studies have revisited this pathway to test the social stress model by directly examining the effects of familial conflict in childhood on SUDs in adolescence through the mechanism of psychological stress. The association between adverse family environments and indicators of psychological stress has been substantiated, as in a recent

study, Shelton and Harold (2008) found that inter-parental conflict was significantly associated with both internalizing and externalizing problems over a one-year period among a cohort of 387 children aged 11 to 13 living in the United Kingdom. Additionally, indicators of psychological stress have been found to predict the development of substance use disorders. For example, in a birth cohort of 1,265 children in New Zealand, conduct problems in childhood and adolescence were associated with an increased risk of substance use disorders in early adulthood (Fergusson et al., 2007). However, few studies (Knight et al., 1998) have combined these two pathways to determine if family conflict leads to substance use disorders through the mechanism of psychological stress, and furthermore, we are not aware of any studies that have attempted to quantify the extent to which the relationship between family conflict and adolescent substance use disorders is attributable to psychological stress.

The present study is motivated by the Transactional Model of Stress and Coping (TMSC), a framework for understanding ways of coping with stressful situations. Within this framework, stressful experiences are seen as person-environment transactions, and the way in which individuals appraise stressors directly contributes to their method of coping (Wenzel et al., 2002). Individual control over stressors is a key component of this model, where lower control is associated with worse coping strategies. This study postulates that since children have little control over their family situation, living in a stressful family environment fosters maladaptive coping, including substance misuse. Additionally, within the TMSC framework, social support (i.e., the help individuals receive from members of their social network) is seen to have "stress-buffering" effects on health behaviors (Cohen and Wills, 1985). Therefore, we also hypothesize that social support received outside of the immediate family (because the stress is coming from within the family) will buffer the effects of familial conflict on subsequent SUDs in late adolescence.

More broadly, the study's conceptual model is framed within the context of life course epidemiology, examining late childhood as a sensitive period, a developmental period in which certain exposures can have a greater effect on the risk of subsequent outcomes compared to other periods in the life course (Kuh et al., 2003). This period is important in the etiologic understanding of SUDs as a way of coping with psychological stress. Coping skills are learned in childhood, and while it is possible to refine these competencies later in life, if pro-social coping mechanisms are not learned, the risk for maladaptive coping (such as substance use) increases dramatically (Molina et al., 2005).

The objective of this study is to investigate whether familial conflict during childhood increases the risk of adolescent SUDs, and if so, whether this increased risk is due to higher levels of psychological stress, and whether this risk can be mitigated by social support. We sought to overcome the limitations of prior research by: (1) using longitudinal data to establish the temporal ordering of the relationship between familial conflict and the development of SUDs in adolescence; and (2) quantifying the extent to which this relationship is attributable to psychological stress. We hypothesize that: (1) familial conflict during childhood will increase the risk of developing a substance use disorder during adolescence; (2) familial conflict in childhood will lead to increased symptoms of psychological stress (i.e., internalizing or externalizing problems), which will in turn increase the risk of developing SUDs in adolescence; and (3) support received outside of the immediate family in childhood will buffer the effects of familial conflict on the development of SUDs in late adolescence.

2. Methods

2.1. Sample and Data

This study utilized data from the Project on Human Development in Chicago Neighborhoods (PHDCN)(Earls et al., 2002), a large-scale study that examined the influence of families and

neighborhoods on child and adolescent development (Sampson et al., 1997). The longitudinal component of the PHDCN followed participants for seven years over three waves of data collection. The PHDCN used a three-stage design to identify and enroll participants in the longitudinal study. In the first stage, a stratified probability sample of 80 neighborhoods was selected from 343 enumerated neighborhood clusters. Neighborhood clusters were aggregates of census tracts, as described by Earls and Buka (1997). Second, 20 block groups were selected at random from each of the 80 neighborhoods. Third, a systematic random sample of residences was drawn for the sampled block groups and all households in each block group (approximately 40,000 in total) were enumerated and screened for the presence of age-eligible children. A total of 8,347 eligible children, adolescents, and young adults were identified, and subsequently grouped into 7 age cohorts (within 6 months of birth and aged 3, 6, 9, 12, 15, and 18 years). Of the 8,347 children identified, 74.6% were enrolled (n=6,228) and interviewed, along with their primary caregivers, between 1994–1997. The follow-up rates were 85.9% at Wave II, conducted between 1997–1999 (n=5,338 of 6,212 children remaining), and 78.2% at Wave III, conducted between 2000–2001 (n=4,850 of 6,203 children remaining).

The current study included all participants from cohorts 12 and 15 (ages 10 to 16 upon enrollment; n=1,517). These cohorts were selected because they would likely represent children and young adolescents who were still living at home at the time of the interview for a study that focused on the exposure of the home environment, and at an age when they were at risk for initiating experimentation with drug use.

2.2. Measures

Measures were drawn from all three waves of the Longitudinal Cohort Study. Familial conflict and social support were assessed at Wave I (baseline) when the participants were aged 10 through 16; psychological stress was assessed at Wave II; and substance use disorders were assessed at Wave III, when participants were older adolescents and emerging adults (Arnett, 2000) (aged 15 through 22). Therefore, the study design allows the temporality of the hypothesized risks for substance use disorders to be established.

2.2.1. Primary outcome: Substance use disorder at Wave III—Substance use disorder was defined as any DSM-IV (American Psychiatric Association, 1994) diagnosis of alcohol dependence or marijuana abuse or dependence in the past 12 months. The measure of substance use disorders was adapted by PHDCN investigators from the 1991 National Household Survey on Drug Abuse (Chen et al., 1997; Kandel et al., 1997; Reardon and Buka, 2002). Chen et al. (1997) and Kandel et al. (1997) have demonstrated that these assessments provide valid approximations of DSM-IV diagnoses of abuse and dependence. Alcohol abuse was not analyzed in view of criticisms that the threshold for alcohol abuse in the DSM-IV is too low to be considered pathological (de Bruijn and van den Brink, 2005; Hasin, 2003).

2.2.2. Familial conflict at Wave I—The Family Environment Scale (FES) (Moos and Moos, 1986), originally designed to understand the social and environmental characteristics of families, was used to assess familial conflict. The FES was completed by primary caregivers. Familial conflict was measured using the conflict subscale of the FES, which consists of nine ‘true’ or ‘false’ items capturing overt familial conflict, including statements such as, “We fight a lot in our family,” and “Family members sometimes hit each other,” as well as reverse coded statements such as, “Family members rarely become openly angry,” and “Family members hardly ever lose their tempers.” The number of endorsed statements was summed to produce a total score ranging from 0–9. This Conflict sub-scale has been shown to have good internal consistency reliability (Cronbach’s $\alpha = 0.72$) (Boyd et al., 1997), which held true for the PHDCN sample (Cronbach’s $\alpha = 0.70$). The total score was standardized to a mean of 0 and standard deviation of 1, with higher scores reflecting higher levels of conflict.

2.2.3. External social support at Wave I—External social support was operationalized as social support received from individuals outside of the immediate family. As part of the Provision of Social Relations scale (Turner et al., 1983), which provides information about the amount and source of social support received from family and friends, youth participants were asked if they had one person in particular who provides them with help when they need it. Response options included: (1) immediate family (parent or sibling), (2) extended family, (3) friend or neighbor, (4) church member/leader, (5) school or community member/leader, or (6) other. Participants were allowed to identify up to three individuals. External social support was analyzed as an ordinal variable with 4 categories: 0, 1, 2 or 3 individuals outside of the participants' immediate family from whom they receive help when needed.

2.2.4. Mediators: Psychological stress at Wave II—Because manifestations of psychological stress can be internal or external, symptoms of psychological stress were examined with two separate measures: internalizing problems and externalizing problems. These problems were measured using the Youth Self Report (YSR) (Achenbach, 1991), a self-report extension of the Child Behavior Checklist (CBCL) (Achenbach and Edelbrock, 1983), which was administered to youth participants in Wave II. The Internalizing Behavior scale is comprised of symptoms related to social withdrawal, somatic complaints, and anxious/depressed behaviors and the Externalizing Behavior scale is comprised of behaviors related to aggression and delinquency. Reliability and validity of the YSR have been demonstrated in prior studies (Achenbach, 1991).

2.2.5. Potential confounding factors—The following variables were included as potential confounders, as they have all been shown in previous studies to be associated with both familial conflict and substance use disorders: history of parental substance use problems (Biederman et al., 2000; El-Sheikh and Flanagan, 2001; Fergusson et al., 2008), history of parental depression (Burke, 2003; Davies and Windle, 1997; Weissman et al., 2006), physical abuse/punishment (Fergusson et al., 2008; Meyerson et al., 2002), primary caregiver marital status (Borine et al., 1991; Hayatbakhsh et al., 2006), family structure (Baer, 1999; Barrett and Turner, 2006), and family socio-economic status (SES; Emery and Laumann-Billings, 1998; McLoyd, 1998; Melchior M et al., 2007). To determine whether participants' parents had a history of substance use problems, primary caregivers were asked which family members had either a drinking problem and/or a drug use problem. Participants whose primary caregivers identified that the participant's mother or father had either of these problems were considered to have a parental history of substance use problems (dichotomous). History of parental depression was measured in a similar fashion. Primary caregivers were asked which family members ever suffered from depression and participants were considered to have a family history of depression if their primary caregiver responded affirmatively on this question for the participants' parents (dichotomous). The physical abuse/punishment measure was based on questions from a derivative of the Conflict Tactics Scale for Parent and Child (Straus et al., 1998) that was administered to the primary caregivers at Wave I. Children of primary caregivers who reported ever doing any of the following were considered to have experienced physical abuse/punishment (dichotomous): (1) thrown something at the participant, (2) kicked, bit or hit the participant, (3) hit the participant with something, (4) beat up the participant, or (5) burned or scalded the participant. Primary caregivers were asked their marital status, which was categorized as married, single, or partnered, and were also asked about the structure of the family, which was categorized as a two parent household, one parent household, or the primary caregiver was not the participant's parent. Finally, family SES was used as a standardized composite of parental education, income, and occupation, derived through principal components analysis (Molnar et al., 2003; Reardon and Buka, 2002). We also controlled for gender, race/ethnicity, and age cohort of the child.

2.3. Analyses

2.3.1. Association between familial conflict and SUDs—Logistic regression models were estimated to evaluate the association between familial conflict at Wave I and SUDs at Wave III. Separate models for each covariate were fitted to obtain unadjusted odds ratios, and then a model with all covariates was fitted to obtain adjusted odds ratios. In addition, to estimate the magnitude of the effect of familial conflict on SUDs, using a dichotomous indicator of familial conflict (dichotomized at one standard deviation above the mean), we calculated the attributable risk fraction (ARF), or the proportion of SUDs among adolescents exposed to familial conflict in childhood that is attributable to the conflict [i.e., (the incidence of SUDs in the exposed – the incidence of SUDs in the unexposed)/(the incidence of SUDs in the exposed)]. Because this analysis investigates the temporal sequence between familial conflict, psychological stress, and SUDs, those who met criteria for SUDs at Wave I (n=96) were excluded from all analyses.

2.3.2. Mediation analysis of psychological stress at Wave II—To evaluate whether psychological stress is a potential mediator of the relation between familial conflict in Wave I and SUDs at Wave III, we conducted a product of coefficients test for the effect of psychological stress as an intervening variable (MacKinnon et al., 2002). For this test, we estimated two regression models: the first regressed symptoms of psychological stress on familial conflict to determine the effect of familial conflict on psychological stress, and the second regressed adolescent SUDs on familial conflict while adjusting for the effects of psychological stress, to assess the effect of familial conflict on SUDs while holding psychological stress constant. We then calculated the product of coefficients, αβ, where α represents the coefficient of familial conflict as a predictor of psychological stress in the first regression model, and β represents the coefficient of psychological stress as a predictor of SUDs, controlling for familial conflict, in the second regression model. Statistical significance of the mediated effect was determined by the asymmetric distribution of products test, where lower and upper confidence limits were calculated based on the product of αβ and a critical value (Meeker et al., 1981) multiplied by the standard error of αβ (MacKinnon et al., 2002). Evidence of mediation is supported if the confidence interval does not cover zero. The mediation effect size was determined by the proportion of the total effect that was attributable to the mediation (i.e., the mediated effect divided by the total effect) (MacKinnon, 2008). We conducted this test for internalizing and externalizing problems separately. Because this study was attempting to establish a temporal sequence between familial conflict, psychological stress, and SUDs, participants with elevated internalizing (n=238) or externalizing (n=225) scores at Wave I, defined as those with scores greater than one standard deviation above the mean, were excluded from the mediational analyses.

2.3.3. Social support at Wave I as a buffering factor of the relation between familial conflict and SUDs—The potential for social support at Wave I to buffer, or mitigate, the effects of familial conflict on SUDs was evaluated by adding the interaction term between social support (ordinal) and familial conflict (continuous) to the fully adjusted logistic regression model. A statistically significant, negative interaction term would be suggestive of a diminishing effect of conflict on SUDs with increasing social support.

For all regression analyses, including the tests for mediation and effect modification, we used generalized estimating equations (Hardin and Hilbe, 2003) to account for the clustering of individuals within neighborhoods.

2.3.4. Multiple imputation of missing data—In the first wave of the study, there were 1,421 participants that did not meet criteria for an SUD, and were therefore eligible for inclusion in the current analysis. However, by the third wave, 1,098 (77%) participants remained.

Furthermore, at Wave III, data for 109 participants were not available for certain survey questions. To assess the factors associated with attrition in the sample, we conducted a series of logistic regression analyses with missing data at either Wave II or III as the outcome. To reduce the potential bias that could result from dropping all participants with missing data (Little and Rubin, 2002), using the PROC MI procedure in SAS version 9.1 (SAS Institute Inc, Cary, NC), we employed multiple imputation techniques to generate five complete datasets, which were then analyzed, and the resulting odds ratios were combined using the PROC MIANALYZE procedure. This technique accounts for sampling variability across imputations, and therefore yields valid estimates and standard errors (Allison, 2001; Little and Rubin, 2002). The Markov Chain Monte Carlo (MCMC) method of imputation was used. All covariates included in the analyses were also included in the imputation model. Continuous variables were rounded to the appropriate decimal place and ordinal and dichotomous variables were restricted to the numeric bounds of the original categories and were rounded to the nearest integer (Allison, 2001). This method assumes that missing data occurred at random, conditional on the observed covariates, and therefore reduces bias relative to a complete-case analysis.

3. Results

3.1. Sample Characteristics

The demographic profile as well as parental and family characteristics of the sample is presented in the first column of Table 1. Over half of the sample was female (51.0%), and the ethnic composition of the sample was diverse, as 44.8% of the sample identified as Hispanic/Latino, 37.4% as Black, and 14.0% as White. The mean age of the sample was 13.5 (SD: 2.4) at Wave I and 18.1 (SD: 1.6) at Wave III. The majority of participants lived in two-parent households (63.7%), and approximately half (54.6%) of participants' primary caregivers were married, 34.0% were single, and 11.4% were otherwise partnered. Regarding parental and family characteristics, 15.6% of the participants had a parental history of substance use problems, 13.3% had a parental history of depression, and 38.2% had a history of parent-child physical abuse/punishment.

Of the 283 participants who met criteria for a substance use disorder at Wave III (19.9%), 162 (57.2%) met criteria based on alcohol dependence and 174 (61.5%) met criteria based on marijuana abuse or dependence (with an 18.7% overlap of the disorders). Additionally, 61.1% met criteria for only one disorder (33.2% alcohol dependence, 5.8% marijuana abuse, and 22.1% marijuana dependence), 27.8 met criteria for two disorders, and 11.1% met criteria for all three.

The second and third columns of Table 1 present the demographic characteristics according to the presence of absence of substance use disorders at Wave III. For example, in the first row, which describes the sample distribution by gender, 696 (49.0%) participants were male (column 1), and of those, 174 (25%) met criteria for a substance use disorder at Wave III (column 2) and 522 (75.0%) did not (column 3).

3.2. Attrition

The attrition analysis revealed that the following variables were associated with an increased odds of missing data either by dropping out of the study or through survey non-response: living in a one-parent household compared to living in a two-parent household (OR: 1.34; CI: 1.08–1.67), having a primary caregiver who is single compared to being married (OR: 1.32; CI: 1.04–1.67), and identifying as African American/Black compared to identifying as white (OR: 1.40; CI:1.00–1.94). However, to the extent that differences were explained by observed variables, the technique of multiple imputation resolved the differences that resulted from missing data.

3.3. Risk of Developing Substance Use Disorders

In the bivariate analyses, familial conflict at Wave I was significantly associated with SUDs in Wave III, indicating that a one standard deviation increase in familial conflict was associated with a 24% higher odds of a substance use disorder (OR: 1.24; 95% CI: 1.05–1.46). Additionally, males were more likely to have a SUD at Wave III (OR: 1.61; CI: 1.19–2.18), as were individuals whose parents had a history of substance use problems (OR: 1.46; CI: 1.01–2.11). Finally, a one standard deviation increase in SES was associated with a 15% higher odds of having an SUD (OR: 1.15; CI: 1.03–1.29). The results of the bivariate analyses are presented in Table 2 (column 1).

In the fully adjusted logistic regression model, the association between familial conflict and substance use disorders was unchanged (OR=1.23; CI=1.02, 1.47). Other significant covariates in the adjusted model were gender and family SES (Table 2, column 2). Finally, we added the interaction term between familial conflict and social support to the adjusted model to test the buffering hypothesis described above; however, it was not significant (p=0.815).

For the calculation of the attributable risk fraction with the dichotomous indicator of familial conflict (dichotomized at one standard deviation above the mean), we found that among those exposed to familial conflict in childhood (n=237), 64 (27.0%) developed an SUD in adolescence, and among those who were not exposed to family conflict in childhood (n=1,184), 222 (18.8%) developed an SUD in adolescence. Therefore, the estimated attributable risk fraction is 0.304 [i.e., (0.270–0.188)/(0.270)] (95% CI: 0.137, 0.471), indicating that 30.4% of SUDs in the sample would have been averted if elevated levels of familial conflict were eliminated.

3.4. Evidence for psychological stress as a mediator of the relation between familial conflict and SUDs

We conducted two tests of the hypothesis that psychological stress mediates the association between familial conflict and SUDs: one for external markers of stress, and one for internal markers of stress (Table 3). There was evidence that externalizing problems partially mediates the association between familial conflict in childhood and SUDs in adolescence. Familial conflict at Wave I was significantly associated with externalizing problems in Wave II (linear regression coefficient, β=0.22; 95% CI: 0.08–0.37; p=0.002). When externalizing problems at Wave II were added to the adjusted logistic regression models of SUDs, the relation between familial conflict and SUDs was attenuated (OR: 1.20; CI: 0.98–1.47; p=0.197). Finally, the product of coefficients test was significant (CI: 0.012–0.014), further supporting the hypothesis. The mediation effect size was 0.30, indicating that externalizing problems contributed to approximately 30% of the total association of familial conflict on the development of substance use disorders.

In contrast, internalizing problems were not found to mediate the relation between familial conflict and SUDs. Familial conflict was not significantly related to internalizing problems at Wave II, and the confidence interval of the product of coefficients test included zero (−0.002, 0.0126).

4. Discussion

The goal of this study was to investigate whether familial conflict during childhood increases the risk of developing substance use disorders in adolescence, and if so, whether this increased risk is due to higher levels of psychological stress. Furthermore, we sought to determine whether this risk could be mitigated by social support. Our primary hypothesis was substantiated in this study, as evidenced by our finding that controlling for a number of

demographic and parental and family characteristics, children who live in families with higher levels of familial conflict when they were younger had a significantly greater risk for developing substance use disorders in late adolescence and emerging adulthood. This is important because not only is it consistent with other studies examining this association (Rhodes and Jason, 1990; Vakalahi, 2001), this is one of the only studies to examine this relationship using longitudinal data, thereby avoiding the alternative reverse causation hypotheses. Furthermore, we found that almost one-third of the cases of substance use disorders in the sample would have been prevented if the adolescents had not lived in family environments with elevated levels of familial conflict. This underscores the need for prevention efforts to focus on adversity in the home.

The social stress model of substance use was supported in this analysis through the test of mediation with external manifestations of psychological stress. Previous studies have examined the pathways from adverse family environments to psychological stress, and from psychological stress to substance use disorders independently (Englund et al., 2008; Fergusson et al., 2007; Harold et al., 1997; Shelton and Harold, 2008; Zucker, 2008); however to the best of our knowledge, no other studies have explored these relationships simultaneously. This study found evidence that familial conflict leads to substance use disorders, at least partially, through externalizing problems, which could have important prevention implications. If children are living in adverse family environments, practitioners can focus intervention efforts on behaviors indicative of externalizing problems, such as aggression, hyperactivity, violence, and delinquency. Directing prevention efforts at the first stage of the pathway can have an even broader impact on the outcomes of these children, with the ultimate goal of substance abuse and dependence prevention.

While external manifestations of psychological stress were found to partially mediate the association between familial conflict and SUDs, internal manifestations of stress (i.e., internalizing problems) did not. One possible explanation for these discrepant findings is that the way children appraise familial conflict may have a differential impact on their psychological adjustment. For example, in previous studies examining the relationship between familial conflict and child adjustment using the cognitive-contextual framework, perceived threat in relation to inter-parental conflict has been shown to be predictive of internalizing problems (Grych et al., 2000; Grych et al., 2003), while self-blame has been shown to predict externalizing problems (Dadds et al., 1999; Grych et al., 2003). Therefore, familial conflict, as measured in the current study, may lead to more self-blaming types of appraisals among children rather than threatening types, which may account for the lack of association between familial conflict and internalizing problems, as, for example, self-blaming could lead to risk taking representative of externalizing behaviors. This study was not able to explore the youths' appraisal of familial conflict and therefore, further research must be conducted to test this hypothesis.

Another possible explanation is that the null finding with internalizing problems may be attributable to low statistical power. Familial conflict, as assessed by the primary caregiver, may not be a reliable measure, which would decrease the power of this analysis. At Wave I, only primary caregivers were inquired about the family environment. While this may be a reliable measure among caregivers, because internalizing problems at Wave II and SUDs at Wave III were assessed from the youth's perspective, assessing the exposure from the primary caregiver's perspective may reduce the reliability of this measurement.

Finally, external social support, as measured in the first wave of the PHDCN, was not significantly associated with SUDs in adolescence as a main effect, and furthermore, was not found to buffer the effects of familial conflict on subsequent SUDs in late adolescence. There are several potential explanations for this finding, which was counter to expectations based on

prior research indicating that social support has "stress-buffering" effects on health behaviors (Cohen and Wills, 1985), and more specifically, on substance use behaviors (Peirce et al., 2000). External social support later in adolescence might offer greater protection against the effects of familial conflict; however, the Provision of Social Relations scale was not administered to participants at Waves II or III, so this possibility could not be assessed in the current study. Additionally, this analysis was restricted to support received outside of the immediate family. In the face of an adverse family environment, children may receive their support from their immediate family members. We did not test whether social support in childhood, including support received from the immediate family members, buffered the relationship between familial conflict and SUDs because only 6% of the sample did not have any social support as measured by the PHDCN. Finally, the measure used to assess external social support was limited in that it only inquired about social support received by up to three individuals. The restricted range of this variable may have weakened its relationship with SUDs, thus reducing the power to detect an effect. Additional research would need to be conducted in order to test this relationship further.

4.1. Limitations

Given that the PHDCN is an observational study, the ability to draw causal inferences is limited; however, the prospective assessments of familial conflict, psychological stress, and SUDs enabled us to establish the temporality of associations of hypothesized risks for SUDs. Second, the study sample is restricted to the city of Chicago. While the PHDCN represents a unique opportunity to examine the developmental pathways of risk behaviors at both the individual and neighborhood level, findings are not necessarily generalizable to all populations. However, the multi-stage sampling design of the PHDCN, which resulted in a large, gender balanced, ethnically diverse sample, helped to strengthen the external validity of the study.

A further limitation is that the majority of measures were collected via self-report, which is most problematic for assessments of socially undesirable substance use behaviors in adolescence. However, studies that have examined the validity of self-reports of alcohol and marijuana use have concluded that this method of data collection, while not ideal, is moderately valid (Secades-Villa and Fernandez-Hermida, 2003; Williams and Nowatzki, 2005). Furthermore, alcohol and marijuana are considered to be the least stigmatized substances, which increases the validity of the self-report measure because individuals are more likely to accurately report their use of these substances compared to drugs that are more highly stigmatized (Rosay et al., 2007; Williams and Nowatzki, 2005). Finally, attrition of participants in Waves II and III resulted in missing data. However, to the extent that values on missing covariates were dependent on observed variables, the multiple imputation procedures accounted for selection biases due to attrition.

Finally, while the current study investigated the pathway from high-risk family environments in childhood to SUDs in adolescence through the mechanism of psychological stress, there are other possible mechanisms through which the exposure could lead to substance use disorders that we did not examine. For example, these relationships could be examined through the mechanism of modeling; more specifically, if conflict in the home environment leads to parental substance use, then adolescents may model these behaviors. Additionally, we did not explore the role that peers play in this relationship, as for instance, children living in home environments with high levels of conflict may turn to deviant peer groups for support, which could lead to substance use and subsequent disordered use. Furthermore, while we controlled for family socioeconomic status in the analyses, the relationship between familial conflict and substance use could, at least in part, be due to residual confounding, which could either attenuate or nullify the effect. Lastly, certain parenting practices may be compromised as a result of high levels of familial conflict, including supervision/monitoring, consistent

discipline, communication, and physical presence, which could all impact subsequent substance use behaviors.

4.2. Conclusion

In summary, we report evidence to support the hypothesis that living in an adverse family environment in childhood, specifically in a family with elevated levels of conflict, has a significant influence on subsequent substance use disorders in adolescence, even after controlling for a number of demographic and parental and family characteristics. Furthermore, we extend the current literature by demonstrating that the effect of familial conflict on SUDs is at least partially explained by increased levels of external, but not internal manifestations of psychological stress and that external social support did not appear to mitigate these effects. We conclude that familial conflict in childhood is especially problematic because it can put children at increased risk for externalizing problems and subsequent substance use disorders, and that external social support does not help to buffer these effects.

The most notable strength of this study is that the data were longitudinal in nature with three points of data collection between 1994 and 2001, allowing us to examine the relationship between familial conflict in late childhood/early adolescence on the development of substance use disorders in late adolescence/emerging adulthood prospectively. Using the three waves, we were able to measure familial conflict, psychological stress, and SUDs using a prospective design with temporal ordering of the associations, which strengthened our ability to make inferences about causality.

Future research should investigate additional pathways by which familial conflict earlier in life can lead to the development of substance use disorders in late adolescence and emerging adulthood. When these pathways are more clearly understood, public health practitioners will be able to identify, develop, and test interventions that address the impact of early family environmental determinants of substance use disorders, with the ultimate goal of preempting these problems.

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Table 1

Sample characteristics, broken down by the primary outcome, substance use disorders – 1,421 participants in the Project on Human Development in Chicago Neighborhoods.

	Sample Distribution N (%)[a] (n=1,421)	Among those with SUDs at Wave III (n=283) n (%)[b]	Among those with SUDs at Wave III (n=1,138) n (%)[b]
Wave I Demographics			
Gender			
Male	696 (49.0)	174 (25.0)	522 (75.0)
Female	725 (51.0)	109 (15.0)	616 (85.0)
Race/Ethnicity			
Hispanic	635 (44.8)	119 (18.7)	516 (81.3)
Black	532 (37.4)	107 (21.4)	425 (78.6)
White	199 (14.0)	47 (23.6)	152 (76.4)
Other	55 (3.8)	10 (18.2)	45 (81.8)
Age Cohort			
12	811 (57.1)	144 (17.8)	667 (82.2)
15	610 (42.9)	139 (27.8)	471 (26.8)
Wave I Parental and Family Characteristics			
Standardized mean Family Conflict score (SD)	0.00 (1.00)	0.17 (1.05)	−0.04 (0.98)
	Median: −0.35	Median: 0.15	Median: −0.35
	Min: −1.36;Max: 3.17	Min: −1.36; Max: 3.17	Min: −1.36; Max: 3.17
Family structure			
Two parent household	905 (63.7)	186 (20.6)	719 (79.4)
One parent household	426 (30.0)	82 (19.2)	344 (80.8)
Living with guardian (not parent)	90 (6.3)	15 (16.7)	75 (83.3)
Caregiver marital status			
Married	776 (54.6)	150 (19.3)	626 (80.7)
Single	483 (34.0)	92 (19.0)	391 (81.0)
Partnered	162 (11.4)	41 (25.3)	121 (74.7)
Average family SES[c]	−0.16 (1.39)	0.05 (1.4)	−0.21 (1.4)
Family history of substance use			
No	1199 (84.4)	228 (19.0)	971 (81.0)
Yes	222 (15.6)	55 (24.8)	167 (75.2)
Family history of depression			
No	1232 (86.7)	236 (19.2)	996 (80.8)
Yes	189 (13.3)	47 (24.9)	142 (75.1)
Participant Mental Health			
History of parent-child physical abuse			
No			
Yes			

	Sample Distribution N (%)[a] (n=1,421)	Among those with SUDs at Wave III (n=283) n (%)[b]	Among those with SUDs at Wave III (n=1,138) n (%)[b]
Mean internalizing problem score (SD) (Wave II)	11.3 (7.6)	12.6 (7.8)	11.0 (7.5)
	Median: 10	Median: 11	Median: 10
	Min: 0; Max: 55	Min: 0; Max: 55	Min: 0; Max: 55
Mean externalizing problem score (SD) (Wave II)	9.13 (5.3)	11.8 (5.6)	8.45 (5.0)
	Median: 8	Median: 12	Median: 8
	Min: 0; Max: 31	Min: 0; Max: 31	Min: 0; Max: 31
External social support (Wave I)			
None	420 (29.6)	77 (18.3)	343 (81.7)
1 person	500 (35.2)	87 (17.4)	413 (82.6)
2 people	325 (22.9)	82 (25.2)	243 (74.8)
3 people	176 (12.3)	37 (21.0)	139 (79.0)

[a] Column number and percent

[b] Column number and row percent

[c] 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.

Table 2

Bivariate and multivariable logistic regression models examining the association of family conflict at baseline (Wave I) and substance use disorders at final follow-up (Wave III) (n=1,421)

	Biv ariate Models	Final Multivariable Model
Variable	**OR (95% CI)**	**OR (95% CI)**
Family conflict score (standardized)	1.24* (1.05, 1.46)	1.22* (1.03, 1.44)
Demographics		
Gender		
Female	1.00	1.00
Male	1.61** (1.19, 2.18)	1.61** (1.18, 2.19)
Race/Ethnicity		
Hispanic	1.00	1.00
Black	1.19 (0.84, 1.68)	1.04 (0.68, 1.59)
White	1.35 (0.86, 2.05)	0.97 (0.61, 1.53)
Other	1.05 (0.47, 2.36)	0.87 (0.39, 1.95)
Age Cohort		
12	1.00	1.00
15	1.13 (0.98, 1.29)	1.12 (0.98, 1.29)
Family Characteristics		
Parental history of substance use		
No	1.00	1.00
Yes	1.46* (1.01, 2.11)	1.34 (0.92, 1.96)
Parental history of depression		
No	1.00	1.00
Yes	1.25 (0.81, 1.92)	1.25 (0.81, 1.92)
Caregiver marital status		
Married	1.00	1.00
Single	0.95 (0.64, 1.41)	0.92 (0.57, 1.48)
Partnered	1.35 (0.83, 2.19)	1.21 (0.75, 1.95)
Family structure		
Two parent household	1.00	1.00
One parent household		
Living with guardian (not parent		
Family SES	1.15* (1.03, 1.29)	1.14* (1.02, 1.28)
Participant Mental Health		
History of parent-child physical abuse		
No		
Yes		
External social support at Wave I		
None	1.00	1.00
1 person	0.92 (0.53, 1.57)	0.91 (0.54, 1.55)
2 people	1.48 (0.89, 2.46)	1.49 (0.91, 2.44)
3 people	1.18 (0.68, 2.12)	1.15 (0.62, 2.14)

Variable	Biv ariate Models OR (95% CI)	Final Multivariable Model OR (95% CI)

*$p < 0.05$

**$p < 0.01$

Table 3

Bivariate and multivariable logistic regression models testing the effects of internalizing and externalizing problems as mediating variables in the association between family conflict at baseline assessment (Wave I) and substance use disorders at final follow-up (Wave III) (n=1,421)

	Bivariate Models	**Multivariable Models[d]**	
		Final Model with Externalizing Problems	**Final Model with Internalizing Problems**
Variable	**OR (95% CI)**	**OR (95% CI**	**OR (95% CI**
Family conflict score (standardized)	1.24* (1.05, 1.46)	1.17 (0.92, 1.50)	1.19* (1.01, 1.41)
Externalizing problems (standardized)	1.79*** (1.46, 2.20)	1.13** (1.07, 1.18)	NA
Internalizing Problems at Wave II (standardized)	Internalizing problems 1.03*** (1.02, 1.05)	NA	1.36** (1.12, 1.65)

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

[d]Each multivariable model adjusted for all demographic and family characteristic variables as detailed in Table 3.