Published in final edited form as:
Addict Res Theory. 2014 June 1; 22(3): 239–250. doi:10.3109/16066359.2013.830713.

Parental Warmth and Risks of Substance Use in Children with Attention-Deficit/Hyperactivity Disorder: Findings from a 10–12 Year Longitudinal Investigation

Mini Tandon, D.O.,
Department of Psychiatry, Washington University School of Medicine, 660 South Euclid Avenue, St. Louis, MO 63110

Rebecca Tillman, M.S.,
Department of Psychiatry, Washington University School of Medicine, 660 South Euclid Avenue, St. Louis, MO 63110

Edward Spitznagel, Ph.D., and
Department of Mathematics, Washington University in St. Louis, One Brookings Drive, St. Louis, MO 63130

Joan Luby, M.D.
Department of Psychiatry, Washington University School of Medicine, 660 South Euclid Avenue, St. Louis, MO 63110

Abstract

Objective—The study examined factors in the risk trajectory for Substance Use Disorder (SUD) over a 10–12 year period in children with ADHD.

Method—N=145 children between the ages of 7 and 16 with ADHD and healthy controls were assessed every 2 years for 10–12 years as part of a larger, longitudinal investigation. Onset of substance use disorder was examined using Cox proportional hazards modeling, and included child and parent psychopathology, and parental warmth as well as other key factors.

Results—Low paternal warmth and maternal SUD were predictors of SUD in n=59 ADHD participants after adjusting for gender, child ODD, paternal SUD, maternal/paternal ADHD, maternal/paternal major depressive disorder (MDD), maternal/paternal anxiety, and low maternal warmth in the Cox model.

Conclusions—Longitudinal study findings suggest that in addition to the established risk of ADHD and maternal SUD in development of child SUD, low paternal warmth is also associated with onset of SUD. This was evident after controlling for pertinent parent and child psychopathology. These findings suggest that paternal warmth warrants further investigation as a key target for novel interventions to prevent SUD in children with ADHD. More focused

Correspondence to Mini Tandon, D.O., Department of Psychiatry, Washington University School of Medicine, 660 S. Euclid Ave., Campus Box 8134, St. Louis, MO 63110-1093, USA. Telephone: 314-286-0180; Fax: 314-286-1696; tandonm@psychiatry.wustl.edu.

Declaration of Interest
The authors report no conflicts of interest.

investigations examining paternal parenting factors in addition to parent and child psychopathology in the risk trajectory from ADHD to SUD are now warranted.

Keywords

Attention-Deficit/Hyperactivity Disorder (ADHD); substance use; parenting; warmth; paternal

INTRODUCTION

Many studies have examined developmental risk factors associated with adolescent and young adult onset SUD, including a variety of forms of childhood psychopathology (Wilens et al., 2011). In particular, the role of ADHD in the risk for SUD has been widely studied (Armstrong & Costello, 2002; Barkley, DuPaul, & McMurray, 1990; Biederman, Monuteaux, Mick, Wilens, et al., 2006; Biederman, Monuteaux, Mick, Spencer, et al., 2006; Brook, Brook, Zhang, & Koppel, 2010; Elkins, McGue, & Iacono, 2007; Fergusson, Horwood, & Ridder, 2007; Flory & Lynam, 2003; Lee, Humphreys, Flory, Liu, & Glass, 2011; Molina et al., 2007; Molina & Pelham, 2003; Wilens et al., 2011). The increased risks for SUD in children with ADHD are further supported by several recent meta-analyses of prospective cohort studies of children with ADHD followed to adolescence and/or adulthood (Charach, Yeung, Climans, & Lillie, 2011; Lee et al., 2011). Despite this rich body of literature, an area which lacks clarity in the risk trajectory from ADHD to SUD is the role of parenting factors. Parenting practices have been found to be associated with risks for SUD in some studies and not in others, and findings have varied by age, gender, dimension of parenting studied (e.g., warmth, discipline, monitoring), and informant (parent, child, clinician-observer) (Chassin & Handley, 2006).

The Role of Parenting in Child SUD

Parenting is a well-established risk/protective factor for development of more general forms of adolescent and adult psychopathology, including risk for substance use disorder (Andersson & Eisemann, 2003; Baumrind, 1991; Blackson, Tarter, & Mezzich, 1996; Chassin et al., 2005; Chassin, Presson, Rose, & Sherman, 1998; Choquet, Hassler, Morin, Falissard, & Chau, 2008; Coombs & Landsverk, 1988; Enns, Cox, & Clara, 2002; King & Chassin, 2004; Marshal & Chassin, 2000; Mezzich et al., 2007; Parker & Benson, 2004; Pires & Jenkins, 2007; Wills, Resko, Ainette, & Mendoza, 2004). Parenting factors examined have included parenting style, (Baumrind, 1991) discipline, (Mezzich et al., 2007) monitoring, (Barnes, Hoffman, Welte, Farrell, & Dintcheff, 2006; Dick et al., 2007; Flannery, Williams, & Vazsonyi, 1999; Patock-Peckham, King, Morgan-Lopez, Ulloa, & Moses, 2011) and responsiveness or measures of parental warmth/coldness (Kendler, Myers, & Prescott, 2000). Some studies have suggested that monitoring is the dimension of parenting with the strongest effect on risk for SUD (Barnes & Farrell, 1992; Griffin, Botvin, Scheier, Diaz, & Miller, 2000). The dimension of parenting known as parental responsiveness (also referred to as parental warmth or supportiveness) has also been found to exert strong effects in the risk trajectory for SUD (Kendler et al., 2000). Kendler et al. (2000) found “coldness” to be the most significant factor predicting psychopathology when coldness, protectiveness and authoritarianism were examined together in an epidemiological sample of adult female twins retrospectively reporting on parenting and adult psychiatric

outcomes. In additional studies, both parental rejection and warmth independently predicted SUD after taking into account possible mediation by deviant peer affiliation and child ADHD in the large Canadian National Longitudinal Survey of Children and Youth of children ages 10 to 17 years (Pires & Jenkins, 2007). Despite these findings, the mechanism by which parental warmth is associated with SUD remains unclear. Kendler et al. (2000) have suggested that the association with maternal "coldness" is related to comorbidities such as maternal depression. However, child ADHD was not specifically considered in this investigation or many others examining parental influences on substance use (Bogenschneider, Wu, Raffaelli, & Tsay, 1998).

Until recently, most studies have focused on the role of mothers in the parent-child relationship and risk for later childhood psychopathology (Lamb, 2000); however, paternal involvement has gained increasing attention and has been consistently associated with positive child outcomes in numerous investigations (Lamb, 1997; Marsiglio & Cohan, 2000). The role of fathers has been of increased interest in parenting studies focused on risk for psychopathology in general, and substance use disorders specifically (Bronte-Tinkew, Moore, & Carrano, 2006; Parke, 2000). Coombs and Landsverk (1988) found paternal warmth to be significantly associated with child substance use. Specific to fathers, substances were frequently used by 67.5% of those children reporting that they were "not close at all," compared to 36% reporting "moderately-close" and 16% reporting "very close" (Coombs & Landsverk, 1988). Woodward, Taylor, and Dowdney (1998) have suggested that low levels of paternal warmth may contribute to development of conduct disorder in children with ADHD, thereby further increasing the risk for SUD.

While multiple studies support parenting as a predictor of child SUD, several studies have also failed to find such an association. Parental warmth and physical discipline in kindergarteners were not associated with later risk for SUD (Kaplow, Curran, Dodge, & Conduct Problems Prevention Research Group, 2002). The authors suggested, however, that the lack of association found may have been related to parent self report of warmth which is known to have questionable validity. Taken together, there is a dearth of literature examining the role of parenting in the risk trajectory to SUD and specifically within populations with ADHD. Furthermore, despite the established risks for SUD in children with ADHD, (Barkley & Murphy, 2006; Biederman et al., 1997; Fergusson et al., 2007; Wilens et al., 2011), the role of parental warmth remains underinvestigated. Given this gap in the literature, and the finding that parenting has been established as an early and modifiable risk factor in a number of child and adolescent psychiatric outcomes (Bauman et al., 2002; Chronis et al., 2007; Eyberg, Boggs, & Algina, 1995; Webster-Stratton, 1998), the following study examined the role of parental warmth in the risk trajectory of child ADHD to adolescent onset SUD. We hypothesized that low parental warmth (both maternal and paternal) would predict adolescent onset SUD in children with ADHD.

METHODS

Participants

Study participants were children between the ages of 7 and 16 years old (76.7% male), who had DSM-IV ADHD (with hyperactivity, i.e., hyperactive/impulsive subtype [H] or

combined type [C], not inattentive type [I]) consecutively ascertained from outpatient pediatric and psychiatric clinics as a comparison group for participants with child Bipolar I disorder (BP-I) for the Phenomenology and Course of Pediatric Bipolar Disorders study (NIMH R01 MH-53063 to Barbara Geller, M.D.). For the current study, we included only those participants with ADHD and healthy controls. Participants with BP-I were excluded as their risk trajectory to SUD is thought to be unique and will be explored in a separate analysis. Participants were comprehensively assessed (see measures below) every 2 years for a 10 or 12-year period [n=103 for 10 years, n=37 for 12 years (the study ended mid-way through collection of year 12 data), and n=5 dropouts]. There were no missed assessments other than those occurring after discontinuation in dropouts. Children were in good physical health but had a Children's Global Assessment Scale (CGAS) (Bird, Canino, Rubiostipec, & Ribera, 1987; Shaffer et al., 1983) score of ≤60 to establish definite clinical impairment, in addition to onset of symptoms prior to age 7 and duration greater than or equal to 6 months. Exclusion criteria were IQ<70, epilepsy or other major medical or neurological disorders, pervasive developmental disorders, schizophrenia, baseline substance dependency, and pregnancy.

Rationale for inclusion/exclusion criteria was based on the Phenomenology and Course of Pediatric Bipolar Disorders study and is described in detail elsewhere (Geller, Tillman, Craney, & Bolhofner, 2004). Specifically, ADHD-H and ADHD-C but not ADHD-I were included as a psychiatric comparison group (given focused study goals). Children in the ADHD group could not have MDD (based on original study aims), but could have CD and/or ODD, given their typical comorbidity with ADHD in children. Participants could have MDD on follow-up. Further, participants who were pregnant or had SUD were included only if onset occurred subsequent to baseline assessment. A minimum age of 7 was established to increase interview credibility; a maximum age of 16 was established to ensure participants would be teenagers at the 2-year follow-up assessment.

Screenings for exclusion criteria for all new consecutive cases were conducted by non-blind research nurses who were different than the blinded nurses who conducted in-laboratory psychiatric assessment once telephone screenings occurred. N=94 Healthy controls (HC) were those matched to the original child BP-I group based on age, gender, ethnicity, socioeconomic status (SES), and zip code and had CGAS scores greater than or equal to 70 (non-impaired). Controls had the same exclusion criteria as the other two groups and could not have a history or could not meet full criteria for the diagnosis of bipolar disorder, ADHD, or MDD. Healthy controls did not meet criteria for any Axis I disorder at baseline and were obtained from a random survey to match the original study bipolar group, while the ADHD group was obtained via consecutive new case ascertainment along with bipolar participants.

Measures

Diagnostic Assessment—The Washington University in St. Louis Kiddie Schedule for Affective Disorders and Schizophrenia (WASH-U-KSADS) (Geller, William, Zimerman, & Frazier, 1996) is a semi-structured interview that was administered by experienced research clinicians to mothers about their children and to children about themselves. It was developed

from the KSADS (Puig-Antich & Ryan, 1986) by adding onset and offset of lifetime and current symptoms for DSM-IV diagnoses. The WASH-U-KSADS has established reliability and validity to parent and teacher reports (Geller et al., 1998; Geller et al., 2001). Teacher ratings were not addressed in the current study. To score the WASH-U-KSADS, child and parent responses were combined by using the most severe rating, in accordance with the methods described by Bird, Gould, and Staghezza (1992).

SUD was defined using DSM-IV criteria including for alcohol, marijuana and other illicit drug use (DSM-IV, American Psychiatric American, 1994). The Substance Use Inventory for WASH-U-KSADS was used to assess DSM-IV SUDs and was given to participant and parent separately (Geller et al., 1998). The Substance Use Inventory provides data on type, quantity, frequency, and onsets and offsets of tobacco, alcohol, and drug use, though child tobacco data were not addressed in this manuscript. Age of SUD onset was the youngest age at which all DSM-IV criteria were met for SUD. The Substance Dependency Disorders Template to the WASH-U-KSADS was also given separately to participants and parents. A positive endorsement from either parent or child was counted toward the diagnosis. It contains information on tolerance, withdrawal, and impairment in a semi-structured format similar to the WASH-U-KSADS.

All research materials, including school reports and separate videotapes of mothers and children, were reviewed in consensus conference with research nurses and a senior clinician. Raters were blind to group status at baseline assessment. They were trained to inter-rater reliability (kappa = 0.82-1.00) and recalibrated yearly (Geller et al., 2001).

Global Functioning. The CGAS measures severity based on global impairment from psychiatric symptoms and related adaptation in psychosocial functioning in school, social, work, and family contexts. On this scale, 0 is worst, 100 is best, and ≤ 60 is definite clinical impairment. The CGAS score is the lowest level of functioning during the rating period.

Socioeconomic status was established from the Hollingshead Four-Factor Index of Social Status (Hollingshead, 1976).

Parental Warmth, including both maternal and paternal self and child ratings, was obtained from the Psychosocial Schedule for School Age Children-Revised, a semi-structured measure used to assess relationships between 7 to 18 year-old participants and their parents, siblings, teachers, peers, and parental marital relationships (PSS-R) (Puig-Antich, Lukens, & Brent, 1986). Similar to the scoring of the WASH-U-KSADS, participant and parent scores on the PSS-R were combined by using the most severe score for each item (Bird et al., 1992). A score of 1 on maternal and paternal warmth indicated mutual concern and affection, a score of 2 indicated mutual concern and affection, but with some distance, a score of 3 indicated a not particularly close relationship, and a score of 4 indicated dislike and avoidance. Parental warmth was scored as the most severe of the maternal and paternal warmth scores. Paternal warmth scores were based on the father figure with whom the child had most contact. All participants in this analysis had maternal warmth scores of 1 or 2 (on a scale of 1 to 4). Although parental warmth was collected across study waves, only warmth as measured at baseline assessment was used in this study. The Hollingshead Four-Factor

Index of Social Status is embedded in the PSS-R. Human studies at Washington University in St. Louis approved the informed consent process. Written informed consent from parents and written assent from children were obtained (Geller et al., 2000).

Statistical Analyses—Participant characteristics were compared in ADHD and HC groups using Chi-square tests for categorical variables and t-tests for the continuous variables baseline age, final assessment age, and ADHD severity score.

Potential predictors of SUD onset were modeled in three stepwise selection multivariate Cox proportional hazards models, one model for ADHD participants, one for HC participants, and one for ADHD and HC participants combined. Independent variables thought to predict child SUD included gender, age, SES, ODD, number of life events, maternal/paternal SUD, maternal/paternal smoking, maternal/paternal ADHD, paternal antisocial personality disorder (ASPD) (no mothers had ASPD), maternal/paternal MDD, maternal/paternal anxiety, and maternal/paternal warmth.

The cumulative probability of SUD onset was estimated stratifying across significant predictors from the Cox models using the Kaplan-Meier method. Kaplan-Meier analysis is used to model how a population evolves over time and allows for dropouts and differing ages and lengths of follow-up time.

RESULTS

Participants

For the original study, n=81 ADHD participants were ascertained from 1468 total new consecutive cases. Thus, 5.5% (81/1468) of consecutive new cases fit the ADHD-H or ADHD-C categories, and did not meet criteria for child BP-I or MDD.

For the current study, of the n=81 participants with ADHD, n=55 never switched to BP-I over the course of the study, and were therefore retained in the ADHD group. There was n=1 ADHD participant who discontinued the study after the baseline assessment and therefore was not included in the analyses. Of the n=94 healthy controls, 3 switched to BP-I, so they were excluded from the current analyses. An additional n=19 HC participants switched to ADHD during follow-up, so they were included in the ADHD group (At baseline, 13 of 94 HC participants had at least 1 ADHD symptom, and the most ADHD symptoms an HC participant had at baseline was 6. This would account for several of the HC becoming ADHD at later time points. The only diagnostic requirement to be included in the HC group was not having any disorder from the WASH-U-KSADS at baseline therefore HC participants could have symptoms of any disorder, but not meet full criteria for a diagnosis). For participants who switched to ADHD after baseline, their baseline characteristics (age, SES, parental warmth, etc.) were taken from the assessment corresponding to the time of ADHD onset. Therefore, there were n=73 ADHD participants and n=72 HC participants in the analyses that follow. N=23 participants were not included in the final analysis due to missing parental data. There were n=59 fathers of ADHD participants and n=63 fathers of HC participants available to the study. Therefore, n=122 participants were in the final analysis, n=59 ADHD and n=63 HC participants. Participants

included vs. not included in the final analysis did not differ on the baseline variables age, gender, race, SES, living situation (with whom the child resides), ADHD severity, temperament, presence of ODD during the study, length of follow-up, or onset of SUD during follow-up. No participants were diagnosed with comorbid Conduct Disorder (CD) in this sample. There was a high threshold for making a diagnosis of CD using the WASH-U-KSADS in the original study. As detailed above, there were n=73 ADHD and n=72 HC participants. N=4 ADHD participants and n=1 HC participant dropped out of the study. Average ages at each follow-up and at the final assessment were 12.5±2.6 at 2 years, 14.6±2.5 at 4 years, 16.6±2.6 at 6 years, 18.6±2.6 at 8 years, 20.7±2.6 at 10 years, 22.0±2.1 at 12 years, and 21.0±2.8 at the final assessment (either 10- or 12-year follow-up). The length of follow-up differed in the two groups (ADHD: 9.1±3.8 years vs. HC: 10.1±0.6 years, t=2.09, p=0.0401) because for switchers to the ADHD group, follow-up was defined as the length of time from the wave of ADHD onset until the final follow-up assessment. The ADHD and HC groups were analyzed separately. The main rationale for analyzing groups separately was to account for different ascertainment strategies for the ADHD and HC groups, a more conservative approach. However, notably when groups are combined, the study results remain.

Characteristics of the sample are presented in Table 1. There were significantly more males in the group of children with than without ADHD. Similarly, there was an increased diagnosis of ODD in the group with than without ADHD. Paternal SUD was significantly higher in the group with ADHD than without. Furthermore, maternal and paternal ADHD were significantly increased in the group of children with compared to without ADHD. Maternal and paternal MDD, maternal anxiety, and low maternal and paternal warmth were significantly increased in the group of children with ADHD compared to those without ADHD. Living situation (both bio-parents) differed significantly in the ADHD vs. control group but this difference did not change the findings when added as a covariate in the model. Age distribution at baseline and assessment are listed in Tables 1A and 1B.

Predictors of SUD

Maternal SUD and low paternal warmth significantly increased risk for onset of child SUD in the group with ADHD. Specifically, in the ADHD group, a stepwise selection Cox proportional hazards model of SUD onset was run with independent variables gender, age, SES, ODD, number of life events, maternal/paternal SUD, maternal/paternal smoking, maternal/paternal ADHD, paternal ASPD, maternal/paternal MDD, maternal/paternal anxiety, and maternal/paternal warmth. The resulting model included the independent variables maternal SUD and paternal warmth, as shown in Table 2A. The covariates gender, ODD, paternal SUD, maternal/paternal ADHD, maternal/paternal MDD, maternal/paternal anxiety, and maternal warmth were then added to the model. The resulting model is shown in Table 2B. The final model has significant hazard ratios of 66.94 for maternal SUD and 4.49 for paternal warmth. A test for collinearity of the independent variables was conducted for the final model, and no covariates were collinear (variance inflation factors ranged from 1.12 to 1.56). Figure 1 illustrates the cumulative probability of SUD onset by paternal warmth in ADHD subjects.

A similar stepwise selection Cox proportional hazards model of SUD onset was run in the HC group, but no variables remained after stepwise selection. This is likely due to small sample size and limited power as only 3 HC participants developed SUD during follow-up.

Results of the stepwise selection Cox proportional hazards model of SUD onset in ADHD and HC participants combined are shown in Table 3. Child ADHD, maternal SUD, and low paternal warmth significantly increased risk for onset of child SUD (Table 3A). The covariates gender, ODD, paternal SUD, maternal/paternal ADHD, maternal/paternal MDD, maternal/paternal anxiety, and maternal warmth were then added to the model. The resulting model is shown in Table 3B. The final model has significant hazard ratios of 6.65 for male gender, 22.44 for maternal SUD, 5.43 for maternal anxiety, and 3.50 for paternal warmth. A test for collinearity of the independent variables was conducted for the final model, and no covariates were collinear (variance inflation factors ranged from 1.10 to 1.77). Figure 2 illustrates the cumulative probability of SUD onset by paternal warmth in ADHD and HC subjects.

The mean (SD) age of SUD onset in the N=21 participants with SUD was 17.4 (2.0) years. N=16 of 21 participants with SUD recovered (no longer had SUD at the end of the follow-up period). Table 4 reviews drugs specifically used through the 8-year follow-up.

DISCUSSION

Study results support the hypotheses that low paternal warmth is a predictor of SUD in adolescence, in children with ADHD. These results held when ADHD subjects were analyzed separately and when ADHD and HC groups were combined. In contrast to our hypothesis, maternal warmth was not found to be a predictor of child SUD. However, we cannot rule out that this was a function of the low variance in maternal warmth scores in this study sample. Study findings highlight parenting, in particular paternal warmth, as an underinvestigated and robust predictor of the risk trajectory from ADHD to SUD. Findings suggest that more focus on fathers is warranted to understand the risk trajectory to SUD. Whether warmth could be a target in preventive intervention is worthy of investigation and has clear implications for onset of SUD in children with ADHD.

Maternal SUD was found to be a predictor of child SUD in our study. This is consistent with findings reported in several prior investigations (Chassin, Pillow, Curran, Molina, & Barrera, 1993; Chassin, Pitts, DeLucia, & Todd, 1999). Current findings confirm multiple prior investigations showing that childhood ADHD remains a key predictor of SUD in adolescence and young adulthood in prospective studies (Biederman et al., 1997; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Molina & Pelham, 2003; Wilens et al., 2011), and in retrospective adult studies (Wilens, Biederman, Mick, Faraone, & Spencer, 1997). However, current study results showing maternal SUD as a predictor of adolescent or young adult SUD stand in contrast to recent findings by Wilens et al. (2011).

The study presents several strengths. First, in the current study, both participants and parents were queried for substance use, perhaps increasing the ability to detect substance use in both

parents and children more accurately. Second, participants in the current study were assessed every two years over 12 years rather than only one final follow up. This frequent follow-up may have allowed detection of parental psychopathology not detected in other samples (Wilens et al., 2011). This design difference may explain why findings from this study are discrepant from those reported by Wilens et al. (2011). Additional study strengths included the examination of the role of parental warmth in this risk trajectory from ADHD to SUD. Elucidation of key modifiable predictors of SUD in addition to child ADHD remains critical to preventive intervention efforts. Findings that low paternal warmth is a robust predictor of SUD replicate prior available research on the central role of parenting in the risk for SUD (Chassin & Handley, 2006).

There are several key limitations to the study. A type I error cannot be ruled out given the sample size, and findings should be considered preliminary. The overall sample size and high threshold for CD diagnosed in the original study warrant focus in a larger, future investigation. Prior study findings focusing on the predictive role of CD could not be replicated given the high threshold for making the CD diagnosis using the WASH-U-K-SADS in the current sample; prior studies reviewed suggest conduct problems mediate the relationship between hyperactivity and SUD (Tarter, Kirisci, Feske, & Vanyukov, 2007); the comorbidity of CD with ADHD predicts or worsens risk for SUD (Molina & Pelham, 2003; Molina, Smith, & Pelham, 1999) and criminality (Moffitt, 1990); and that, after controlling for CD, ADHD no longer predicts SUD (Brook et al., 2010; Lynskey & Hall, 2001). However, Wilens et al. (2011) found ADHD to predict SUD even after controlling for CD, which in our sample was unnecessary. Current study findings should be considered preliminary in this context. Next, the use of DSM-IV, which does not distinguish between onset of SUD in adults compared to children, can be viewed as a limitation. While the application of adult DSM-IV SUD criteria to adolescents has been debated in the literature, it remains widely used in empirical studies (Deas, Riggs, Langenbucher, Goldman, & Brown, 2000; Martin & Winters, 1998; Winters, 2013). In addition to the use of adult definition of SUD, the measure of SUD as a dichotomous outcome can be seen as a limitation. However, this limitation is minimized by the findings that a dichotomous measure of SUD is valid given evidence for common liability risk for multiple substances as reviewed in the Familial Aggregation of Common Psychiatric and Substance Use Disorders (Kendler, Davis, & Kessler, 1997). Most of the SUD in this sample was attributed to marijuana and alcohol.

Similar to the sample investigated by Wilens et al. (2011), current study participants were mostly white and middle class, limiting generalizability of these findings to the broader population. Further, medication use was not addressed. Recent findings suggest that stimulant treatment for ADHD has not been associated with increasing or decreasing the risk for adolescent SUD (Mannuzza et al., 2008; Molina et al., 2007). As such, the study does not inform medication issues in the course of ADHD to SUD. Larger, future studies focusing on medication in this risk trajectory are warranted. The study does not assess the role of peer relationships which have been established as strong predictors of adolescent substance use even after taking parenting dimensions into consideration (Barnes & Farrell, 1992). Such relationships would be of interest in future more focused investigations relating risk factors in the trajectory from ADHD to SUD. Finally, the control group (non ADHD

healthy group) consisted of only 3 participants who developed SUD, and therefore the study was not adequately powered to inform parenting risks pertaining to development of SUD in children without ADHD. Future focused investigations of parenting using larger, longitudinal samples of children with ADHD and CD and without ADHD who develop SUD would further elucidate risk and protective mechanisms to inform preventive interventions.

Acknowledgments

Data collection for this investigation was supported by NIMH R01 MH-53063 to Barbara Geller, M.D. Investigator effort was supported by NIDA/AACAP K12 DA000357 to Mini Tandon, D.O., and NIMH R01 MH 64769-02 to Joan Luby, M.D.

REFERENCES

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders (4th ed.) (DSM-IV). Washington, D.C.: APA; 1994.

Andersson P, Eisemann M. Parental rearing and individual vulnerability to drug addiction: A controlled study in a Swedish sample. Nordic Journal of Psychiatry. 2003; 57:147–156. [PubMed: 12745778]

Armstrong TD, Costello EJ. Community studies on adolescent substance use, abuse, or dependence and psychiatric comorbidity. Journal of Consulting and Clinical Psychology. 2002; 70:1224–1239. [PubMed: 12472299]

Barkley R, DuPaul G, McMurray M. Comprehensive evaluation of attention deficit disorder with and without hyperactivity as defined by research criteria. Journal of Consulting and Clinical Psychology. 1990; 58:775–789. [PubMed: 2292627]

Barkley, R.; Murphy, K. Attention-deficit hyperactivity disorder, 3rd ed.: A clinical workbook. New York, NY US: Guilford Press; 2006.

Barnes GM, Farrell MP. Parental Support and Control as Predictors of Adolescent Drinking, Delinquency, and Related Problem Behaviors. Journal of Marriage and Family. 1992; 54:763–776.

Barnes GM, Hoffman JH, Welte JW, Farrell MP, Dintcheff BA. Effects of Parental Monitoring and Peer Deviance on Substance Use and Delinquency. Journal of Marriage and Family. 2006; 68:1084–1104.

Bauman K, Ennett S, Foshee V, Pemberton M, King T, Koch G. Influence of a Family Program on Adolescent Smoking and Drinking Prevalence. Prevention Science. 2002; 3:35–42. [PubMed: 12002557]

Baumrind D. The Influence of Parenting Style on Adolescent Competence and Substance Use. The Journal of Early Adolescence. 1991; 11:56–95.

Biederman J, Monuteaux M, Mick E, Wilens T, Fontanella J, Poetzl K, Kirk T, Masse J, Faraone S. Is Cigarette Smoking a Gateway to Alcohol and Illicit Drug Use Disorders? A Study of Youths with and without Attention Deficit Hyperactivity Disorder. Biological Psychiatry. 2006; 59:258–264. [PubMed: 16154546]

Biederman J, Monuteaux MC, Mick E, Spencer T, Wilens TE, Silva JM, Snyder LE, Faraone SV. Young adult outcome of attention deficit hyperactivity disorder: a controlled 10-year follow-up study. Psychological Medicine. 2006; 36:167–179. [PubMed: 16420713]

Biederman J, Wilens T, Mick E, Faraone S, Weber W, Curtis S, Thornell A, Pfister K, Jetton J, Soriano J. Is ADHD a risk factor for psychoactive substance use disorders? Findings from a four-year prospective follow-up study. Journal of the American Academy of Child and Adolescent Psychiatry. 1997; 36:21–29. [PubMed: 9000777]

Bird H, Gould M, Staghezza B. Aggregating data from multiple informants in child psychiatry epidemiological research. Journal of the American Academy of Child and Adolescent Psychiatry. 1992; 31:78–85. [PubMed: 1537785]

Bird HR, Canino G, Rubiostipec M, Ribera JC. Further Measures of the Psychometric Properties of the Childrens Global Assessment Scale. Archives of General Psychiatry. 1987; 44:821–824. [PubMed: 3632256]

Blackson TC, Tarter RE, Mezzich AC. Interaction between childhood temperament and parental discipline practices on behavioral adjustment in preadolescent sons of substance abuse and normal fathers. American Journal of Drug and Alcohol Abuse. 1996; 22:335–348. [PubMed: 8841683]

Bogenschneider K, Wu M-y, Raffaelli M, Tsay JC. Parent Influences on Adolescent Peer Orientation and Substance Use: The Interface of Parenting Practices and Values. Child Development. 1998; 69:1672–1688. [PubMed: 9914646]

Bronte-Tinkew J, Moore KA, Carrano J. The Father-Child Relationship, Parenting Styles, and Adolescent Risk Behaviors in Intact Families. Journal of Family Issues. 2006; 27:850–881.

Brook DW, Brook JS, Zhang C, Koppel J. Association between attention-deficit/hyperactivity disorder in adolescence and substance use disorders in adulthood. Archives of Pediatrics and Adolescent Medicine. 2010; 164:930–934. [PubMed: 20921350]

Charach A, Yeung E, Climans T, Lillie E. Childhood Attention-Deficit/Hyperactivity Disorder and Future Substance Use Disorders: Comparative Meta-Analyses. Journal of the American Academy of Child and Adolescent Psychiatry. 2011; 50:9–21. [PubMed: 21156266]

Chassin L, Handley ED. Parents and families as contexts for the development of substance use and substance use disorders. Psychol Addict Behav. 2006; 20:135–137. discussion 140–132. [PubMed: 16784356]

Chassin L, Pillow DR, Curran PJ, Molina BS, Barrera M Jr. Relation of parental alcoholism to early adolescent substance use: a test of three mediating mechanisms. Journal of Abnormal Psychology. 1993; 102:3–19. [PubMed: 8436697]

Chassin L, Pitts SC, DeLucia C, Todd M. A longitudinal study of children of alcoholics: predicting young adult substance use disorders, anxiety, and depression. Journal of Abnormal Psychology. 1999; 108:106–119. [PubMed: 10066997]

Chassin L, Presson CC, Rose J, Sherman SJ, Davis MJ, Gonzalez JL. Parenting style and smoking-specific parenting practices as predictors of adolescent smoking onset. Journal of Pediatric Psychology. 2005; 30:333–344. [PubMed: 15863430]

Chassin L, Presson CC, Rose JS, Sherman SJ. Maternal socialization of adolescent smoking: Intergenerational transmission of smoking-related beliefs. Psychology of Addictive Behaviors. 1998; 12:206–216.

Choquet M, Hassler C, Morin D, Falissard B, Chau N. Perceived parenting styles and tobacco, alcohol and cannabis use among French adolescents: gender and family structure differentials. Alcohol and Alcoholism. 2008; 43:73–80. [PubMed: 17932077]

Chronis AM, Lahey BB, Pelham WE Jr, Williams SH, Baumann BL, Kipp H, Jones HA, Rathouz PJ. Maternal depression and early positive parenting predict future conduct problems in young children with attention-deficit/hyperactivity disorder. Developmental Psychology. 2007; 43:70–82. [PubMed: 17201509]

Coombs RH, Landsverk J. Parenting Styles and Substance Use during Childhood and Adolescence. Journal of Marriage and the Family. 1988; 50:473–482.

Deas D, Riggs P, Langenbucher J, Goldman M, Brown S. Adolescents are not adults: developmental considerations in alcohol users. Alcoholism, Clinical and Experimental Research. 2000; 24:232–237.

Dick DM, Pagan JL, Viken R, Purcell S, Kaprio J, Pulkkinen L, Rose RJ. Changing Environmental Influences on Substance Use Across Development. Twin Research and Human Genetics. 2007; 10:315–326. [PubMed: 17564520]

Elkins IJ, McGue M, Iacono WG. Prospective effects of attention-deficit/hyperactivity disorder, conduct disorder, and sex on adolescent substance use and abuse. Archives of General Psychiatry. 2007; 64:1145–1152. [PubMed: 17909126]

Enns MW, Cox BJ, Clara I. Parental bonding and adult psychopathology: results from the US National Comorbidity Survey. Psychological Medicine. 2002; 32:997–1008. [PubMed: 12214798]

Eyberg SM, Boggs SR, Algina J. Parent-child interaction therapy: a psychosocial model for the treatment of young children with conduct problem behavior and their families. Psychopharmacology Bulletin. 1995; 31:83–91. [PubMed: 7675994]

Fergusson DM, Horwood LJ, Ridder EM. Conduct and attentional problems in childhood and adolescence and later substance use, abuse and dependence: results of a 25-year longitudinal study. Drug and Alcohol Dependence. 2007; 88(Suppl 1):S14–S26. [PubMed: 17292565]

Flannery DJ, Williams LL, Vazsonyi AT. WHO ARE THEY WITH AND WHAT ARE THEY DOING? Delinquent Behavior, Substance Use, and Early Adolescents' After-School Time. American Journal of Orthopsychiatry. 1999; 69:247–253. [PubMed: 10234390]

Flory K, Lynam D. The Relation Between Attention Deficit Hyperactivity Disorder and Substance Abuse: What Role Does Conduct Disorder Play? Clinical Child and Family Psychology Review. 2003; 6:1–16. [PubMed: 12659448]

Geller B, Cooper TB, Sun K, Zimerman B, Frazier J, Williams M, Heath J. Double-blind and placebo-controlled study of lithium for adolescent bipolar disorders with secondary substance dependency. Journal of the American Academy of Child and Adolescent Psychiatry. 1998; 37:171–178. [PubMed: 9473913]

Geller B, Tillman R, Craney JL, Bolhofner K. Four-year prospective outcome and natural history of mania in children with a prepubertal and early adolescent bipolar disorder phenotype. Archives of General Psychiatry. 2004; 61:459–467. [PubMed: 15123490]

Geller, B.; William, M.; Zimerman, B.; Frazier, J. Washington University in St. Louis Kiddie Schedule for Affective Disorders and Schizophrenia (WASH-U-KSADS). Washington University in St Louis; 1996.

Geller B, Zimerman B, Williams M, Bolhofner K, Craney JL, DelBello MP, Soutullo C. Reliability of the Washington University in St. Louis Kiddie Schedule for Affective Disorders and Schizophrenia (WASH-U-KSADS) mania and rapid cycling sections. Journal of the American Academy of Child and Adolescent Psychiatry. 2001; 40:450–455. [PubMed: 11314571]

Geller B, Zimerman B, Williams M, Bolhofner K, Craney JL, Delbello MP, Soutullo CA. Diagnostic characteristics of 93 cases of a prepubertal and early adolescent bipolar disorder phenotype by gender, puberty and comorbid attention deficit hyperactivity disorder. Journal of Child and Adolescent Psychopharmacology. 2000; 10:157–164. [PubMed: 11052405]

Griffin KW, Botvin GJ, Scheier LM, Diaz T, Miller NL. Parenting practices as predictors of substance use, delinquency, and aggression among urban minority youth: moderating effects of family structure and gender. Psychology of Addictive Behaviors. 2000; 14:174–184. [PubMed: 10860116]

Hollingshead, AB. Four Factor Index of Social Status. Yale University Department of Sociology; 1976.

Kaplow JB, Curran PJ, Dodge KA. Conduct Problems Prevention Research Group. Child, parent, and peer predictors of early-onset substance use: a multisite longitudinal study. Journal of Abnormal Child Psychology. 2002; 30:199–216. [PubMed: 12041707]

Kendler KS, Davis CG, Kessler RC. The familial aggregation of common psychiatric and substance use disorders in the National Comorbidity Survey: a family history study. British Journal of Psychiatry. 1997; 170:541–548. [PubMed: 9330021]

Kendler KS, Myers J, Prescott CA. Parenting and adult mood, anxiety and substance use disorders in female twins: an epidemiological, multi-informant, retrospective study. Psychological Medicine. 2000; 30:281–294. [PubMed: 10824649]

King KM, Chassin L. Mediating and moderated effects of adolescent behavioral undercontrol and parenting in the prediction of drug use disorders in emerging adulthood. Psychology of Addictive Behaviors. 2004; 18:239–249. [PubMed: 15482079]

Lamb, ME. The role of the father in child development. John Wiley & Sons; 1997.

Lamb ME. The History of Research on Father Involvement. Marriage & Family Review. 2000; 29:23–42.

Lee SS, Humphreys KL, Flory K, Liu R, Glass K. Prospective association of childhood attention-deficit/hyperactivity disorder (ADHD) and substance use and abuse/dependence: a meta-analytic review. Clinical Psychology Review. 2011; 31:328–341. [PubMed: 21382538]

Lynskey MT, Hall W. Attention deficit hyperactivity disorder and substance use disorders: Is there a causal link? Addiction. 2001; 96:815–822. [PubMed: 11399213]

Mannuzza S, Klein RG, Bessler A, Malloy P, LaPadula M. Adult outcome of hyperactive boys: Educational achievement, occupational rank, and psychiatric status. Archives of General Psychiatry. 1993; 50:565–576. [PubMed: 8317950]

Mannuzza S, Klein RG, Bessler A, Malloy P, LaPadula M. Adult psychiatric status of hyperactive boys grown up. The American Journal of Psychiatry. 1998; 155:493–498. [PubMed: 9545994]

Mannuzza S, Klein RG, Truong NL, Moulton JL, Roizen ER, Howell KH, Castellanos FX. Age of methylphenidate treatment initiation in children with ADHD and later substance abuse: prospective follow-up into adulthood. The American Journal of Psychiatry. 2008; 165:604–609. [PubMed: 18381904]

Marshal MP, Chassin L. Peer Influence on Adolescent Alcohol Use: The Moderating Role of Parental Support and Discipline. Applied Developmental Science. 2000; 4:80–88.

Marsiglio W, Cohan M. Contextualizing Father Involvement and Paternal Influence. Marriage & Family Review. 2000; 29:75–95.

Martin CS, Winters KC. Diagnosis and assessment of alcohol use disorders among adolescents. Alcohol Health and Research World. 1998; 22:95–105. [PubMed: 15706783]

Mezzich AC, Tarter RE, Kirisci L, Feske U, Day BS, Gao Z. Reciprocal influence of parent discipline and child's behavior on risk for substance use disorder: a nine-year prospective study. American Journal of Drug and Alcohol Abuse. 2007; 33:851–867. [PubMed: 17994481]

Moffitt TE. Juvenile Delinquency and Attention Deficit Disorder: Boys' Developmental Trajectories from Age 3 to Age 15. Child Development. 1990; 61:893–910. [PubMed: 2364762]

Molina BSG, Flory K, Hinshaw SP, Greiner AR, Arnold LE, Swanson JM, Hechtman L, Jensen PS, Vitiello B, Hoza B, Pelham WE, Elliott GR, Wells KC, Abikoff HB, Gibbons RD, Marcus SUE, Conners CK, Epstein JN, Greenhill LL, March JS, Newcorn JH, Severe JB, Wigal T. Delinquent Behavior and Emerging Substance Use in the MTA at 36 Months: Prevalence, Course, and Treatment Effects. Journal of the American Academy of Child and Adolescent Psychiatry. 2007; 46:1028–1040. [PubMed: 17667481]

Molina BSG, Pelham WE Jr. Childhood predictors of adolescent substance use in a longitudinal study of children with ADHD. Journal of Abnormal Psychology. 2003; 112:497–507. [PubMed: 12943028]

Molina BSG, Smith BH, Pelham WE. Interactive effects of attention deficit hyperactivity disorder and conduct disorder on early adolescent substance use. Psychology of Addictive Behaviors. 1999; 13:348–358.

Parke RD. Father Involvement. Marriage & Family Review. 2000; 29:43–58.

Parker JS, Benson MJ. Parent-adolescent relations and adolescent functioning: self-esteem, substance abuse, and delinquency. Adolescence. 2004; 39:519–530. [PubMed: 15673227]

Patock-Peckham JA, King KM, Morgan-Lopez AA, Ulloa EC, Moses JM. Gender-specific mediational links between parenting styles, parental monitoring, impulsiveness, drinking control, and alcohol-related problems. Journal of Studies on Alcohol and Drugs. 2011; 72:247–258. [PubMed: 21388598]

Pires P, Jenkins JM. A growth curve analysis of the joint influences of parenting affect, child characteristics and deviant peers on adolescent illicit drug use. Journal of Youth and Adolescence. 2007; 36:169–183.

Puig-Antich, J.; Lukens, E.; Brent, D. Psychosocial Schedule for School Age Children–Revised in 1986 and 1987. Western Psychiatric Institute and Clinic; 1986.

Puig-Antich, J.; Ryan, N. The Schedule for Affective Disorders and Schizophrenia for School-Age Children (Kiddie-SADS)-1986. Pittsburgh, PA: Western Psychiatric Institute and Clinic; 1986.

Shaffer D, Gould MS, Brasic J, Ambrosini P, Fisher P, Bird H, Aluwahlia S. A Childrens Global Assessment Scale (Cgas). Archives of General Psychiatry. 1983; 40:1228–1231. [PubMed: 6639293]

Tarter RE, Kirisci L, Feske U, Vanyukov M. Modeling the pathways linking childhood hyperactivity and substance use disorder in young adulthood. Psychology of Addictive Behaviors. 2007; 21:266–271. [PubMed: 17563150]

Webster-Stratton C. Preventing conduct problems in Head Start children: strengthening parenting competencies. Journal of Consulting and Clinical Psychology. 1998; 66:715–730. [PubMed: 9803690]

Wilens TE, Biederman J, Mick E, Faraone SV, Spencer T. Attention deficit hyperactivity disorder (ADHD) is associated with early onset substance use disorders. Journal of Nervous and Mental Disease. 1997; 185:475–482. [PubMed: 9284860]

Wilens TE, Martelon M, Joshi G, Bateman C, Fried R, Petty C, Biederman J. Does ADHD predict substance-use disorders? A 10-year follow-up study of young adults with ADHD. Journal of the American Academy of Child and Adolescent Psychiatry. 2011; 50:543–553. [PubMed: 21621138]

Wills TA, Resko JA, Ainette MG, Mendoza D. Role of parent support and peer support in adolescent substance use: a test of mediated effects. Psychology of Addictive Behaviors. 2004; 18:122–134. [PubMed: 15238054]

Winters KC. Advances in the science of adolescent drug involvement: implications for assessment and diagnosis - experience from the United States. Current Opinion in Psychiatry. 2013; 26:318–324. [PubMed: 23695531]

Woodward L, Taylor E, Dowdney L. The Parenting and Family Functioning of Children with Hyperactivity. Journal of Child Psychology and Psychiatry. 1998; 39:161–169. [PubMed: 9669229]

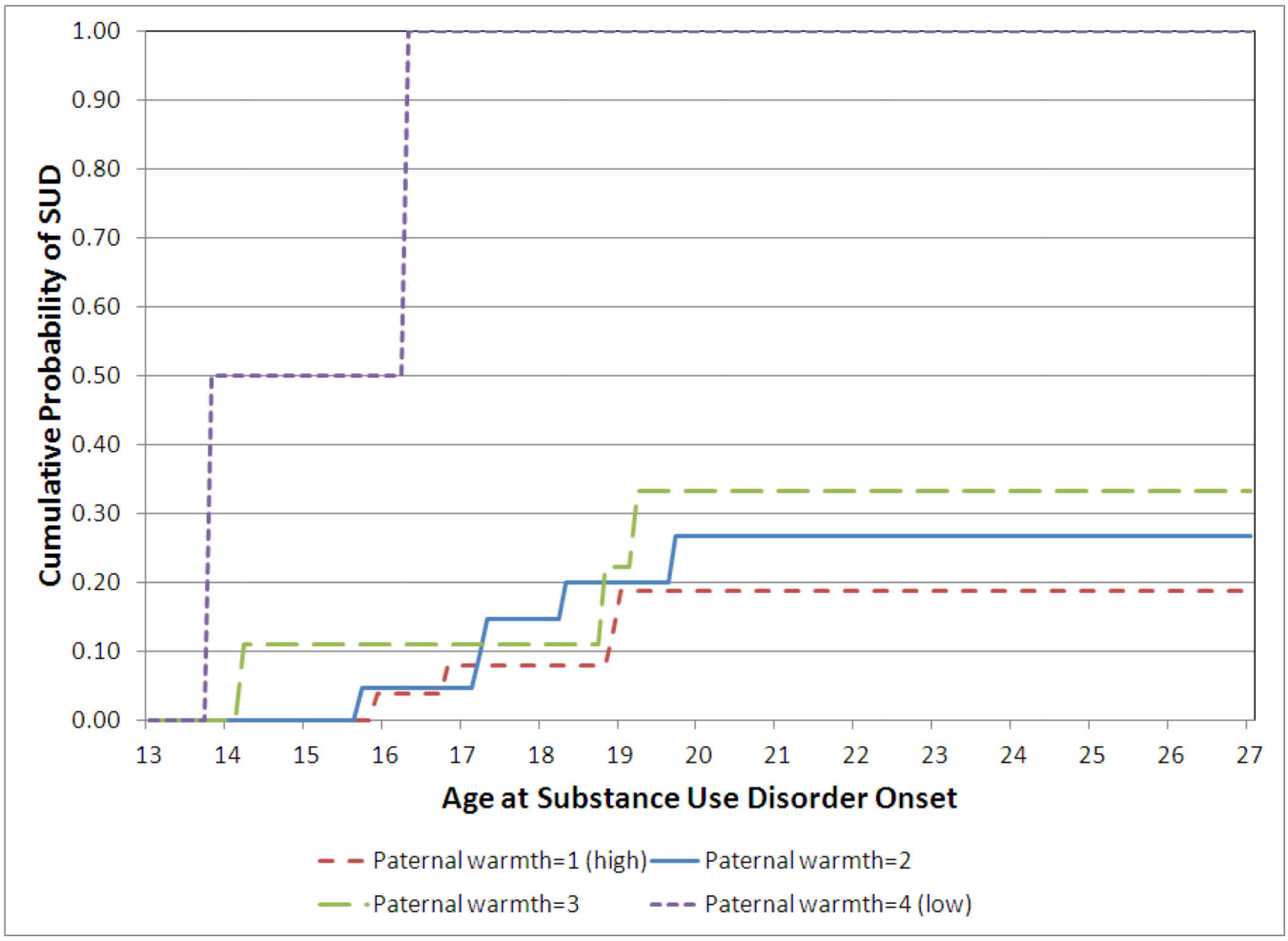

Figure 1. Kaplan-Meier Probability of SUD Onset in N=59 ADHD Participants by Paternal Warmth

ADHD=attention-deficit/hyperactivity disorder, SUD=substance use disorder;

The distribution of paternal warmth was N=26 score 1 (high), N=22 score 2, N=9 score 3, and N=2 score 4 (low).

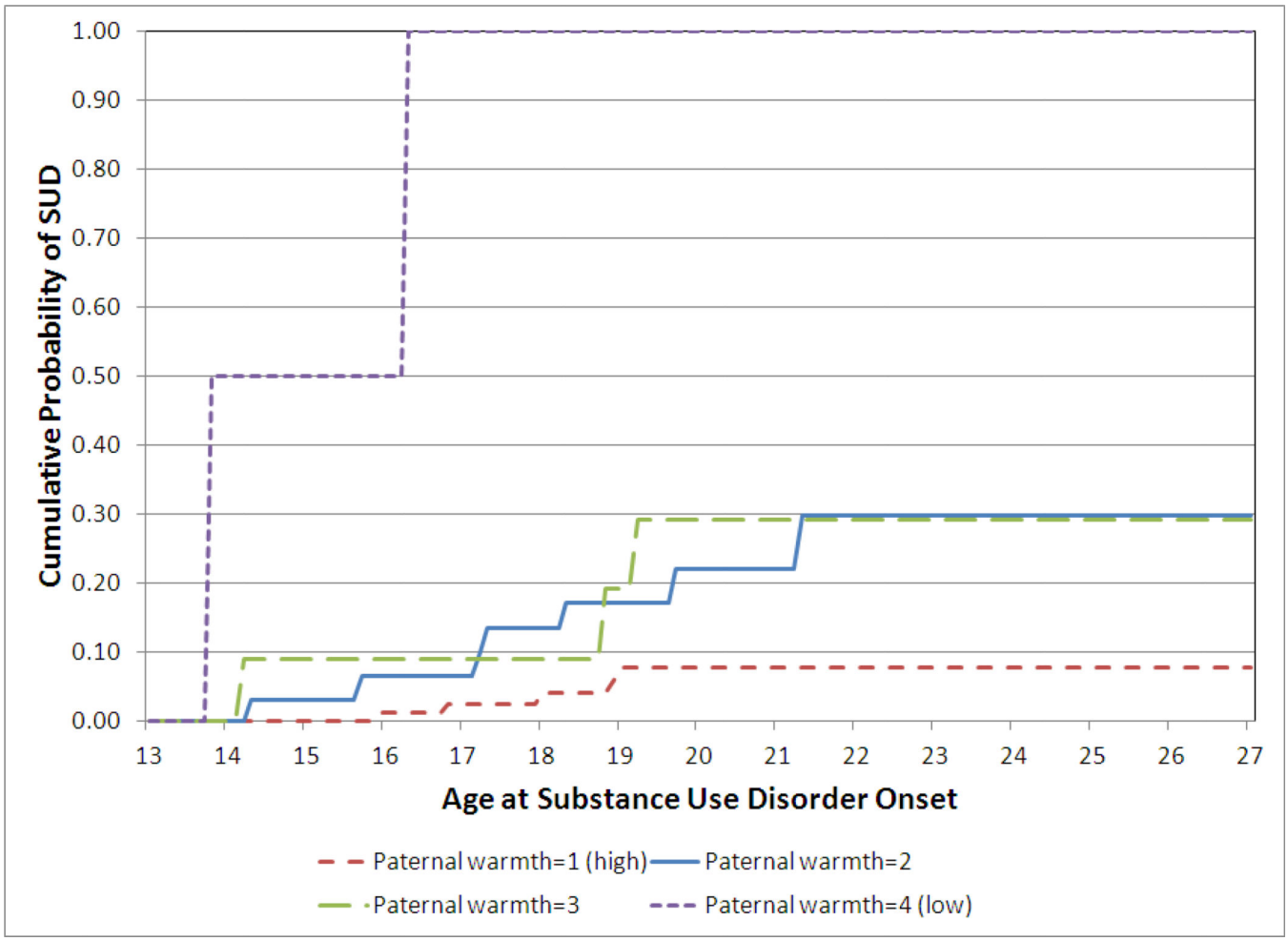

Figure 2. Kaplan-Meier Probability of SUD Onset in N=122 ADHD and HC Participants by Paternal Warmth

ADHD=attention-deficit/hyperactivity disorder, SUD=substance use disorder;

The distribution of paternal warmth was N=78 score 1 (high), N=31 score 2, N=11 score 3, and N=2 score 4 (low).

Table 1

Characteristics of the Sample

	Total (N=145)		ADHD (N=73)		HC (N=72)			
	%	N	%	N	%	N	χ^2	p
Male gender	67.6	98	76.7	56	58.3	42	5.59	0.018
SES								
5 (highest)	37.2	54	41.1	30	33.3	24	F.E.	0.725
4	42.1	61	41.1	30	43.1	31		
3	18.6	27	16.4	12	20.8	15		
2	2.1	3	1.4	1	2.8	2		
1 (lowest)	0.0	0	0.0	0	0.0	0		
Race								
Caucasian	89.7	130	90.4	66	88.9	64	F.E.	0.740
African American	7.6	11	8.2	6	6.9	5		
Asian	1.4	2	1.4	1	1.4	1		
Other	1.4	2	0.0	0	2.8	2		
Baseline living situation								
Both bio parents	78.7	111	65.2	45	91.7	66	F.E.	0.001
Both bio parents, joint custody	2.1	3	4.3	3	0.0	0		
Bio mom, step-dad	11.4	16	17.4	12	5.6	4		
Bio mom, significant other	0.7	1	1.4	1	0.0	0		
Bio mom only	7.1	10	11.6	8	2.8	2		
SUD during follow-up	14.5	21	24.7	18	4.2	3	12.29	<0.001
ODD	14.5	21	27.4	20	1.4	1	19.80	<0.001
Parental SUD	34.4	44	40.6	26	28.1	18	2.22	0.137
Maternal SUD	7.0	9	4.7	3	9.4	6	F.E.	0.492
Paternal SUD	29.9	38	38.1	24	21.9	14	3.98	0.046
Parental smoking	33.6	43	34.4	22	32.8	21	0.04	0.852
Maternal smoking	17.2	22	15.6	10	18.8	12	0.22	0.639
Paternal smoking	24.4	31	27.0	17	21.9	14	0.45	0.503
Parental ADHD	24.2	31	39.1	25	9.4	6	15.37	<0.001
Maternal ADHD	9.4	12	17.2	11	1.6	1	9.20	0.002
Paternal ADHD	17.3	22	27.0	17	7.8	5	8.15	0.004
Parental ASPD	4.7	6	7.8	5	1.6	1	F.E.	0.208
Maternal ASPD	0.0	0	0.0	0	0.0	0	--	--
Paternal ASPD	4.7	6	7.9	5	1.6	1	F.E.	0.115
Parental MDD	53.1	68	70.3	45	35.9	23	15.18	<0.001
Maternal MDD	44.5	57	57.8	37	31.3	20	9.14	0.003
Paternal MDD	22.0	28	33.3	21	10.9	7	9.27	0.002
Parental anxiety	48.4	62	59.4	38	37.5	24	6.13	0.013
Maternal anxiety	33.6	43	48.4	31	18.8	12	12.64	<0.001
Paternal anxiety	20.5	26	19.0	12	21.9	14	0.16	0.693

	Total (N=145)		ADHD (N=73)		HC (N=72)			
	%	N	%	N	%	N	χ^2	p
Low parental warmth	39.0	55	59.4	41	19.4	14	23.67	<0.001
Low maternal warmth	17.0	24	27.5	19	6.9	5	10.58	0.001
Low paternal warmth	34.5	48	54.4	37	15.5	11	23.27	<0.001
	Mean	SD	Mean	SD	Mean	SD	t	p
Baseline age	11.42	3.49	11.78	4.07	11.06	2.76	1.24	0.216
Final assessment age	21.04	2.75	20.93	2.76	21.15	2.76	0.49	0.625
ADHD severity score	7.44	7.79	14.48	4.38	0.31	0.97	26.95	<0.001

A Baseline Age Distribution

	Total (N=145)	ADHD (N=73)	HC (N=72)
Age 7	13.8 (20)	15.1 (11)	12.5 (9)
Age 8	16.6 (24)	12.3 (9)	20.8 (15)
Age 9	18.6 (27)	23.3 (17)	13.9 (10)
Age 10	4.8 (7)	4.1 (3)	5.6 (4)
Age 11	9.0 (13)	8.2 (6)	9.7 (7)
Age 12	7.6 (11)	6.8 (5)	8.3 (6)
Age 13	5.5 (8)	2.7 (2)	8.3 (6)
Age 14	8.3 (12)	8.2 (6)	8.3 (6)
Age 15	4.8 (7)	1.4 (1)	8.3 (6)
Age 16	11.0 (16)	17.8 (13)	4.2 (3)

B Age Distribution at Last Assessment

	Total (N=145)	ADHD (N=73)	HC (N=72)
Age 13	1.4 (2)	2.7 (2)	0.0 (0)
Age 14	0.0 (0)	0.0 (0)	0.0 (0)
Age 15	0.7 (1)	1.4 (1)	0.0 (0)
Age 16	0.0 (0)	0.0 (0)	0.0 (0)
Age 17	9.0 (13)	8.2 (6)	9.7 (7)
Age 18	15.2 (22)	8.2 (6)	22.2 (16)
Age 19	13.1 (19)	11.0 (8)	15.3 (11)
Age 20	13.1 (19)	19.2 (14)	6.9 (5)
Age 21	14.5 (21)	21.9 (16)	6.9 (5)
Age 22	6.9 (10)	6.8 (5)	6.9 (5)
Age 23	9.7 (14)	8.2 (6)	11.1 (8)
Age 24	6.9 (10)	5.5 (4)	8.3 (6)
Age 25	6.2 (9)	4.1 (3)	8.3 (6)
Age 26	2.8 (4)	1.4 (1)	4.2 (3)
Age 27	0.7 (1)	1.4 (1)	0.0 (0)

ADHD=attention-deficit/hyperactivity disorder, ASPD=antisocial personality disorder, F.E. = Fisher's Exact Test, HC=healthy control, MDD=major depressive disorder, ODD=oppositional defiant disorder, SES=socioeconomic status, SUD=substance use disorder

Table 2

Multivariate Cox Proportional Hazards Models of SUD Onset in ADHD Participants

2A – Stepwise Selection Model	N	Est.	SE	HR	95% CI	χ^2	p
Overall model	59					15.54	<0.001
Maternal SUD		4.01	1.04	55.04	(7.1, 425.6)	14.75	<0.001
Paternal warmth (1=high, 4=low)		1.06	0.37	2.90	(1.4, 6.0)	8.12	0.004

2B – Covariates Added	N	Est.	SE	HR	95% CI	χ^2	p
Overall model	59					17.33	0.138
Male gender		1.34	0.89	3.80	(0.7, 21.6)	2.27	0.132
ODD		0.29	0.82	1.34	(0.3, 6.6)	0.13	0.721
Maternal SUD		4.20	1.64	66.94	(2.7, 1660.9)	6.58	0.010
Paternal SUD		−0.82	0.82	0.44	(0.1, 2.2)	1.01	0.315
Maternal ADHD		0.78	0.89	2.19	(0.4, 12.6)	0.77	0.381
Paternal ADHD		1.02	0.74	2.77	(0.6, 11.8)	1.89	0.170
Maternal MDD		−0.67	0.73	0.51	(0.1, 2.2)	0.83	0.362
Paternal MDD		−0.44	0.92	0.65	(0.1, 3.9)	0.22	0.636
Maternal anxiety		0.53	0.73	1.70	(0.4, 7.1)	0.53	0.466
Paternal anxiety		0.43	0.98	1.53	(0.2, 10.5)	0.19	0.662
Maternal warmth (1=high, 4=low)*		0.48	0.68	1.62	(0.4, 6.2)	0.50	0.479
Paternal warmth (1=high, 4=low)		1.50	0.52	4.49	(1.6, 12.4)	8.47	0.004

*Maternal warmth was measured on a 1–4 scale, although no participants had scores of 3 or 4;

ADHD=attention-deficit/hyperactivity disorder, CI=confidence interval, Est.=Estimate, HR=hazard ratio, MDD=major depressive disorder, ODD=oppositional defiant disorder, SE=standard error, SUD=substance use disorder

Table 3

Multivariate Cox Proportional Hazards Models of SUD Onset in ADHD and HC Participants

3A – Stepwise Selection Model	N	Est.	SE	HR	95% CI	χ^2	p
Overall model	122					18.82	<0.001
ADHD		1.92	0.79	6.85	(1.5, 32.1)	5.97	0.015
Maternal SUD		3.03	0.85	20.64	(3.9, 108.7)	12.76	<0.001
Paternal warmth (1=high, 4=low)		0.97	0.32	2.64	(1.4, 4.9)	9.21	0.002

3B – Covariates Added	N	Est.	SE	HR	95% CI	χ^2	p
Overall model	122					27.70	0.010
Male gender		1.89	0.85	6.65	(1.3, 35.2)	4.96	0.026
ADHD		1.50	0.92	4.49	(0.7, 27.0)	2.69	0.101
ODD		−0.32	0.76	0.73	(0.2, 3.2)	0.18	0.673
Maternal SUD		3.11	0.99	22.44	(3.3, 154.7)	9.97	0.002
Paternal SUD		−0.58	0.71	0.56	(0.1, 2.3)	0.67	0.414
Maternal ADHD		1.27	0.75	3.55	(0.8, 15.6)	2.82	0.093
Paternal ADHD		0.77	0.68	2.15	(0.6, 8.2)	1.26	0.261
Maternal MDD		−1.09	0.65	0.34	(0.1, 1.2)	2.84	0.092
Paternal MDD		−0.80	0.72	0.45	(0.1, 1.9)	1.22	0.269
Maternal anxiety		1.69	0.69	5.43	(1.4, 20.9)	6.05	0.014
Paternal anxiety		0.71	0.72	2.04	(0.5, 8.4)	0.98	0.322
Maternal warmth (1=high, 4=low)*		0.99	0.67	2.69	(0.7, 9.9)	2.20	0.138
Paternal warmth (1=high, 4=low)		1.25	0.41	3.50	(1.6, 7.8)	9.30	0.002

*Maternal warmth was measured on a 1–4 scale, although no participants had scores of 3 or 4;

ADHD=attention-deficit/hyperactivity disorder, CI=confidence interval, Est.=Estimate, HC=healthy control, HR=hazard ratio, MDD=major depressive disorder, ODD=oppositional defiant disorder, SE=standard error, SUD=substance use disorder

Table 4

Substances Used Through 8-Year Follow-up

Diagnostic Group	Substance Through 8-Year Follow-Up	N
ADHD	Alcohol	4
	Marijuana	5
	Alcohol & Marijuana	1
	Unknown	8
HC	Alcohol	1
	Marijuana	0
	Alcohol & Marijuana	2
	Unknown	0