Parent and Child Psychopathology and Suicide Attempts among Children of Parents with Alcohol Use Disorder

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Abstract

Parents with psychopathology such as alcohol use disorder (AUD) that confers risk for suicide attempt (SA) may have children who are more likely to develop such psychopathology and to attempt suicide, suggesting that risk may be "transmitted" from parents to children. We examined this phenomenon during the transition from childhood to adolescence, when risk for SA increases dramatically. A cohort of 418 children were examined at average age 9.4 (range 7–14) years at enrollment (Time 1, childhood) and approximately five years later, prior to reaching age 18 (Time 2, adolescence). One or both biological parents, oversampled for AUD, were also interviewed. Structural equation models (SEM) examined father-child, mother-child, and either/both parent-child associations. The primary outcome was SA over follow-up among offspring, assessed at Time 2. As hypothesized, parental antisocial personality disorder predicted conduct disorder symptoms in offspring both during childhood and adolescence (parent-child model, father-child model) and maternal AUD predicted conduct disorder symptoms during childhood (mother-child model). However, we did not find evidence to support transmission of depression from parents to offspring either during childhood or adolescence, and parent psychopathology did not show statistically significant associations with SA during adolescence. In conclusion, we conducted a rare study of parent-to-child "transmission" of risk for SA that used a prospective research design, included diagnostic interviews with both parents and offspring, and examined the transition from childhood to adolescence, and the first such study in children of parents with AUD. Results provided mixed support for hypothesized parent-child associations.

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Keywords

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1. Introduction

A suicide attempt (SA) is more likely to occur during adolescence than at any other time in the life course (Kessler, Borges, & Walters, 1999). SA during youth is a potent risk factor for eventual suicide (Hawton & Harriss, 2007) and indeed some experts regard it as "*the single most potent risk factor for youth suicide*", p.375 (Bridge, Goldstein, & Brent, 2006). Accordingly, the prevention of suicide in youth demands a focus on the understanding and prevention of SA.

Prior suicidal behavior, depression, and externalizing psychopathology (e.g., impulsive aggression, alcohol and drug use disorders, conduct problems) confer risk for SA and suicide during adolescence (Brent, Johnson, Perper et al., 1994; Fergusson, Woodward, & Horwood, 2000; Gould, King, Greenwald et al., 1998; Shaffer, Gould, Fisher et al., 1996). It has been argued that models of suicidal behavior during youth must account for the potential transmission of such risk factors from parents to offspring (Brent & Mann, 2006). In support of this argument, parents with histories of suicidal behavior have children that are at increased risk for SA (Kim, Seguin, Therrien et al., 2005; Lieb, Bronisch, Hofler et al., 2005; Melhem, Brent, Ziegler et al., 2007) and parents displaying key risk factors for suicidal behavior including depressive disorders and various forms of externalizing psychopathology have children that are at increased risk for these difficulties (Brent, Oquendo, Birmaher et al., 2002; Harold, Rice, Hay et al., 2010; Kim, Seguin, Therrien et al., 2005; van Goozen, Fairchild, Snoek et al., 2007). Integrating such data, Brent and Mann (2006) proposed that parental psychopathology including depression and impulsive aggression, respectively, are key influences on these difficulties in offspring which in turn promote risk for SA during youth.

Attempted suicide is rare during childhood and prevalence increases dramatically in adolescence (Lewinsohn, Rohde, Seeley et al., 2001). Therefore, prospective studies of SA etiology that span the period from childhood to adolescence when risk emerges may be especially informative, particularly if data are gathered from both parents and youth, yet few published studies have met these standards (Fergusson, Woodward, & Horwood, 2000; King, Kerr, Passarelli et al., 2010; Lieb, Bronisch, Hofler et al., 2005; Melhem, Brent, Ziegler et al., 2007). These rigorous prospective studies were an important step forward although they were limited to the use of low-risk community samples (Fergusson, Woodward, & Horwood, 2000; Lieb, Bronisch, Hofler et al., 2005) or very high risk clinical, psychiatric samples (King, Kerr, Passarelli et al., 2010; Melhem, Brent, Ziegler et al., 2007), with unclear generalizability to other populations.

Although Brent and Mann (2006) presented their ideas to explain the influence of a parent who has attempted suicide on their adolescent child's risk for SA, their ideas have wider implications. For example, individuals with AUD are at elevated risk for suicidal behavior (Kessler et al., 1999; Wilcox, Conner, & Caine, 2004) as well as major risk factors for

suicidal behavior including depression and externalizing psychopathology (Hasin, Stinson, Ogburn et al., 2007), with increased potential that their children will develop such difficulties (Glowinski et al., 2004), yet there is a meager database on the influences of parents with AUD on offspring SA. Indeed, we were only able to identify one investigation of SA among children of parents with AUD that contained detailed diagnostic interviews with both parents and children (Glowinski et al., 2004). The study was limited to a cross-sectional design. Interestingly, it showed that paternal psychopathology may be especially influential on offspring SA.

The purpose of the current investigation was to add to the small database of prospective studies of SA in youth that span childhood and adolescence and include detailed interviews from parents and children (Fergusson et al., 2000; King et al., 2010; Lieb et al., 2005; Melhem et al., 2007), and to conduct the first such study in children of parents with AUD. Informed by Brent and Mann's (2006) ideas, we examined the influences of parent SA, depression, and externalizing psychopathology on these behaviors in youth assessed during childhood and approximately five years later, during adolescence. We hypothesized that parent depression and externalizing psychopathology would show direct associations with these behaviors in youth which would, in turn, be directly associated with SA in youth. We also hypothesized that a history of parent SA would show a direct association with SA in offspring. As discussed above, parent(s) with AUD and their offspring are an important population to test these hypotheses due to elevated rates of psychopathology and suicidal behavior. Along with an analysis of parental influences generally, we analyzed mother-child and father-child relationships in separate models because maternal and paternal influences on offspring psychopathology often differ (Connell & Goodman, 2002), a pattern that may extend to the study of SA among children of parents with AUD (Glowinski et al., 2004).

2. Method

2.1 Procedure

Data were gathered for the Collaborative Study on the Genetics of Alcoholism (COGA), a multicenter family study in the U.S. COGA examines individuals with alcohol dependence recruited from treatment centers (probands), first degree relatives of these individuals, as well as non-alcohol dependent comparison families recruited through various population sources (e.g., motor vehicle registration). COGA investigators have created several datasets from various studies. For the current analysis, we examined a cohort of children who were on average age 9.4 years at enrollment (range 7–14 years) and who were reassessed approximately five years later, with all reassessments occurring prior to reaching age 18. Individuals who were not reassessed at 5 years, approximately 22%, were excluded from analyses. At least one biological parent to the children (i.e., adult COGA participant) was also interviewed around the time of the baseline assessment. The cohort was assembled between 1991 and 1998 and the follow-ups were completed between 1997 and 2004. An exemption from the University of Rochester IRB was granted to perform these secondary analyses of de-identified COGA data.

2.2. Measures

Information about the presence/absence of alcohol use disorders, drug use disorders, mood disorders, SA, and conduct disorder and antisocial personality disorder among parents were obtained with the adult version of the Semi-Structured Assessment for the Genetics of Alcoholism, SSAGA (Bucholz et al., 1994). Diagnostic sections of the SSAGA have been intensively studied and show solid reliability (Bucholz et al., 1994; Bucholz et al., 1995; Kramer et al., 2009) and validity (Hesselbrock, Easton, Bucholz et al., 1999). A child/adolescent version of the SSAGA was administered to children ages 17 and younger (Kuperman, Schlosser, Kramer et al., 2001). This youth version was primarily based on the adult SSAGA, but used age-appropriate language. The various diagnostic sections of the child/adolescent version are reliable, with kappa coefficients averaging 0.72 (Kuperman, Schlosser, Kramer et al., 2001). An abbreviated version of the SSAGA, the Family History Module (Rice, Reich, Bucholz et al., 1995), was administered to obtain data from one parent about the other when one of the parents was not available for interview. This abbreviated interview has demonstrated reliability (Rice, Reich, Bucholz et al., 1995) and includes assessments of substance use disorders, mood disorders, conduct/antisocial personality disorder, and SA. For the current analyses diagnoses were based on criteria described in Diagnostic and Statistical Manual, 3rd edition, revised (American Psychiatric Association, 1987).

2.2.1. Time 1 (T1) assessments in children—T1 data were assessed during the baseline assessment and covered lifetime history up to the time of the T1 assessment. We created a continuous measure of T1 suicidality with range 0–2: 0) no history of suicidal thoughts or attempt; 1) history of suicidal thoughts only; 2) history of suicide attempt (SA). The latter was assessed with the item "Have you ever tried to kill yourself?" (Preuss, Schuckit, Smith, et al., 2002). Conduct disorder symptoms were used to assess the externalizing domain (Dick, 2007; Krueger & Markon, 2006). Conduct disorder symptoms were selected to assess external symptoms in youth because they are in evidence at an early age, prior to the onset of other externalizing variables that confer risk for SA in youth assessed by the SSAGA, for example substance use disorders (Lahey, Miller, Gordon et al., 1999; Wagner & Anthony, 2002). We created a continuous variable with range 0–2: 0) no conduct disorder symptoms, 1) 1-plus symptoms but no diagnosis, 2) conduct disorder diagnosis. To assess depression we also created a continuous variable with range 0–2: 0) no history of depressed mood or anhedonia, 1) history of depressed mood and/or anhedonia lasting at least two weeks but no major depressive episode, and 2) major depressive episode. Depressive symptoms were coded if they were substance-induced or occurred independent of substance use. We used symptoms of conduct disorder and depression, as opposed to diagnoses alone, to increase sensitivity to detect psychopathology in youth.

2.2.2. T1 assessment in parents—T1 data in parents were generally obtained near to the time of the children's baseline assessment and covered lifetime history up to T1. History of SA was assessed with the same SA item used with children (described above). Externalizing psychopathology was assessed with presence or absence, respectively, of alcohol use disorder including alcohol abuse or dependence, drug use disorder including any non-alcohol abuse or dependence diagnosis, and antisocial personality disorder. Depression

was based on presence or absence of major depressive disorder episode including substance-induced depression and depression independent of substance use.

2.2.3. Time 2 (T2) assessments (children only)—T2 data were based on the time period since the T1 assessment. SA at T2 was assessed with the aforementioned SA item. For children who reported a history of SA at T1, we examined the date of the last SA to confirm that the attempt occurred over follow-up. Conduct disorder symptoms and depressive symptoms were assessed at T2 in offspring using the same measures described for T1.

2.3. Analyses

SA at T2 among children was the outcome in all analyses. For descriptive purposes, unadjusted comparisons between children with a suicide attempt at T2 vs. non-attempters at T2 were made on all predictors. For these comparisons Fisher's Exact Test for categorical predictors were used to handle cells with small numbers of observations (Fisher, 1954).

Three structural equation (SEM) models (Kaplan, 2000) were used to examine 1) mother-child, 2) father-child, and 3) parent-child relationships, respectively. The latter model was run if data from either parent (or both) were available, and a parent exposure (e.g., depression) was coded present if it were observed in either parent (or both). SEM is an extension of regression models to address limitations of the latter when applied to relationships involving multiple variables over time (Kaplan, 2000). In a regression model, there exists a clear distinction between dependent and independent variables and the model relates the dependent to a set of independent variables. SEM is designed to handle the fact that a variable may serve as both an independent and a dependent variable in an analysis; SA at T1 is an example in our study (see Figure 1). In all models, parents were assumed to influence their children rather than vice-versa and, among children, the prospective influences of T1 variables on T2 were also examined. For each SEM model, we assessed model fit using the Chi-square and Root Mean Square Error of Approximation (RMSEA) goodness of fit statistics (MacCallum, Browne, & Sugawara, 1996). We used probit link function and reported standardized coefficients in all the models. The unit of analysis when fitting SEM for the data was the family, rather than individual subjects as in standard regression, to account for the non-independence of data within families. All SEM analyses were performed using Mplus (Muthen & Muthen, 2006). The contribution of variables in statistical models were tested using two-sided tests at the $p < .05$ level.

3. Results

3.1. Descriptive data

Three hundred seven families participated and 7 families were excluded from analyses due to missing data. About two-thirds of families (N=203, 67.7%) had a single child participant, with mean 1.4 ± 0.6 children per family (range 1–4).

Descriptive results are provided in Table 1. The data are presented for the sample in the father-child SEM analysis (N=290 children of 199 fathers), the mother-child analysis (N=394 children of 269 mothers), and the parent-child analysis (N=418 children of 300

parents consisting of mother, father, or both). In each sample the data are stratified by the outcome, showing a comparison of children who made a suicide attempt at T2 to non-attempters at T2. Seventeen children of 12 fathers had a T2 suicide attempt (father-child sample), 18 children of 12 mothers had a T2 attempt (mother-child sample), and 19 children of 13 parents had a T2 attempt (parent-child sample). A large number of parents had a lifetime history of SA including 24 (12.1%) fathers, 39 (14.5%) mothers, and 60 (20.0%) either parent. Most parents had a history of alcohol use disorder including 162 (81.4%) fathers, 120 (44.6%) mothers, and 247 (82.3%) either parent.

Univariate comparisons on all predictors between children with a suicide attempt at T2 and non-attempters are presented in Table 1. None of the parental psychopathology variables including parent, father, or mother history of SA, AUD, drug use disorder, or antisocial personality disorder were associated with a T2 suicide attempt in offspring at a statistically significant level, with the lone exception of maternal drug use disorder history. With the exception of conduct disorder at T1, the other child predictors were associated with a suicide attempt at T2 including depressive symptoms at T1 and T2, conduct disorder symptoms at T2, and SA at T1. Among the child covariates (age, sex, race-ethnicity), only sex was statistically significant, with girls being more likely to make an attempt at T2.

3.2. SEM results

The SEM results are presented in Figure 1 including results for the father-child model (A), mother-child model (B), and parent-child model (C). Tests of model fit indicated the Chi-square test was significant (p<0.001) and the RMSEA value was between 0.05 and 0.1 for each model, indicative of adequate fit: father-child model, $X^2(30) = 83.2$, p 0.001 and RMSEA = 0.076; mother-child model, $X^2(30) =103.1$, p 0.001 and RMSEA = 0.071; parent-child model, $X^2(30) =99.5$, p 0.001 and RMSEA= 0.073. In each SEM model, female sex was associated with increased risk for SA in youth at T2; the other covariates (age, race) were not associated with SA at a statistically significant level (bottom of figure).

3.2.1. Father-child SEM model results (A)—These results show statistically significant paths to the outcome from child symptoms of depression at T2, conduct disorder at T2, and suicidality at T1 (bottom of figure). Moving distally from the outcome (and up the model), father antisocial personality disorder (ASPD) at T1 was associated with child conduct disorder symptoms at T1 and T2, symptoms of child conduct disorder and child depression at T1 were associated with child suicidality at T1, and symptoms of conduct disorder at T1 were associated with conduct disorder symptoms at T2. Finally, as depicted at the top of the model, father drug use disorder (DUD) at T1 was associated with father SA history at T1.

3.2.2. Mother-child SEM model results (B)—These results show, in the bottom half of the figure, paths to the outcome that are similar to those obtained in the father-child model. In the upper half of the model, there are some noteworthy differences from the father-child model; namely, in the mother-child model, maternal alcohol use disorder (AUD) was associated with child conduct disorder at T1 and maternal history of SA at T1 (whereas in the father-child model, there were no statistically significant paths from paternal AUD).

3.2.3. Parent-child SEM model results (C)—These results show, in the bottom half of the figure, paths to the outcome that are similar to those obtained in the other models. In the upper half of the parent-child model, parental ASPD at T1 was associated with child conduct disorder symptoms at T1 and T2 (similar to the father-child model). Several variables were associated with parent history of SA at T1 including parental AUD (similar to the mother-child model) and parental depression and ASPD (which were not associated with SA in mothers or fathers in the other models). Finally, the path coefficients from child depression at T1 to child depression at T2 were nearly identical in the three models although it did not reach statistical significance in the father-child model.

4. Discussion

4.1. Summary

The current analyses showed that child depressive symptoms and conduct disorder symptoms, an externalizing variable, were associated with suicide attempt (SA) during childhood and again when assessed approximately five years later, during adolescence, consistent with the theoretical ideas presented by Brent and Mann (2006). Depressive symptoms, conduct disorder symptoms, and suicidality assessed in childhood also predicted these symptoms later, during adolescence, illustrating continuity of psychopathology during development. We also found evidence of "transmission" of vulnerability from parents to offspring as predicted by Brent and Mann. In particular, parental antisocial personality disorder predicted conduct disorder symptoms in offspring both during childhood (T1) and adolescence (T2) (parent-child model, father-child model) and maternal alcohol use disorder predicted conduct disorder symptoms during childhood (mother-child model). However, we did not find evidence to support transmission of depression from parents to offspring either during childhood (T1) or adolescence (T2). Interestingly, none of the father, mother, or parent variables showed a statistically significant association with SA during adolescence (see comparisons in Table 1). Moreover, father, mother, and parent SA were not associated with child suicidality at T1 in any of the SEM models, inconsistent with our hypothesis of more or less direct transmission of suicidality from parents to offspring. Finally, consistent with Brent and Mann's ideas and the general literature, lifetime parental psychopathology including depression (i.e., major depressive episode) and externalizing variables (i.e., AUD, drug use disorder, and antisocial personality disorder) were associated with a history of parent SA in one or more SEM models (i.e., father, mother, parent).

4.2. Limitations and strengths

There were limitations of the study. We did not examine the role of genetic factors. The high prevalence of father AUD history (81.4%) in the cohort may have contributed to the nonsignificant association between parent AUD and parent SA in analyses of fathers but not mothers where there was a more optimal distribution of AUD history (44.6% of mothers) for detecting associations. The limited number of suicide attempts over follow-up among youth warrant cautious interpretation of nonsignificant results. For example we did not identify a statistically significant relationship between parent- and offspring suicidal behavior, an association shown in prior studies (Kim, Seguin, Therrien et al., 2005; Lieb, Bronisch, Hofler et al., 2005; Melhem, Brent, Ziegler et al., 2007). The limited number of suicide

attempters also ruled out more detailed analyses of sex differences, for example through separate analyses of male- and female youth. Although a limitation, all of the hypothesized paths to youth suicide attempts tested in our conceptual model were statistically significant with the lone exception of the path from parent SA to offspring SA (see figure 1). Therefore, the relatively small number of SA in youth was generally not a limiting factor in testing our conceptual model. Moreover, there is precedence for prospective analyses of a limited numbers of suicide attempts over time, attributable to the low incidence rate of suicidal behavior. For example, a prior test of several predictors emphasized by Brent and Mann (2006) contained 11 suicide attempts over follow-up (Melhem, Brent, Ziegler et al., 2007). Although it is true that researchers have analyzed some large and informative longitudinal community surveys such as the National Longitudinal Study of Adolescent Health that contained large numbers of suicide attempts (Borowsky, Ireland, & Resnick, 2001; Kidd, Henrich, Brookmeyer et al., 2006), such studies have not contained detailed interviews with parents and offspring. A large and informative Scandinavian registry analysis of suicide attempts in offspring contained data on parents and offspring (Stenager & Qin, 2008), although a limitation is the reliance on clinical records' data. We also did not examine important relational variables (e.g., suboptimal family environment) and negative life experiences (e.g., child abuse) that were discussed by Brent and Mann (2006). The sample is not population representative.

There were several strengths of the analyses. The current study featured a prospective design, detailed diagnostic assessments, and interviews with both parents and offspring, representing the first such study in an AUD sample. We tested a theoretical model that examined interrelationships among predictors including potential parent-to-child pathways in addition to tests of relationships between predictors and the SA outcome. A history of SA was common among parents in the sample, underscoring the importance of examining intergenerational pathways to suicidal behavior in families with AUD. The direct and clear phrasing of the SA item in the current study, "Have you ever tried to kill yourself?", likely elicited reports about suicidal acts containing at least some intent to die as opposed to more ubiquitous non-suicidal self-injury (Silverman, Berman, Sanddal et al., 2007), acknowledging that the phrasing of the SA item likely came at a cost to sensitivity.

5. Conclusions

Results of the current study reaffirm that depressive symptoms and externalizing behaviors (i.e., conduct disorder symptoms) confer risk for SA among adolescent children of parents with AUD, a vulnerable population. Moreover, the results suggest that when these risk factors present during childhood they are predictive of future difficulties during adolescence, when risk for SA peaks. Results also indicate that some forms of externalizing parental psychopathology (i.e., antisocial personality disorder in fathers, alcohol use disorders in mothers) predict offspring conduct disorder symptoms which, in turn, promote risk for SA, consistent with the idea that parental risk for suicidal behavior is transmitted to children through externalizing behaviors, among other factors. The data also suggest that child risk factors (i.e., depressive symptoms, conduct disorder symptoms, prior suicidality) more so than parental risk factors reliably foretell SA during adolescence, suggesting that risk identification and intervention efforts may be best served by focusing on youth from high-

risk families who themselves manifest symptoms suggestive of risk. Depressive symptoms as well as suicidality during childhood predict SA five years later, suggesting their importance in risk recognition and prevention efforts. Finally, future studies of SA among children of parents with AUD and other vulnerable families will be maximally informative if they contain additional contextual information about family environment and child abuse.

Acknowledgments

Abbreviations

SA	suicide attempt
AUD	alcohol use disorder
DUD	drug use disorder
COGA	Collaborative Study on the Genetics of Alcoholism

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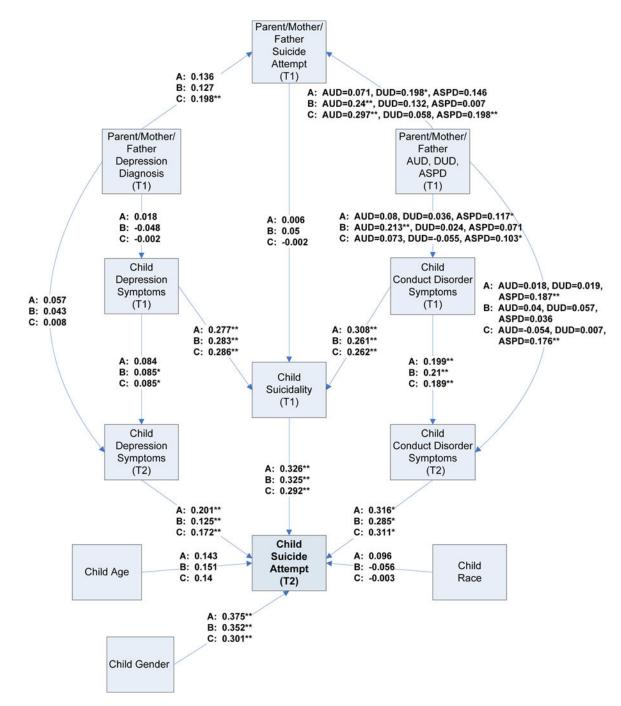
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Figure 1.

Models of parent- and child influences on adolescent suicide attempts

Notes:

>Results depict three structural equation models (SEM) using **father-child (A)**, **mother-child (B)**, and **parent-child (C)** data.

>Sample sizes: father-child model (290 children, 199 fathers), mother-child model (394 children, 269mothers), parent-child model (418 children, 300 parents)

>All numbers shown are standardized coefficients.

>T2=assessment at mean follow-up of approximately 5 years.

>*p < 0.05 / **p < 0.01

>AUD=alcohol use disorder in parent, mother, or father; DUD=drug use disorder in parent, mother, or father; ASPD=antisocial personality disorder in parent, mother, or father.

Table 1

Unadjusted Comparisons of Child Suicide Attempters and Non-Attempters at Time 2.

Child Predictors	Father-child sample			Mother-child sample			Parent-child sample		
	Suicide Attempters (T2) N (%)	Non-Attempters (T2) N (%)	p-value*	Suicide Attempters (T2) N (%)	Non-Attempters (T2) N (%)	p-value*	Suicide Attempters (T2) N (%)	Non-Attempters (T2) N (%)	p-value*
T1 Depression			0.0048			0.0168			0.0105
0, none	10 (58.8%)	353 (87.2%)		12 (66.7%)	329 (87.5%)		12 (63.2%)	347 (87.0%)	
1, symptoms	5 (29.4%)	43 (10.6%)		4 (22.2%)	39 (10.4%)		5 (26.3%)	43 (10.8%)	
2, disorder	2 (11.8%)	9 (2.2%)		2 (11.1%)	8 (2.1%)		2 (10.5%)	9 (2.3%)	
T2 Depression			0.0026			0.0217			0.0020
0, none	13 (76.5%)	260 (95.2%)		15 (83.3%)	356 (94.7%)		15 (79.0%)	378 (94.7%)	
1, symptoms	0 (0.0%)	7 (2.6%)		0 (0.0%)	12 (3.2%)		0 (0.0%)	13 (3.3%)	
2, disorder	4 (23.5%)	6 (2.2%)		3 (16.7%)	8 (2.1%)		4 (21.1%)	8 (2.0%)	
T1 Conduct Sxs			0.1264			0.2349			0.1305
0, none	9 (52.9%)	201 (73.6%)		10 (55.6%)	270 (71.8%)		10 (52.6%)	284 (71.2%)	
1, symptoms	7 (41.2%)	65 (23.8%)		7 (38.9%)	91 (24.2%)		8 (42.1%)	100 (25.1%)	
2, disorder	1 (5.9%)	7 (2.6%)		1 (5.6%)	15 (4.0%)		1 (5.3%)	15 (3.8%)	
T2 Conduct D/o			0.0211			0.0255			0.0098
0, none	3 (17.7%)	108 (39.6%)		4 (22.2%)	146 (38.8%)		4 (21.1%)	160 (40.1%)	
1, symptoms	6 (35.3%)	114 (41.8%)		6 (33.3%)	164 (43.6%)		6 (31.6%)	170 (42.6%)	
2, disorder	8 (47.1%)	51 (18.7%)		8 (44.4%)	66 (17.6%)		9 (47.4%)	69 (17.3%)	
T1 Suicidality – Continuous			<.0001			<.0001			<.0001
0, none	10 (58.8%)	232 (85.0%)		11 (61.1%)	317 (84.3%)		11 (57.9%)	336 (84.2%)	
1, ideation	4 (23.5%)	40 (14.7%)		4 (22.2%)	56 (14.9%)		5 (26.3%)	60 (15.0%)	
2, attempt	3 (17.7%)	1 (0.4%)		3 (16.7%)	3 (0.8%)		3 (15.8%)	3 (0.8%)	

Parent Predictors	Father-child			Mother-child			Parent(s)-child		
	Suicide Attempters (T2) N (%)	Non-Attempters (T2) N (%)	p-value*	Suicide Attempters (T2) N (%)	Non-Attempters (T2) N (%)	p-value*	Suicide Attempters (T2) N (%)	Non-Attempters (T2) N (%)	p-value*
T1 Suicide Attempt			0.1258			0.2548			0.0889
No	9 (75.0%)	178 (89.5%)		9 (75.0%)	233 (86.6%)		8 (61.5%)	232 (80.8%)	

Parent Predictors	Father-child			Mother-child			Parent(s)-child		
	Suicide Attempters (T2)	Non-Attempters (T2)	p-value*	Suicide Attempters (T2)	Non-Attempters (T2)	p-value*	Suicide Attempters (T2)	Non-Attempters (T2)	p-value*
	N (%)	N (%)		N (%)	N (%)		N (%)	N (%)	
Yes	3 (25.0%)	21 (10.5%)		3 (25.0%)	36 (13.4%)		5 (38.5%)	55 (19.2%)	
T1 Depressive D/o			0.5478			0.8021			0.6125
No	11 (91.7%)	170 (85.4%)		9 (75.0%)	210 (78.1%)		10 (76.9%)	202 (70.1%)	
Yes	1 (8.3%)	29 (14.6%)		3 (25.0%)	59 (21.9%)		3 (23.1%)	85 (29.6%)	
T1 Alcohol Use D/o			0.5797			0.5024			0.8254
No	2 (16.7%)	47 (23.6%)		8 (66.7%)	153 (56.9%)		2 (15.4%)	51 (17.8%)	
Yes	10 (83.3%)	152 (76.4%)		4 (33.3%)	116 (43.1%)		11 (84.6%)	236 (82.2%)	
T1 Drug Use D/o			0.7213			0.0395			0.2395
No	6 (50.0%)	89 (44.7%)		11 (91.7%)	168 (62.5%)		7 (53.9%)	108 (37.6%)	
Yes	6 (50.0%)	110 (55.3%)		1 (8.3%)	101 (37.6%)		6 (46.2%)	179 (62.4%)	
T1 Antisocial PD			0.1735			0.7805			0.2381
No	7 (58.3%)	151 (75.9%)		11 (91.7%)	252 (93.7%)		8 (61.5%)	218 (76.0%)	
Yes	5 (41.7%)	48 (24.1%)		1 (8.3%)	17 (6.3%)		5 (38.5%)	69 (24.0%)	

Covariates (child variables)	Father-child			Mother-child			Parent(s)-child		
	Suicide Attempters	Non-Attempters	p-value*	Suicide Attempters	Non-Attempters	p-value*	Suicide Attempters	Non-Attempters	p-value*
	N (%)	N (%)		N (%)	N (%)		N (%)	N (%)	
T1 Age, M +/− SD	9.9 +/−2.1	9.3 +/−1.8	0.1648	9.9 +/−2.1	9.3 +/−1.8	0.1599	9.9 +/−2.0	9.4 +/−1.8	0.1567
Sex			0.0052			0.0063			0.0166
Female	14 (82.4%)	128 (46.9%)		15 (79.0%)	184 (48.9%)		15 (79.0%)	194 (48.6%)	
Male	3 (17.7%)	145 (53.1%)		3 (16.7%)	192 (51.1%)		4 (21.1%)	205 (51.4%)	
Race-ethnicity			0.3834			0.8004			1.0000
White non-Hispanic	11 (64.7%)	207 (75.8%)		13 (72.2%)	254 (67.7%)		13 (68.4%)	268 (67.3%)	
Non-White	6 (35.3%)	66 (24.2%)		5 (27.8%)	121 (32.3%)		6 (31.6%)	130 (32.7%)	

Notes.

Sample sizes: father-child sample includes 190 fathers and 299 children; mother-child sample includes 269 mothers and 394 children; parent-child sample includes 300 parent(s) and 418 children

T2 suicide attempt (outcome): 17 children of 12 fathers had a T2 suicide attempt (father-child sample); 18 children of 12 mothers had a T2 attempt (mother-child sample); 19 children of 13 parent(s) had a T2 attempt (parent-child sample)

T1 = time 1, T2 = time 2, sxs = symptoms, hx = history

*
P-values based on comparisons using Fisher's Exact Test

**
One parent allowed a missing value