Published in final edited form as:
Drug Alcohol Depend. 2014 May 1; 138: 130–136. doi:10.1016/j.drugalcdep.2014.02.020.

Risks for Early Substance Involvement Associated with Parental Alcoholism and Parental Separation in an Adolescent Female Cohort*

Mary Waldron[1,2,†], **Ellen L. Vaughan**[1], **Kathleen K. Bucholz**[2], **Michael T. Lynskey**[3], **Carolyn E. Sartor**[2,4], **Alexis E. Duncan**[2,5], **Pamela A.F. Madden**[2], and **Andrew C. Heath**[2]

[1]School of Education, Indiana University, Bloomington, IN, USA

[2]Midwest Alcoholism Research Center, Department of Psychiatry, Washington University School of Medicine, St. Louis, MO, USA

[3]Addictions Department, Institute of Psychiatry, King's College, London, UK

[4]Department of Psychiatry, Yale University School of Medicine, New Haven, CT , USA

[5]George Warren Brown School of Social Work, Washington University, St. Louis, MO, USA

Abstract

Background—We examined timing of substance involvement as a joint function of parental history of alcoholism and parental separation during childhood.

Method—Data were drawn from a large cohort of female like-sex twins [*n* = 613 African Ancestry (AA), *n* = 3550 European or other Ancestry (EA)]. Cox proportional hazards regression was conducted predicting age at first use of alcohol, first alcohol intoxication, first use and regular use of cigarettes, and first use of cannabis and other illicit drugs from dummy variables coding for parental alcoholism and parental separation. Propensity score analysis was also conducted comparing intact and separated families by predicted probability of parental separation.

Results—In EA families, increased risk of substance involvement was found in both alcoholic and separated families, particularly through ages 10 or 14 years, with risk to offspring from alcoholic separated families further increased. In AA families, associations with parental alcoholism and parental separation were weak and with few exceptions statistically nonsignificant. While propensity score findings confirmed unique risks observed in EA families, intact and separated AA families were poorly matched on risk-factors presumed to predate parental

*Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:...

†Please address correspondence to Mary Waldron, Department of Counseling and Educational Psychology, School of Education, Indiana University, 201 N. Rose Avenue, Bloomington, IN 47405. Phone: 812-856-8334. Fax: 812-856-8333. mwaldron@indiana.edu..

separation, especially parental alcoholism, requiring cautious interpretation of AA survival-analytic findings.

Conclusion—For offspring of European ancestry, parental separation predicts early substance involvement that is not explained by parental alcoholism nor associated family background characteristics. Additional research is needed to better characterize risks associated with parental separation in African American families.

Keywords

adolescent substance use; parental separation or divorce; parental alcoholism

1. INTRODUCTION

Substance use during early adolescence remains a considerable public health concern. According to recent data, 33%, 19% and 16% of U.S. 8th graders, ages 13-14 on average, report lifetime use of alcohol, cigarettes, and cannabis, respectively (Johnston et al., 2012). In addition to immediate risks of harm, such as unintentional accidents, sexual risk-taking and other victimization (Hingson and Winter, 2003), early use has been linked to both initiation and escalation in use of harder substances (Fergusson et al., 2006; Kandel et al., 2006). Given that age at first use is strongly predictive of alcohol and other drug use disorders (Anthony and Petronis, 1995; Grant and Dawson, 1997), including severity and duration of disorder (Hingson et al., 2006), identifying risk-factors for early initiation is essential for targeted substance abuse prevention.

Parental separation or divorce during childhood and parental history of alcoholism are two among a host of risks-factors examined. Compared to children from intact families, children whose parents separate report more frequent use of alcohol and other drugs during adolescence (Doherty and Needle, 1991; Donovan and Molina, 2011; Hoffman and Su, 1995; Short, 1998), with higher rates of problem use also observed (Fergusson et al., 1994; Hoffman and Johnson, 1998). Compared to children of nonalcoholic parents, children of alcoholics (COAs) initiate alcohol and other drug use at younger ages (Chassin et al., 1991; Wong et al., 2006) and likewise report more frequent use of a range of substance classes (Chassin et al., 1996; Sher et al., 1991). COAS also report higher rates of problem use, particularly problem drinking (Lieb et al., 2002; Russell, 1990; Schuckit and Smith, 1996).

Currently, we know little regarding the separate effects of parental separation versus parental alcoholism on offspring substance involvement. Two studies examined problem drinking in adult offspring as a function of parental alcohol problems and family structure, and both documented unique risk associated with parental separation (Dube et al., 2002; Thompson et al., 2008). To our knowledge, there is a single study of parental separation and *timing* of substance use controlling for parental alcoholism. In a predominantly Caucasian Australian sample of twin parents and their children, Waldron and colleagues (2014) examined associations between parental separation and onset of drinking, drinking to intoxication, smoking, regular smoking, and cannabis use. Employing a Children-of-Twins design to control for genetic and environmental risks from parental alcohol and cannabis

dependence, parental separation predicted earlier initiation across substance class, with pronounced effects observed during very early adolescence.

The goal of this study was to conduct a joint analysis of parental alcoholism and parental separation on offspring early alcohol, tobacco, and illicit drug involvement in a population-representative U.S. sample of European and African ancestry female twins. Using a survival-analytic framework, we compared offspring from nonalcoholic intact families to offspring from (i) alcoholic separated, (ii) alcoholic intact, and (iii) nonalcoholic separated families. We also employed propensity score methods to infer, within a counterfactual framework, whether observed risks are unique to parental separation or due to unmeasured confounders, including parental alcoholism. The importance of considering counterfactuals has long been recognized (e.g., Rosenbaum and Rubin, 1983), but rarely implemented in addictions research (Heath et al., in press). By successfully matching offspring from separated and intact families across a range of predicted probabilities, based on predictors such as parental alcoholism, our confidence in the specificity of risks associated with parental separation is greatly increased; to the extent that we are unable to match across the spectrum of risk of separation, our confidence in such effects is undermined.

2. Methods

2.1. Participants

Data were drawn from the Missouri Adolescent Female Twin Study (Heath et al., 1999; 2002), a prospective study targeting the total cohort of female like-sex twin pairs born in Missouri to Missouri-resident parents, identified from birth records, for the period July 1, 1975-June 30, 1985 [*N*=370 African American (AA), 1999 European or other Ancestry (EA) pairs, the latter total including 26 of Asian/Pacific Islander or other ancestry]. A cohort-sequential sampling design was used, with initial cohorts of 13, 15, 17, and 19 year-old twins and their families recruited during the first two years of data-collection and continued recruitment of 13 year-olds in years three through four. In addition to baseline telephone diagnostic interviews conducted with parents, all available twin pairs were targeted for three waves of telephone interviews (Waves 1, 4 and 5, at median ages 15, 22 and 24, respectively). Subsamples of twins completed a brief one-year follow-up (Wave 2) and/or a three-year retest interview (Wave 3). For each wave, participants gave verbal consent (or assent if minors) following procedures approved by the institutional review board at Washington University. A summary of participation rates is provided elsewhere (Waldron et al., 2013).

Survival analyses are based on parent interviews and Waves 1, 3, 4 and 5 twin interviews. Twins were selected if they had data on lifetime substance (alcohol, tobacco or illicit drug) use and both parental alcoholism and parental separation, resulting in a sample of 4163 individual twins (613 AA, 3550 EA) from 2139 families (320 AA, 1819 EA). At last completed assessment, twins ranged in age from 12 years (Wave 1) to 31 (Wave 5), with AA twins approximately one year older on average [AA *M(SD)*=17.91(3.79)] than EA twins [*M(SD)*=16.71(3.30), t_1=8.10, p=0.0001]. Additional sample characteristics are provided in Supplementary Table S1[1], separately by race/ethnicity and presence versus absence of parental alcoholism and parental separation.

2.2. Measures

Measures derive largely from the Semi-Structured Assessment of the Genetics of Alcoholism (SSAGA; Bucholz et al., 1994; Hesselbrock et al., 1999), a semi-structured interview developed for the Collaborative Study on the Genetics of Alcoholism (Begleiter et al., 1995). The SSAGA has well-documented validity (Hesselbrock et al., 1999), with excellent retest and inter-rater reliability (Bucholz et al., 1994, 1995). Parents completed a telephone adaptation of the SSAGA-II—the DSM-IV update to the DSM-IIIR-based SSAGA. Twins completed either the child or adolescent version of the SSAGA-II, also adapted for telephone administration.

2.2.1. Substance involvement—Onset of drinking, drinking to intoxication, smoking and regular smoking, and use of cannabis and other illicit drugs (e.g., cocaine, heroin, hallucinogens) were assessed in each of Waves 1, 3 and 4 twin interviews. At Wave 5, new (past two-year) onsets of cannabis use were also assessed. Regular smoking was defined as having smoked 100 or more cigarettes lifetime, or between 21-99 cigarettes at least (i) 3-4 days per week for 3 weeks (Waves 1 and 3) or (ii) 1-2 days per week for 2 months (Wave 4). For each substance use variable, youngest reported age of onset was coded; however, youngest and first reports were highly correlated (r>0.90, p<0.05).

2.2.2. Parental separation—Parental separation prior to twins' age 18 was coded from parent and Waves 1, 3, 4 and 5 twin interviews. Consistent with previous work (Waldron et al., 2013), parental separation was defined as change in marital and/or cohabitation status of biological parents for reasons of relationship dissolution. Twins' age at parental separation was coded from year parents' marriage ended or, if missing, age last lived with both parents. Age at separation in families where separation occurred prior to twins' birth was coded as a fraction of a year.

2.2.3. Parental alcoholism—History of parental alcoholism was coded from parent self-report of alcohol dependence (AD), parent ratings of coparent dependence symptoms, and twin ratings of each parent as a problem and excessive drinker. Parent interviews included self-report assessment of lifetime history of AD, with AD symptoms experienced by the twins' biological coparent assessed using an adaptation of the Family History Assessment Module (Rice et al., 1995). Temporal clustering of coparent symptoms was not assessed, thus a probable dependence diagnosis without requiring 12-month clustering was coded for coparent AD. Twin ratings of each parent were drawn from Wave 4, when all twins were aged 18 or older. Twin interviews did not ask detailed questions about parental dependence symptoms; instead, twins were asked whether "drinking ever caused your biological (mother/father) to have problems with health, family, job or police, or other problems," an item that originated in the Family History Research Diagnostic Criteria assessment (Andreasen et al., 1977), and whether they ever felt that their biological parent was an "excessive drinker." Endorsement of both problem and excessive drinking items was required to code a parent positive by twin report. Consistent with earlier analyses (Waldron

[1]Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:...

et al., 2012, 2013), a parent was coded positive based on self-report or any family history rating.

2.2.4. Control variables—Correlated risks that might separately increase the likelihood of offspring substance involvement were modeled as covariates in survival analyses. As summarized in Table S1[2], covariates included maternal age at twins' birth, maternal educational attainment, history in either parent of smoking and comorbid psychopathology (parent self-report of DSM-IV conduct disorder [CD] and major depressive disorder [MDD]), offspring psychopathology (parent report of DSM-IV inattention, hyperactivity and oppositional defiant disorder [ODD], and offspring self-report of CD, social anxiety disorder [SAD] and MDD) including a non-diagnostic measure of suicidality (offspring self-report of prior suicide ideation, plan or attempt), and childhood risk-factors (offspring self-report of physical abuse and sexual abuse prior to age 18).

2.2.5. Zygosity—Zygosity was coded from parent and twin responses to standard questions regarding twin similarity and the degree to which twins were confused by others (Nichols and Bilbro, 1966).

2.3. Analytic Strategy

The present study uses data from individual twins to examine phenotypic associations between timing of substance involvement and both parental separation and parental alcoholism. Analyses were performed in STATA version 12 (StataCorp, 2011), with the Huber-White robust variance estimator used to compute standard errors and confidence intervals adjusted for non-independence (i.e., the correlated nature) of twin-family data. Comparisons of EA and AA families were conducted as part of preliminary analyses only. After confirming well-documented racial/ethnic differences, for example, in patterns of substance use during adolescence (Johnston et al., 2012) and family structure (Raley and Bumpass, 2003; Bramlett and Mosher, 2002), survival and propensity score analyses were conducted separately for EA and AA families. Rather than conducting pooled analyses modeling interactions with race/ethnicity, we were compelled to stratify because of the potential (and subsequently documented) problem of *covariate inbalance*, i.e., the existence of combinations of covariate and outcome values present in EA families but absent in AA families—under such conditions, a pooled analysis can lead to biased estimates (Rosenbaum and Rubin, 1983; Rubin, 2006).

Survival analysis was used to assess likelihood as well as timing of substance involvement, separately for each substance use variable (alcohol use, alcohol intoxication, cigarette use, regular smoking, cannabis use, and other illicit drug use). In preliminary descriptive analyses, cumulative failure curves were estimated using the Kaplan-Meier survivor function (Kaplan and Meier, 1958), with log-rank tests to identify significant differences in equality of survivor functions by race/ethnicity. While monozygotic (MZ) twinning occurs at random, dizygotic (DZ) twining has been linked to both maternal age and socioeconomic status (Bulmer, 1970); thus, to identify limitations to the generalizability of twin data, log-

[2]Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:...

ranks tests were also conducted for zygosity. Cox proportional hazards regression was used to examine risk of substance involvement without and with adjustment for control variables. The Efron approximation (Efron, 1977) was applied for survival ties. Because age of onset of AD is available from parent self-report only, parental alcoholism was modeled as a time-invariant predictor. Parental separation was modeled as time-varying to ensure onset before or at the same time as initiation of substance involvement. This was achieved using *person-year* data, with each line of data representing a single year of life for each twin. Intact families were right-censored at twins' age at last interview if younger than age 18, and in the case of parental death during childhood, right-censored at twins' age when their parent(s) died. Dummy codes were computed from person-year data to distinguish alcoholic separated, alcoholic intact and nonalcoholic separated families, with nonalcoholic intact families comprising the reference group. Control variables with available ages of onset (CD, SAD, MDD, suicidality, and physical and sexual abuse) were examined as time-varying predictors in adjusted models. To examine potential violation of the proportional hazards assumption, such as might be the case if the effects of parental alcoholism and/or parental separation on substance involvement differ across age periods, the Grambsch and Therneau test of Schoenfeld residuals (Grambsch and Therneau 1994) was employed, with age-interactions modeled to correct observed violations (Cleves et al., 2004).

Next, propensity score analysis (PSA) was conducted to compare intact and separated families by predicted probability of separation or *propensity* to separate. (Although more elaborate matching methods exist, we know of none formulated for use with clustered observations; see Rosenbaum, 2010). PSA was limited to data available by mother report to reduce bias associated with paternal reports (where missingness is much higher for separated families; Waldron et al., 2013), resulting in a reduced sample of 1645 twin-families (201 AA, 1444 EA); comparisons of early substance use by PSA were further limited to 2038 individual twins (263 AA, 1775 EA) aged 15 and older. Predicted probabilities of parental separation were derived from logistic regression models predicting separation from variables presumed to predate relationship dissolution, including mother self-report alcohol dependence symptoms, mother report of paternal alcohol problems, maternal age at twins' birth, paternal educational attainment, maternal smoking, and two-way interactions between parental alcohol problems and (i) maternal age at twins' birth and (ii) paternal education. Although maternal education was examined as a covariate in Cox analyses, paternal education was more strongly predictive of separation (versus offspring substance involvement; data not shown), and thus for purposes of PSA, paternal rather than maternal educational attainment was modeled. Likewise, maternal history of smoking was modeled instead of either parent history of smoking. From resulting propensity scores, a 5-level categorical variable was computed with each level containing approximately 20% of the distribution, followed by within-quintile comparisons of family background risks and, conditional on successful matching, offspring substance involvement. In this way it was possible to examine whether effects of parental separation are consistent across the distribution of parental separation risk–in families with low prior probability of separation, in families at intermediate risk, and in families with high prior probability of separation–or whether there are discontinuities not captured by Cox analyses.

3. RESULTS

Prevalence of substance involvement and average ages of onset are shown in Table 1, separately by race/ethnicity. For all substance use variables except cannabis, AA twins were at reduced likelihood of initiation, compared to EA twins (log-rank test $p<0.05$). By twins' age 18, 75% of AA parents and 38% of EA parents had separated, with AA twins experiencing earlier time to parental separation relative to EA twins ($X^2_1=439.06$, $p<0.0001$). Either or both parents were more likely to be coded positive for alcoholism in AA compared to EA families (42% versus 35%, $X^2_1=0.42$, $p<0.01$). Log-rank tests of differences in substance involvement by zygosity were significant in EA families only, such that DZ twins were at increased likelihood of initiation across substance class, compared to MZ twins ($p<0.05$). While zygosity was unrelated to parental separation in either AA or EA families, more EA parents of DZ twins were coded positive for parental alcoholism than EA parents of MZ twins ($X^2_1=4.38$, $p<0.05$). Given differences by zygosity in both timing of substance involvement and parental alcoholism, zygosity was included as an additional covariate in adjusted Cox models.

3.1.Cox Analyses

Hazard ratios from unadjusted and adjusted Cox regression models predicting timing of substance involvement are shown in Table 2 for EA twins. EA twins from alcoholic separated families were at highest risk of early alcohol, tobacco, cannabis and other illicit substance involvement, compared to twins from nonalcoholic intact families. Risks related to misuse were especially pronounced, with twins from alcoholic separated families at 33 times increased likelihood of first alcohol intoxication through age 10, and 3.52 times increased likelihood over ages 11-14. Twins from alcoholic separated families were at nearly 11 times increased likelihood of regular smoking through age 10, and 4.57 times increased likelihood over ages 11-14. Risks associated with intact alcoholic families were moderate, but likewise elevated across substance class, with greater risk for onset by ages 12 or 14. Effects of parental separation absent of parental alcoholism were also observed, particularly for smoking and use of cannabis or other illicit drugs. Twins from nonalcoholic families where parents separated were at nearly three times increased likelihood of smoking before age 11, and nearly two times increased likelihood over ages 11-14. Through age 14, parental separation alone predicted over two times increased likelihood of regular smoking and cannabis use. Twins from nonalcoholic families where parents separated were 3.73 times increased likelihood of other illicit drug use before age 15, and nearly two times increased likelihood from age 15 onwards. Similar but somewhat attenuated effects were observed in covariate-adjusted models.

Hazard ratios from unadjusted and adjusted Cox analyses are shown in Table 3 for AA twins. Effects of parental alcoholism and/or parental separation were nonsignificant, with one exception: AA twins from alcoholic separated families were at 1.88 times increased likelihood of smoking onset through age 14, compared to twins from nonalcoholic intact families. In adjusted models, significant protective effects were observed. Parental alcoholism was associated with 75% decreased likelihood of regular smoking from age 15 onwards if parents were separated, and across adolescence for twins whose parents remained

together. In nonalcoholic AA families where parents separated, AA twins were at 92% decreased likelihood of regular smoking from age 18 onwards. Parental alcoholism predicted 96% decreased likelihood of non-cannabis illicit drug use from age 15 onwards if parents were separated, and 97% decreased likelihood across risk period(s) for twins whose parents remained together; in nonalcoholic families where parents separated, twins were at 93% decreased likelihood of other illicit drug use through age 14.

3.2. Propensity Score Analyses

Among EA families, prevalence of parental separation ranged from 10% (0-20%ile of propensity score distribution) to 79% (>80%ile of propensity score distribution). Within-quintile comparisons of EA family background characteristics are presented in Table 4, with multivariate results provided in Table S2[3]. In summary, parental alcoholism (maternal or paternal) is nearly absent in the lowest quintile of predicted probability of parental separation, but a dominant factor in the highest quintile. A similar pattern is observed for maternal smoking and both maternal age at twins' birth and paternal education, where very young mothers and fathers with less than high school education were over-represented among higher risk quintiles. Given excellent matching on family background, within-quintile comparisons of twin substance involvement by parental separation were conducted. As shown in Table 5, parental separation in EA families continued to predict riskier twin outcomes across the risk spectrum. In most cases, differences within strata reached statistical significance with the single odd exception of alcohol use in 61-80th percentile families, where rates of early alcohol use were unusually low in separated families.

Among AA families, prevalence of parental separation ranged from 38% (0-20%ile of propensity score distribution) to 100% (>80%ile of propensity score distribution). Within-quintile comparisons of AA family background characteristics are presented in Table 6, with results of a multivariate model predicting parental separation provided in Table S3[4]. As shown, imperfect matching is especially evident for parental (maternal or paternal) alcoholism, where a high proportion of alcoholic families fell into the highest quintile of separation risk, where there was no counterfactual, i.e., no stably intact families with an alcoholic parent. Similarly, for maternal age at twins' birth, there were no stably cohabiting or married mothers who gave birth as teens. Consequently, within-quintile comparisons of twin substance involvement by parental separation were not conducted.

4. DISCUSSION

Despite well-documented associations between early substance involvement and both parental alcoholism and parental separation, risks to offspring associated with parental separation have received limited attention in COA research. Using data drawn from a population-representative sample of European and African ancestry female twins, we examined onset of alcohol, tobacco and illicit drug involvement as a joint function of parental separation during childhood and parental history of alcohol dependence, with survival and propensity score analyses conducted separately by race/ethnicity.

[3]Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:...
[4]Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi:...

Among families of European ancestry, effects of parental separation and parental alcoholism on timing of substance involvement were substantial, even in Cox models adjusting for family background, offspring psychopathology, and physical and sexual abuse during childhood. Compared to twins from nonalcoholic intact families, twins from families where a parent was alcoholic or parents separated were more likely to initiate substance involvement during very early adolescence. Risks to twins from alcoholic separated families were further increased, suggestive of a dose-response relationship. Results from propensity score analyses largely confirm survival-analytic findings for European ancestry twins, increasing our confidence in the specificity of risks associated with parental separation. Effects of parental separation on offspring substance involvement were observed across much of the risk spectrum and, despite elevated rates of parental alcoholism, twins from intact families at high predicted probability of separation demonstrated reduced risk, particularly for alcohol and cannabis involvement.

In contrast, we observed little risk from either parental separation or parental alcoholism on timing of substance involvement for African American offspring. In adjusted Cox models, somewhat surprising protective effects were found. However, results from propensity score analyses suggest very cautious interpretation of survival-analytic findings, regardless of covariate control. Intact and separated African American families were poorly matched on risks upstream of parental separation, including parental alcoholism, and no African American families at highest risk of separation remained intact; thus, we have little confidence in comparisons of African American offspring outcomes examined as a function of parental separation. Such findings also call into question use of statistical adjustment for race/ethnicity in research comparing intact and separated families. Because inferences can be made only about a very restricted range of the propensity score distribution for African Americans, if data from European and African ancestry families were pooled and race/ethnicity adjusted statistically, we would in effect be making predictions for African Americans in regions with zero data-points, i.e., intact African American families where parents are at high probability of separation.

Although our study is one of few to examine the separate effects of parental separation versus parental alcoholism on offspring substance involvement, there are a number of limitations. First, parental alcoholism was modeled without regard to onset or remission. Our results likely underestimate risks for offspring exposed to chronic parental alcoholism to the extent that some parents no longer met criteria during childrearing years. We also did not examine potential mediators of risk from parental separation or parental alcoholism, such as compromised parenting behavior (e.g., lax or inconsistent monitoring) and presence of a stepparent, which will be an important focus of future work. Follow-up of twins as they age into periods of highest risk of substance dependence is also planned. For African Americans, in addition to reduced interpretability of survival-analytic findings, we have limited statistical power because of a much smaller sample of African relative to European ancestry twins, which may have (i) obscured potentially important age interactions in survival models and (ii) contributed to inadequate numbers of intact African American families across the range of risk propensity for parental separation. Lastly, given significant differences by zygosity observed in the likelihood of onset of substance involvement, most likely reflecting the importance of peer influences and the wider social networks of fraternal

compared to identical pairs, extension to non-twin samples is critical, including comparison of twins to singletons where data on both are available. Given potential differences by offspring sex, replication of findings in male offspring is also important.

Tailored substance abuse prevention is dependent upon correctly identifying important risk and protective mechanisms. Findings from survival and propensity score analyses of European ancestry families highlight the importance of parental separation for very early substance involvement beyond parental alcoholism. However, risk mechanisms associated with parental separation identified in largely White European ancestry families cannot be assumed to generalize to African American families. To better characterize risk to African American offspring, additional research is needed with increased samples sizes for adequately powered analyses, preferably with matching of families for both parental separation and parental alcoholism.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

REFERENCES

Andreasen NC, Endicott J, Spitzer RL, Winokur G. Family history method using diagnostic criteria: reliability and validity. Arch. Gen. Psychiatry. 1977; 34:1229–1235. [PubMed: 911222]

Anthony JC, Petronis KR. Early-onset drug use and risk of later drug problems. Drug Alcohol Depend. 1995; 40:9–15. [PubMed: 8746919]

Begleiter H, Reich T, Hesselbrock V, Porjesz B, Li T-K, Schuckit M, Edenberg HJ, Rice J. The collaborative study on the genetics of alcoholism. Alcohol Health Res. World. 1995; 19:228–236.

Bucholz KK, Cadoret R, Cloninger CR, Dinwiddie SH, Hesselbrock VM, Nurnberger JI, Reich T, Schmidt I, Schuckit MA. A new semi-structured psychiatric interview for use in genetic linkage studies: A report on the reliability of the SSAGA. J. Stud. Alcohol. 1994; 55:149–158. [PubMed: 8189735]

Bramlett MD, Mosher WD. Cohabitation, marriage, divorce, and remarriage in the United States. Vital Health Stat. Series. 2002; 23:1–93.

Bulmer, MG. The biology of Twinning in Man. Clarendon Press; Oxford: 1970.

Chassin L, Curran PJ, Hussong AM, Colder C. The relation of parent alcoholism to adolescent substance use: a longitudinal follow-up study. J. Abnorm. Psychol. 1996; 105:70–80. [PubMed: 8666713]

Chassin L, Rogosch F, Barrera M. Substance use and symptomatology among adolescent children of alcoholics. J. Abnorm. Psychol. 1991; 100:449–463. [PubMed: 1757658]

Cleves, M.; Gould, WW.; Gutierrez, RG. Introduction to Survival Data Analysis with Stata, Revised Edition. Stata Press; College Station, TX: 2004.

Doherty WJ, Needle RH. Psychological adjustment and substance use among adolescents before and after a parental divorce. Child Dev. 1991; 62:328–337. [PubMed: 2055125]

Donovan JE, Molina BS. Childhood risk factors for early-onset drinking. J. Stud. Alcohol Drugs. 2011; 72:741–51. [PubMed: 21906502]

Dube SR, Anda RF, Felitti VJ, Edwards VJ, Croft JB. Adverse childhood experiences and personal alcohol abuse as an adult. Addict. Behav. 2002; 27:713–725. [PubMed: 12201379]

Efron B. The efficiency of Cox's likelihood function for censored data. J. Am. Stat. Assoc. 1977; 72:557–565.

Fergusson DM, Horwood LJ, Lynskey MT. Parental separation, adolescent psychopathology, and problem behaviors. J. Am. Acad. Child Adolesc. Psychiatry. 1994; 33:1122–1131. [PubMed: 7982863]

Fergusson DM, Boden JM, Horwood LJ. Cannabis use and other illicit drug use: testing the cannabis gateway hypothesis. Addiction. 2006; 101:556–569. [PubMed: 16548935]

Grant BF. Prevalence and correlates of alcohol use and DSM-IV alcohol dependence in the United States: results of the National Longitudinal Alcohol Epidemiologic Survey. J. Stud. Alcohol. 1997; 58:464–473. [PubMed: 9273910]

Grant BF, Dawson DA. Age of onset of alcohol use and its association with DSM–IV alcohol abuse and dependence: results from the National Longitudinal Alcohol Epidemiological Survey. J. Subst. Abuse. 1997; 9:103–110. [PubMed: 9494942]

Grambsch PM, Therneau TM. Proportional hazards tests and diagnostics based on weighted residuals. Biometrika. 1994; 81:515–526.

Hasin DS, Stinson FS, Ogburn E, Grant BF. Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United States: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Arch. Gen. Psychiatry. 2007; 64:830–842. [PubMed: 17606817]

Heath AC, Madden PAF, Bucholz KK. Ascertainment of a twin sample by computerized record matching, with assessment of possible sampling biases. Behav. Genet. 1999; 29:209–219.

Heath AC, Howells W, Bucholz KK, Glowinski AL, Nelson EC, Madden PAF. US female adolescent twin cohort for alcohol studies: assessment of sample representativeness using birth record data. Twin Res. Hum. Genet. 2002; 5:107–112.

Heath AC, Waldron M, Martin NG, Nelson EC, Bucholz KK, Madden PAF. Human mate selection and addiction: a conceptual critique. Behav. Genet. in press.

Hesselbrock M, Easton C, Bucholz KK, Schuckit M, Hesselbrock V. A validity study of the SSAGA: a comparison with the SCAN. Addiction. 1999; 94:1361–1370. [PubMed: 10615721]

Hingson RW, Heeren T, Winter MR. Age at drinking onset and alcohol dependence: age at onset, duration, and severity. Arch. Pediatr. Adolesc. Med. 2006; 160:739–746. [PubMed: 16818840]

Hingson R, Winter M. Epidemiology and consequences of drinking and driving. Alcohol Res. Health. 2003; 27:63–78. [PubMed: 15301401]

Hoffman JP, Su SS. Parental substance use disorder, mediating variables and adolescent drug use: a non-recursive model. Addiction. 1998; 93:1352–1364.

Hoffmann JP, Johnson RA. A national portrait of family structure and adolescent drug use. J. Marriage Fam. 1998; 60:633–645.

Lieb R, Merikangas KR, Hofler M, Pfister H, Isensee B, Wittchen HU. Parental alcohol use disorders and alcohol use and disorders in offspring: a community study. Psychol. Med. 2002; 32:63–78. [PubMed: 11883731]

Johnston, LD.; O'Malley, PM.; Bachman, JG.; Schulenberg, JE. Institute for Social Research. The University of Michigan; Ann Arbor: 2012. Monitoring the Future National Results on Adolescent Drug Use: Overview of Key Findings, 2011..

Kandel DB, Yamaguchi K, Klein LC. Testing the gateway hypothesis. Addiction. 2006; 101:470–472. [PubMed: 16548924]

Kaplan EL, Meier P. Nonparametric estimation from incomplete observations. J. Am. Stat. Assoc. 1958; 53:457–481.

Nichols RC, Bilbro WC. The diagnosis of twin zygosity. Acta Genet. Stat. Med. 1966; 16:265–275. [PubMed: 4959152]

Raley RK, Bumpass LL. The topography of the divorce plateau: levels and trends in union stability in the United States after 1980. Demogr. Res. 2003; 8:245–259.

Rice JP, Reich T, Bucholz KK, Neuman RJ, Fishman R, Rochberg N, Hesselbrock VM, Nurnberger JI Jr. Schuckit MA, Begleiter H. Comparison of direct interview and family history diagnoses of alcohol dependence. Alcohol. Clin. Exp. Res. 1995; 19:1018–23. [PubMed: 7485811]

Rosenbaum PR, Rubin DB. The central role of the propensity score in observational studies for causal effects. Biometrika. 1983; 70:41–55.

Rosenbaum, PR. Design of Observational Studies. Springer; New York, NY: 2010.

Rubin, D. Matched sampling for causal effects. Cambridge University Press; Cambridge, UK: 2006.

Russell, M. Prevalence of alcoholism among children of alcoholics.. In: Windle, M.; Searles, JS., editors. Children of Alcoholics: Critical Perspectives. Guilford Press; New York: 1990. p. 9-38.

Schuckit MA, Smith TL. An 8-year follow-up of 450 sons of alcoholic and control subjects. Arch. Gen. Psychiatry. 1996; 53:202–210. [PubMed: 8611056]

Sher KJ, Walitzer KS, Wood PK, Brent EE. Characteristics of children of alcoholics: putative risk factors, substance use and abuse, and psychopathology. J. Abnorm. Psychol. 1991; 100:427–448. [PubMed: 1757657]

Short J. Predictors of substance use and mental health of children of divorce: a prospective analysis. J. Divorce Remarriage. 1998; 29:147–166.

StataCorp. Stata Statistical Software: Release 12. StataCorp LP; College Station, TX: 2011.

Thompson R, Lizardi D, Keyes KM, Hasin DS. Childhood or adolescent parental divorce/separation, parental history of alcohol problems, and offspring lifetime alcohol dependence. Drug Alcohol Depend. 2008; 98:264–269. [PubMed: 18757141]

Wong MM, Nigg JT, Zucker RA, Puttler LI, Fitzgerald HE, Jester JM, Glass JM, Adams KA. Behavioral control and resiliency in the onset of alcohol and illicit drug use: a prospective study from preschool to adolescence. Child Dev. 2006; 77:1016–1033. [PubMed: 16942503]

Waldron M, Bucholz KK, Lynskey MT, Madden PAF, Heath AC. Alcoholism and timing of separation in parents: findings in a Midwestern birth cohort. J. Stud. Alcohol Drugs. 2013; 74:337–348. [PubMed: 23384382]

Waldron M, Grant JD, Bucholz KK, Lynskey MT, Slutske WS, Glowinski AL, Henders A, Statham DJ, Martin N, Heath AC. Parental separation and early substance involvement: results from children of alcoholic and cannabis-dependent twins. Drug Alcohol Depend. 2014; 134:78–84. [PubMed: 24120074]

Waldron M, Madden PAF, Nelseon EC, Knopik VS, Glowinski AL, Grant JG, Lynskey MT, Jacob T, Sher KJ, Bucholz KK, Heath AC. Interpretability of family history reports of alcoholism is general community samples: findings in a Midwestern U.S. twin birth cohort. Alcohol. Clin. Exp. Res. 2012; 36:1091–1098. [PubMed: 22235921]

Table 1

Prevalence and age of onset of substance involvement, by race/ethnicity.

	European Ancestry (*n* = 3550)	**African Ancestry (*n* = 613)**
Alcohol use, *n* (%)	3050 (86)	456 (75)
age of onset, *M* (*SD*)	15.15 (2.48)	16.48 (3.04)
Alcohol intoxication, *n* (%)	2432 (69)	231 (38)
age of onset, *M* (*SD*)	16.88 (2.37)	18.42 (2.86)
Cigarette use, *n* (%)	2589 (73)	381 (62)
age of onset, *M* (*SD*)	13.64 (3.22)	13.88 (3.68)
Regular smoking, *n* (%)	1395 (39)	102 (17)
age of onset, *M* (*SD*)	15.50 (2.62)	16.76 (2.93)
Cannabis use, *n* (%)	1806 (52)	330 (57)
age of onset, *M* (*SD*)	16.48 (2.59)	16.73 (2.72)
Other illicit drug use, *n* (%)	697 (20)	47 (8)
age of onset, *M* (*SD*)	17.16 (2.60)	18.34 (3.45)

Table 2

Hazard Ratios (and 95% Confidence Intervals) from Cox Regression models predicting timing of substance involvement from parental alcoholism and parental separation in European ancestry twins, unadjusted and adjusted for family background, offspring psychopathology, and childhood risk-factors.

	Alcoholic/Separated		Alcoholic/Intact		Nonalcoholic/Separated	
	Unadjusted (*n* = 779)	**Adjusted (*n* = 697)**	**Unadjusted (*n* = 481)**	**Adjusted (*n* = 446)**	**Unadjusted (*n* = 566)**	**Adjusted (*n* = 519)**
Alcohol use						
< 11	5.45 (3.02 – 9.81)	4.52 (2.41 – 8.48)	3.42 (1.82 -6.45)	3.14 (1.58 – 6.10)	⊤	⊤
11-14	2.53 (2.09 – 3.06)	2.16 (1.73 – 2.69)	1.52 (1.30 -1.79)	1.21 (1.04 – 1.49)	1.63 (1.28 – 2.08)	1.59 (1.22 – 2.08)
≥ 15	1.51 (1.27 – 1.80)	1.44 (1.19 – 1.75)	⊥	⊥	1.24 (1.02 – 1.50)	1.19 (0.97 – 1.46)
Alcohol intoxication						
< 11	33.09 (3.58 – 322.22)	15.39 (1.81 – 130.55)[a]				⊤
11-12	3.52 (2.69 – 4.60)	4.79 (2.59 – 8.97)[a]	⊤	⊤	⊤	1.99 (1.32 – 2.98)
13-14	⊥	2.71 (1.94 – 3.79)	1.44 (1.18 – 1.76)	1.25 (1.00 – 1.56)	1.59 (1.29 -1.96)	⊥
15-17	1.79 (1.46 – 2.18)	1.65 (1.30 – 2.08)	⊥	⊥	⊥	1.40 (1.08 – 1.82)
≥ 18	1.13 (0.80 – 1.50)	1.19 (0.82 – 1.72)			0.65 (0.45 – 0.97)	0.69 (0.45 – 1.07)
Cigarette use						
< 11	4.78 (3.57 – 6.41)	3.13 (2.25 – 4.35)	2.91 (2.13 – 4.00)	2.28 (1.65 – 3.17)	2.96 (2.05 – 4.29)	2.23 (1.49 – 3.23)
11-14	2.39 (1.99 – 2.86)	1.62 (1.35 – 1.94)	1.66 (1.40 – 1.96)	1.25 (1.04 – 1.50)	1.97 (1.61 – 2.40)	1.49 (1.23 – 1.80)
≥ 15	1.78 (1.40 – 2.27)	⊥	⊥	⊥	1.41 (1.07 – 1.84)	⊥
Regular smoking						
< 11	10.66 (4.48 – 25.37)	⊤	5.63 (2.16 – 14.67)	2.95 (1.40 – 6.24)	⊤	⊤
11-14	4.57 (3.37 – 6.19)	2.64 (1.94 – 3.58)	2.23 (1.52 – 3.29)[a]	⊤	2.40 (1.63 – 3.52)	1.57 (1.18 – 2.07)
15-17	2.31 (1.78 – 3.00)[a]	1.46 (1.10 – 1.95)	1.80 (1.31 – 2.47)[a]	1.35 (1.02 – 1.79)	1.54 (1.16 – 2.04)	0.80 (0.42 – 1.51)
≥ 18	1.70 (1.10 – 2.64)[a]	⊥	0.80 (0.41 – 1.59)	0.61 (0.29 – 1.27)	⊥	⊥
Cannabis use						
< 15	5.76 (4.25 – 8.00)	3.57 (2.46 – 5.17)	2.28 (1.49 – 3.48)[a]	1.60 (1.01 – 2.55)[a]	2.88 (1.89 – 4.40)	2.11 (1.36 – 3.28)
≥ 15	2.37 (1.94 – 2.89)	1.57 (1.22-2.02)	1.61 (1.26 – 2.05)[a]	1.20 (0.91 – 1.59)[a]	1.35 (1.05 – 1.73)	1.22 (0.93 – 1.60)
Other illicit drug use						
< 15	6.80 (3.22 – 14.37)	⊤	3.56 (1.49 – 8.52)[a]	⊤	3.73 (1.52 – 9.14)	2.35 (1.11 – 5.01)[a]

	Alcoholic/Separated		Alcoholic/Intact		Nonalcoholic/Separated	
	Unadjusted (*n* = 779)	**Adjusted (*n* = 697)**	**Unadjusted (*n* = 481)**	**Adjusted (*n* = 446)**	**Unadjusted (*n* = 566)**	**Adjusted (*n* = 519)**
15-17	2.98 (2.19 – 4.06)	2.29 (1.59 – 3.30)	2.00 (1.26 – 3.17)[a]	1.66 (1.04 – 2.65)	1.88 (1.30 – 2.72)	1.68 (1.11 – 2.55)[a]
≥ 18	⊥	⊥	0.83 (0.37 – 1.84)	0.70 (0.31 – 1.61)	⊥	⊥

Note. Reference group = nonalcoholic, intact families. Where brackets are shown, reported risk is equivalent across risk periods (age in years).

[a]Post-hoc test equating hazard ratios across risk periods did not show significant heterogeneity ($p > 0.05$), but the proportional hazards assumption was violated and thus an age interaction was modeled with separate hazard ratios reported.

Table 3

Hazard Ratios (and 95% Confidence Intervals) from Cox Regression models predicting timing of substance involvement from parental alcoholism and parental separation in African ancestry twins, unadjusted and adjusted for family background, offspring psychopathology, and childhood risk-factors.

	Alcoholic/Separated		Alcoholic/Intact		Nonalcoholic/Separated	
	Unadjusted (*n* = 208)	**Adjusted (*n* = 174)**	**Unadjusted (*n* = 47)**	**Adjusted (*n* = 42)**	**Unadjusted (*n* = 269)**	**Adjusted (*n* = 210)**
Alcohol use	1.03 (0.73 – 1.46)	0.78 (0.52 – 1.17)	1.06 (0.68 – 1.65)	1.04 (0.61 – 1.77)	0.94 (0.67 – 1.31)	0.94 (0.64 – 1.36)
Alcohol intoxication						
< 15	1.48 (0.88 – 2.50)	4.54 (0.37 – 54.93)[a]	0.99 (0.43 – 2.67)	8.13 (0.66 – 100.07)	0.93 (0.55 – 1.59)	7.90 (0.69 – 91.06)
≥ 15	⊥	0.76 (0.39 – 1.47)[a]	⊥	0.64 (0.23 – 1.80)[a]	⊥	0.62 (0.33 – 1.18)
Cigarettes use						
< 15	1.88 (1.26 – 2.81)	1.66 (0.89 – 3.09)	1.30 (0.80 – 2.12)	0.79 (0.44 – 1.42)	1.09 (0.76 – 1.55)	0.93 (0.61 – 1.41)
≥ 15	1.07 (0.66 – 1.75)	0.88 (0.52 – 1.47)	⊥	⊥	⊥	⊥
Regular smoking						
< 15	2.84 (0.87 – 9.26)	3.22 (0.30 – 34.34)	⊤	⊤	⊤	1.95 (0.18 – 21.42)[a]
15-17	1.01 (0.40 – 2.52)	0.25 (0.08 – 0.74)	0.52 (0.14 – 1.88)	0.25 (0.06 – 0.96)	1.09 (0.50 – 2.38)	0.40 (0.14 – 1.19)[a]
≥ 18	⊥	⊥	⊥	⊥	⊥	0.08 (0.02 – 0.37)
Cannabis use	1.36 (0.85 – 2.20)	0.91 (0.50 – 1.69)	1.32 (0.71 – 2.45)	1.07 (0.52 – 2.21)	1.50 (0.95 – 2.35)	1.24 (0.68 – 2.23)
Other illicit drug use						
< 15	0.69 (0.22 – 2.23)	0.48 (0.03 – 8.85)	0.71 (0.14 – 3.69)	0.03 (0.01 – 0.18)	0.73 (0.23 – 2.23)	0.07 (0.01 – 0.63)
≥ 15	⊥	0.04 (0.01 – 0.21)	⊥	⊥	⊥	1.07 (0.24 – 4.77)

Note. Reference group = nonalcoholic, intact families. Where brackets are shown, reported risk is equivalent across risk periods (age in years).

[a]Post-hoc test equating hazard ratios across risk periods did not show significant heterogeneity ($p > 0.05$), but the proportional hazards assumption was violated and thus an age interaction was modeled with separate hazard ratios reported.

Table 4

Family background characteristics of intact and separated European ancestry families, by quintile of predicted probability of parental separation.

	Predicted Probability of Parental Separation									
	0-20%ile		21-40%ile		41-60%ile		61-80%ile		>80%ile	
	Intact (*n* = 262)	Separated (*n* = 29)	Intact (*n* = 245)	Separated (*n* = 47)	Intact (*n* = 197)	Separated (*n* = 91)	Intact (*n* = 159)	Separated (*n* = 130)	Intact (*n* = 60)	Separated (*n* = 224)
Paternal alcoholism, *n* (%)	4 (2)	0 (0)	9 (4)	1 (2)	15 (8)	4 (4)	37 (23)	32 (25)	37 (62)	169 (75)*
Maternal alcoholism, *n* (%)	4 (2)	0 (0)	4 (2)	2 (4)	2 (1)	0 (0)	6 (4)	7 (5)	8 (13)	35 (16)
Maternal smoking, *n* (%)	56 (21)	7 (24)	124 (51)	33 (70)*	105 (53)	54 (59)	115 (72)	85 (65)	53 (88)	177 (80)
Maternal age at twins' birth[a], *n* (%)										
< 20	1 (0.004)	0 (0)	1 (0.004)	1 (2)	0 (0)	0 (0)	11 (7)	15 (12)	16 (27)	54 (24)
20-24	11 (5)	0 (0)	47 (19)	3 (6)*	65 (33)	30 (33)	68 (43)	59 (45)	19 (32)	78 (35)
≥ 35	49 (19)	6 (21)	13 (5)	3 (6)	6 (3)	1 (1)	4 (3)	4 (3)	3 (5)	3 (1)
Paternal educational attainment[b], *n* (%)										
< 12 years	2 (0.008)	1 (3)	3 (1)	2 (4)	18 (9)	10 (11)	30 (19)	23 (18)	15 (25)	61 (27)
≥ 13 years	248 (95)	27 (93)	153 (62)	32 (68)	81 (41)	38 (42)	43 (27)	28 (22)	9 (15)	38 (17)

Note. Within quintile, intact and separated families differ significantly at

†$p < 0.10$.

*$p < .05$

[a]Reference group = ages 25-34.

[b]Reference group = 12 years.

Table 5

Prevalence of early substance involvement (by age 15) in European ancestry twins from intact and separated families, by quintile of predicted probability of parental separation.

	Predicted Probability of Parental Separation									
	0-20%ile		21-40%ile		41-60%ile		61-80%ile		>80%ile	
	Intact (*n* = 313)	Separated (*n* = 35)	Intact (*n* = 285)	Separated (*n* = 62)	Intact (*n* = 227)	Separated (*n* = 109)	Intact (*n* = 165)	Separated (*n* = 214)	Intact (*n* = 81)	Separated (*n* = 284)
Alcohol use, *n* (%)	67 (21)	11 (31)^{ns}	62 (22)	26 (42)	59 (26)	40 (37)†	61 (37)	42 (20)	14 (17)	119 (43)
Alcohol Intoxication, *n* (%)	24 (8)	5 (14)^{ns}	17 (6)	16 (26)	18 (8)	15 (14)^{ns}	14 (7)	30 (18)	3 (4)	59 (21)
Cigarette use, *n* (%)	78 (25)	17 (49)	103 (36)	37 (60)	95 (42)	65 (60)	87 (41)	101 (61)	51 (63)	191 (68)^{ns}
Regular smoking, *n* (%)	17 (6)	5 (15)^{ns}	24 (9)	14 (23)	22 (10)	16 (15)^{ns}	22 (10)	29 (18)†	13 (16)	76 (27)†
Cannabis use, *n* (%)	12 (4)	4 (12)^{ns}	11 (4)	13 (22)	12 (5)	11 (10)^{ns}	13 (6)	19 (12)^{ns}	5 (6)	53 (19)
Other illicit drug use, *n* (%)	6 (2)	1 (3)^{ns}	0 (0)	4 (7)	1 (< 1)	4 (4)†	2 (1)	10 (6)	2 (3)	10 (4)^{ns}

Note. Within quintile, intact and separated families differ significantly at $p < .05$, unless otherwise noted

Because of imperfect matching on paternal alcoholism in the highest quintile, logistic regression analyses were conducted predicting each substance use variable from parental separation, unadjusted and adjusted for paternal alcoholism, with comparable effects of parental separation observed across all substance use variables.

$^{\dagger}p < 0.10$

$^{ns}p \geq 0.10$.

Table 6

Family background characteristics of intact and separated African ancestry families, by quintile of predicted probability of parental separation.

	Predicted Probability of Parental Separation									
	0-20%ile		21-40%ile		41-60%ile		61-80%ile		>80%ile	
	Intact (*n* = 25)	Separated (*n* = 15)	Intact (*n* = 14)	Separated (*n* = 25)	Intact (*n* = 8)	Separated (*n* = 33)	Intact (*n* = 6)	Separated (*n* = 31)	Intact (*n* = 0)	Separated (*n* = 44)
Paternal alcoholism, *n* (%)	6 (24)	1 (7)	0 (0)	1 (4)	1 (13)	3 (9)	0 (0)	2 (6)	--	21 (48)
Maternal alcoholism, *n* (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	--	9 (20)
Maternal smoking, *n* (%)	16 (64)	11 (73)	13 (93)	19 (76)	6 (75)	21 (63)	3 (50)	17 (55)	--	27 (61)
Maternal age at twins' birth[a], *n* (%)										
< 20	1 (4)	0 (0)	0 (0)	0 (0)	0 (0)	1 (3)	2 (33)	13 (42)†	--	19 (43)
20-24	2 (8)	4 (27)	4 (29)	5 (20)	6 (75)	14 (42)†	2 (33)	16 (52)†	--	13 (30)
≥ 35	1 (4)	1 (7)	0 (0)	0 (0)	1 (13)	2 (6)	0 (0)	1 (3)	--	0 (0)
Paternal educational attainment[b], *n* (%)										
< 12 years	2 (8)	0 (0)	3 (21)	3 (12)	0 (0)	10 (30)	3 (50)	8 (26)	--	13 (30)
≥ 13 years	20 (80)	14 (93)	5 (36)	9 (36)	1 (13)	3 (10)	0 (0)	1 (3)	--	13 (30)

Note. Within quintile, intact and separated families differ significantly at

*$p < .05$

†$p < 0.10$.

[a]Reference group = ages 25-34.

[b]Reference group = 12 years.