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Parental Alcohol Use Disorder and Offspring Marital Outcomes

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Abstract

Aims: We tested whether parental alcohol use disorder (AUD) predicted adult offspring's likelihood of marriage and marriage to an AUD-affected spouse; whether effects differed as a function of the sex or number of affected parents; and whether they were robust to confounders.

Design: Sex-stratified Cox and logistic regression models.

Setting: Sweden.

Participants: 1,171,070 individuals (51% male) born 1965–1975.

Measurements: Obtained from legal, medical, and pharmacy registries. Predictor was parent AUD. Outcomes were marriage and spouse AUD. Adjustments included offspring birthyear and AUD; parental education, marriage, divorce, criminal behavior, and drug abuse.

Findings: Male and female offspring of AUD-affected parents were more likely to marry at younger ages (< 25), illustrative unadjusted $HR_{age\ 20} = 1.22$ [1.17, 1.28] and 1.34 [1.20, 1.39], and were less likely to marry at older ages (> 25), $HR_{age\ 30} = 0.79$ [0.78, 0.81] and 0.82 [0.81, 0.84]. Parental AUD was associated with higher odds of having an affected spouse for males and females, $OR = 1.47$ [1.38, 1.57] and 1.63 [1.56, 1.70]. Effects were more pronounced for those with two versus one AUD-affected parent and adjustments negligibly attenuated effects. Daughters of affected mothers (versus fathers) were more likely to have AUD-affected husbands, $OR = 1.68$ [1.54, 1.84] versus 1.56 [1.48, 1.64], while there was no difference in sons.

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Conclusions: Parental AUD was associated with a higher probability of marriage at younger ages, a lower probability of marriage at older ages, and a higher likelihood of marriage to an affected spouse. Most of these effects got stronger as the number of AUD-affected parents increased, and held after controlling for parents' SES, marital history, other externalizing disorders, and offspring's own AUD status. Daughters of affected mothers were more likely to have an affected spouse.

Alcohol use disorder (AUD) is a common and costly disorder [1], and in 2010 alone affected an estimated 4.1% of the global population and accounted for nearly 18 million disability-adjusted life years [1, 2]. The toll of AUD reaches beyond afflicted individuals, and in particular impacts immediate family members [3]. A number of questions remain about the long-term consequences of parental AUD on offspring. Despite the popular belief that the offspring of AUD-affected parents experience psychosocial difficulties in adulthood, there is often limited empirical support for this perception [4, 5]. Here, we tested whether parental AUD was associated with offspring marital outcomes, including the likelihood of marriage and the probability of marrying an AUD-affected spouse. Mapping these associations is important both for clarifying the legacy of parental AUD on adult offspring's adjustment and for understanding how parental AUD may contribute to offspring's selection into or creation of marital environments that may in turn change their own likelihood of developing AUD [6, 7].

Studies of parental AUD and the likelihood of offspring marriage show mixed results. In cross-sectional analyses of a US national sample (ages 18–39), parental alcohol problems were associated with a higher probability of marriage [8], while analyses of another US national sample (adults 18+) found a lower probability of being married [9]. Finally, in a US community sample (ages 40+), parental AUD was not associated with likelihood of marriage but was associated with earlier age at marriage [10]. It is difficult to directly compare these studies to one another owing to differences in how they operationalized marital status (i.e., current versus lifetime marriage); however, one possible explanation for these mixed results is that age-dependent patterns are obscured in cross-sectional analyses of samples that include a range of different ages.

Although it is popularly believed that parental AUD is associated with an increased likelihood of partnering with an affected spouse [11], the findings are also mixed. Hall et al. [12] found that male AUD probands with problem-drinking mothers were more likely to have problem-drinking wives or wives who were abstinent. Schuckit [13] found that daughters (but not sons) with a family history of AUD were more likely to have AUD-affected spouses. In contrast, McLeod [14] found no association between individuals' parental histories of alcohol or drug use and their spouses' alcohol or drug dependence. Likewise, Schuckit et al. [15] found no elevation in parental AUD among women who married AUD-affected men.

Sher [4] and others [5] suggest that inconsistencies emerging from studies of the outcomes of adult children of AUD-affected parents may be attributable to small sample sizes, restrictive sampling strategies, and imprecise measurement of parental AUD. Thus, there is a need for adequately powered population-based samples with clear measurement to fully

understand the legacy of parental AUD [5, 9, 11]. In the present study, we address these limitations and use population-based data from Swedish national registries to examine the associations between parental AUD and offspring marital outcomes. This sample also makes it possible to extend the prior literature to consider more nuanced questions about whether having two affected parents is more strongly associated than having just one, and whether the sex of the affected parent matters.

We examined two questions: *i*) Is parental AUD associated with the likelihood of offspring marriage and are these effects age dependent? and *ii*) Is parental AUD associated with marrying an AUD affected spouse? For each of these questions, we examined whether the pattern of effects differed as a function of the number of affected parents; the sex of the affected parent; and whether any observed effects of parental AUD on offspring marital outcomes were robust to statistical controls for potential confounders. Selected confounders included offspring year of birth (to account for potential cohort differences), offspring AUD prior to marriage (to control for selection effects), parental education (proxy for socioeconomic status), parental marital history (marriage and divorce), and other parental externalizing behaviors (criminal behavior and drug abuse). We selected these covariates in order to examine uniqueness and specificity of the associations between parental AUD and offspring marital outcomes, above and beyond other correlated factors [16, 17], including the offspring's own AUD.

Methods

Sample

We linked nationwide Swedish registers using unique identification numbers (replaced with anonymized serial numbers) assigned at birth or immigration to all Swedish residents. Only Swedish-born residents were included in the current analyses. Because registry data are available for different time frames, we used individuals born between 1965–1975 to maximize the availability of information across registries while enabling us to include most individuals who had reached the average age of marriage in Sweden and for whom data on parental AUD were available.

Data came from a series of population registries, which are detailed in [7, 18]. Briefly, the population registries used (and the respective data from each) included the: Total Population Register (marital status from 1968 and family ID from 1990); Swedish Census (information on household) every 5 years from 1960 to 1990; Longitudinal Integration Database for Health Insurance and Labour Market Studies Register (information on parental education) from 1990 to 2012; Multigeneration Register (sex, birth year and month, and linking children with their parents); Swedish Hospital Discharge Register (hospitalizations from 1964 to 2012); Outpatient Care Register (information from all outpatient clinics from 2001 to 2012); Swedish Mortality Register (dates and causes of deaths from 1973 to April 2014); Swedish Crime Register (complete national data on all convictions in lower courts from 1973 to 2012); the Swedish Suspicion Register (data on individuals strongly suspected of crime from 1995 to 2012). The ethics committee in Lund, Sweden approved the study; consent was waived.

Measures

Outcome variables.—There were two outcomes of interest: marriage (up to the end of observations, in 2013), and marriage to an individual with a lifetime history of AUD. For the latter outcome, we study marriage before age 40. We elected to consider lifetime AUD, rather than AUD prior to or at the time of marriage, under the rationale that characteristics indicative of AUD risk (on which individuals may select spouses) are likely to be present prior to an AUD-related registration. Only the first marriage was considered for the current analyses. Methods for identifying individuals with AUD are detailed elsewhere [19]. Briefly, individuals were classified as having a lifetime history of AUD if they received an alcohol-related primary or secondary medical diagnosis, at least 2 alcohol-related registrations in the Crime and Suspicion register (e.g., drunk driving), or had a record of a prescription for a drug used to treat AUD (e.g., disulfiram, acamprosate).

Independent variables.—For each marital outcome, we tested the effect of a parental lifetime history of AUD, defined using the same criteria as described above. Parental AUD status was operationalized in multiple ways: i) any AUD parent; ii) number of affected parents; and iii) paternal versus maternal history of AUD.

Confounders.—Covariates included offspring birth year (by year); parental education, operationalized as the highest level of education attained by either parent and categorized into 3 levels (1 = compulsory school, 2 = high school, and 3 = university); parental marriage, operationalized as any of the biological parents ever married; parental divorce, operationalized as any of the biological parents ever divorced; parental criminal behavior (CB), or drug abuse (DA), as determined from medical and legal prescription registers [for detailed information concerning the International Classification of Disease codes and legal convictions for each, see 20]; and offspring AUD prior to marriage. Individuals with missing information on any of the parental characteristics were excluded.

Statistical Methods

We used Cox proportional hazard methods to model the time to marriage, after age 18, as a function of parental AUD, censoring at death, migration, or end of follow-up (2013), whichever came first. Descriptive data suggested a time-varying coefficient for parental AUD, as individuals with parental AUD had a higher probability of marriage at younger ages, but a lower probability at older ages, compared to individuals with no parental AUD. We therefore included two additional parameters representing the change in the hazard ratio (HR) for parental AUD as a function of the logarithm of follow-up time. The first parameters represent how HR changes at younger ages while the second captures the change at older ages. The breakpoint was decided based on lowest AIC in a crude model. To ease interpretation, we present the HR for parental AUD at specific ages. We used logistic regressions for the marriage to an AUD-affected spouse analyses and included only individuals who had married.

For each marital outcome we ran three models. In our primary model, Model 1, we assessed the impact of parental AUD status on each outcome. In Model 2, we included the year of birth and parental covariates (education, marriage, divorce, CB, and DA) to examine the

uniqueness and specificity of parental AUD on offspring marital outcomes above and beyond these potential confounders. In Model 3, we examined whether the effects of parental AUD were robust after the addition of the offspring's own AUD status. We investigated the possible impact of number of affected parents and parental sex by taking the sex of parent into account when only one parent was affected. The comparison of interest for these latter analyses was whether a model that distinguished between maternal and paternal AUD fit better (according to AIC fit statistics, where smaller values indicate better fit) than a model that did not. We present results stratified by offspring sex in view of preliminary analyses that indicated sex differences in some effects. Statistics for comparisons of effects within a model are based on the asymptotic chi-square, and all analyses were run in SAS 9.4 (SAS Institute Inc., Cary, NC, USA).

Results

Descriptive statistics

In total, 1,171,070 individuals (51.40% male) met inclusion criteria for the present study. Descriptive statistics for this sample can be found in Table 1. Descriptive information for the marital outcomes as a function of each operationalization of parental AUD (i.e., AUD in any parent; number of affected parents; and paternal vs. maternal AUD) are shown in Supplementary Tables S1–S3.

Parental AUD and Likelihood of Offspring Marriage

The results from Cox proportional hazard models of marriage as a function of parental AUD are summarized in Table 2. As shown in Model 1 and visually summarized in Figure 1, there were age-dependent associations between parental AUD and likelihood of offspring marriage. At younger ages (prior to approximately 25 years of age), a parental history of AUD was associated with a higher likelihood of marriage. However, for those who did not marry by this time, this association changed direction such that at later ages (approximately ages 25 and older), a parental history of AUD was associated with a lower likelihood of marriage. These hazard ratios were largely the same following the addition of the birth year and the parental education, marriage, divorce, criminal behavior, or drug abuse covariates (Model 2) and the offspring AUD covariate (Model 3).

The results from Cox models of marriage as a function of the number of AUD-affected parents are summarized in Table 3. As shown in Model 1 and visually summarized in Figure 2, there were age-dependent associations between the number of affected parents and likelihood of offspring marriage. At younger ages (prior to approximately 25 years of age), having one or two parents with AUD was associated with a higher likelihood of marriage. However, at later ages (approximately ages 25 and older), having one or two parents with AUD was associated with a lower likelihood of marriage. As shown in the illustrative age-specific parameters summarized in the bottom of Table 3, these associations were generally stronger when individuals had two affected parents versus one. The age-dependent pattern in the hazard ratios was largely the same in the adjusted models (Models 2 and 3), with the exception that some of the comparisons between two and one affected parents were no longer significant at older ages (i.e., >35). Differentiating between parent-specific (i.e.,

paternal or maternal AUD) worsened model fit for male ($AIC_{\text{any one parent}} = 8,071,154.1$ versus $AIC_{\text{parent specific}} = 8,071,156.2$) and female offspring ($AIC_{\text{any one parent}} = 8,574,964.2$ versus $AIC_{\text{parent specific}} = 8,574,966.1$), indicating no differences as a function of the sex of the affected parent.

Parental AUD and Marriage to an AUD-affected Spouse

The results from multivariable logistic regression models of whether one married an AUD affected spouse as a function of parental AUD are summarized in the top of Table 4. As shown in Model 1 and visually summarized in Figure 3A, having an affected parent increased the odds of marrying an affected spouse for both male and female offspring. This association was attenuated somewhat in the adjusted models (Models 2 and 3).

The results from multivariable logistic regression models of whether one married an AUD-affected spouse as a function of the number of affected parents are summarized in the bottom of Table 4. As shown in Model 1 and visually summarized in Figure 3B, having one or two affected parents (compared to no affected parents) increased the odds of having an affected spouse for both male and female offspring. Having two affected parents increased the odds of having an affected spouse compared to having just one affected parent for males, $OR = 1.35 [1.09, 1.67]$ and females, $OR = 1.36 [1.18, 1.55]$. These associations were attenuated somewhat in the adjusted models (Models 2 and 3). Differentiating between maternal and paternal AUD worsened model fit for male offspring ($AIC_{\text{any one parent}} = 67,293.39$ versus $AIC_{\text{parent specific}} = 67,295.24$), but modestly improved model fit for female offspring ($AIC_{\text{any one parent}} = 130,389.47$ versus $AIC_{\text{parent specific}} = 130,389.08$). Thus, while sons' likelihood of marriage to an affected spouse did not differ as a function of the affected parent's sex, daughters of affected mothers (compared to affected fathers) were more likely to have AUD-affected husbands, $OR = 1.68 [1.54, 1.84]$ versus $1.56 [1.48, 1.64]$.

Discussion

Although it has been hypothesized that parental AUD may have enduring consequences for offspring marital outcomes—including whether and to whom offspring marry—previous investigations have produced inconsistent results and have been hampered by key methodological limitations related to sampling and measurement. We aimed to overcome these limitations and examined these questions using Swedish national registry data. We first asked whether parental AUD was associated with likelihood of marriage in the offspring, and found moderate age-dependent effects whereby male and female offspring with an AUD affected parent were more likely to get married at younger ages, but were less likely to get married at older ages. This pattern was stronger for those who had two versus one AUD-affected parent; did not differ as a function of the sex of the affected parent; and held even after covarying for confounders including parental SES and marital behavior, other parental externalizing disorders, and the offspring's own AUD status. Because of the current study's statistical power and consideration of age-dependent effects, these results represent a substantive contribution to questions about the impact of parental AUD on offspring's likelihood of marriage, particularly given inconsistencies across prior research [8–10]. We also note that our finding that parental AUD was associated with a lower likelihood of

marriage at older ages is consistent with study of marital attitudes in a sample of never-married college students, which found that those with a parental history of alcohol abuse had more negative opinions about marriage, and a greater desire to wait for marriage [21].

Next, we asked whether parental AUD was associated with one's likelihood of partnering with an AUD affected spouse. For males and females, having a parent with AUD was associated with a moderate increase (60% and 62%, respectively) in the probability of having an affected spouse compared to those without such a parent. As with the likelihood of marriage analyses, these effects were stronger for those who had two AUD-affected parents versus one, and held after adjusting for confounders. There were some differences as a function of the affected parent's sex, although in the direction opposite the parental image theory [22] hypothesis that offspring select spouses resembling their opposite sex parent. Specifically, daughters of affected mothers were more likely to have an AUD-affected spouse compared to the daughters of affected fathers. Thus, in contrast to a previously mixed literature on this question [12, 14, 23], our analyses of population-based data from Sweden provide strong evidence that parental AUD is associated with increased risk of partnering with an affected spouse.

Multiple mechanisms may contribute to the parent-spouse resemblance for AUD. This may reflect phenotypic selection effects (whereby offspring of AUD affected parents tend to drink more and select partners who have similar levels of alcohol use) or socialization effects (whereby the offspring of AUD affected parents are more likely to have AUD themselves, and this in turn has a causal effect on the spouse's risk of becoming AUD affected) [24]. Because our goal here was to examine whether parental AUD influences marital outcomes, we did not further probe the specific selection and socialization mechanisms. However, we note that parent-spouse AUD resemblance remained significant even after including offspring AUD prior to marriage in the model. This suggests that parental AUD shapes the type of partner one marries even after accounting for the offspring's own premarital AUD phenotype.

Limitations

These results should be interpreted in view of several limitations. First, AUD diagnoses came from population records, and the prevalence of AUD was lower compared to estimates from other epidemiological surveys [25, 26]. The registry-based ascertainment method used here captures the most severely affected individuals, missing individuals with mild to moderate cases of AUD who avoid medical or legal problems. However, there is a high degree of concordance among the registries [27] and the prevalence of ICD AUD in Sweden is similar to that observed here [28]. The inclusion of less severely affected individuals may result in less pronounced effects. Second, we only included legally married couples in our analyses. Although 2011 data indicated that the bulk of Swedes over the age of 20 were married or in a civil or registered partnership (44%), cohabiting relationships without marriage were also relatively common (19%) [29]. Whether the pattern of findings generalizes to non-marital cohabiting relationships is unknown. Third, although we have included a series of covariates to assess the unique and specific effects of parental AUD on offspring marital outcomes, as in all observational studies there is risk that our effects are

attributable to confounding. Finally, these analyses were limited to native-born Swedes, and may not generalize to other cultures or nationalities.

Conclusions

Parental AUD has age-dependent associations with likelihood of marriage and is associated with a higher likelihood of marrying an affected spouse in the Swedish population. Above and beyond documenting the legacy of parental AUD on offspring marital outcomes, the results from this study also expand upon the literature showing that a harmonious marriage to a prosocial partner is protective against the development of AUD [6, 7] to demonstrate that not everyone is equally likely to find him or herself in this type of protective marital environment. Rather, those who are at increased risk of AUD (based on a parental history) find themselves in the least protective marital environments. Importantly, parental AUD is not a perfect predictor of offspring marital outcomes, highlighting the importance of identifying the other factors involved in whether, when, and to whom one marries. Ultimately, we hope that this knowledge will help clinicians and support groups geared towards the offspring of AUD affected parents to raise awareness that their family history can shape the types of environments that increase their own risk for AUD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. Whiteford HA, Ferrari AJ, Degenhardt L, Feigin V, Vos T The global burden of mental, neurological and substance use disorders: an analysis from the Global Burden of Disease Study 2010, PLoS One 2015; 10: e0116820. [PubMed: 25658103]
2. World Health Organization. Global Status Report on Alcohol and Health, Geneva: World Health Organization; 2014.
3. Grant BF Estimates of US children exposed to alcohol abuse and dependence in the family, Am J Public Health 2000; 90: 112–15. [PubMed: 10630147]
4. Sher K Children of alcoholics: a critical appraisal of theory and research Chicago, IL: University of Chicago Press; 1991.
5. Harter SL Psychosocial adjustment of adult children of alcoholics: a review of the recent empirical literature, Clin Psychol Rev 2000; 20: 311–37. [PubMed: 10779897]
6. Leonard KE, Rothbard JC Alcohol and the marriage effect, J Stud Alcohol 1999; 13: 139–46.
7. Kendler KS, Lönn SL, Salvatore J, Sundquist J, Sundquist K Effect of marriage on risk for onset of alcohol use disorder: a longitudinal and co-relative analysis in a Swedish national sample, Am J Psychiatr 2016; 173: 911–18. [PubMed: 27180900]
8. Dawson DA, Grant BF, Harford TC Parental history of alcoholism and probability of marriage, Journal of Substance Abuse 1992; 4: 117–29. [PubMed: 1504637]
9. Watt TT Marital and cohabiting relationships of adult children of alcoholics, J Fam Issues 2002; 23: 246–65.

10. Hill EM, Ross LT, Mudd SA, Blow FC Adulthood functioning: The joint effects of parental alcoholism, gender and childhood socio-economic stress, *Addiction* 1997; 92: 583–96. [PubMed: 9219380]
11. 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: 427–48. [PubMed: 1757657]
12. Hall RL, Hesselbrock VM, Stabenau JR Familial distribution of alcohol use: II. Assortative mating of alcoholic probands, *Behav Genet* 1983; 13: 373–82. [PubMed: 6639562]
13. Schuckit MA Are daughters of alcoholics more likely to marry alcoholics?, *Am J Drug Alcohol Abuse* 1994; 20: 237–45. [PubMed: 8042605]
14. Mcleod JD Social and psychological bases of homogamy for common psychiatric disorders, *J Marriage Fam* 1995; 57: 201–14.
15. Schuckit MA, Smith TL, Eng MY, Kunovac J Women who marry men with alcohol-use disorders, *Alcohol Clin Exp Res* 2002; 26: 1336–43. [PubMed: 12351927]
16. Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M Etiological connections among substance dependence, antisocial behavior, and personality: modeling the externalizing spectrum, *J Abnorm Psychol* 2002; 111: 411–24. [PubMed: 12150417]
17. Wolfinger NH Parental divorce and offspring marriage: early or late?, *Soc Forces* 2003; 82: 337–53.
18. Salvatore JE, Larsson Lönn S, Sundquist J, Sundquist K, Kendler KS Genetics, the rearing environment, and the intergenerational transmission of divorce: a Swedish national adoption study, *Psychol Sci* 2018; 29: 370–78. [PubMed: 29346036]
19. Kendler K, Larsson Lönn S., Salvatore J, Sundquist J, Sundquist K Divorce and the onset of alcohol use disorders: a Swedish population-based longitudinal cohort and co-relative study, *Am J Psychiat* 2017; 174: 451–58. [PubMed: 28103713]
20. Kendler KS, Lonn SL, Maes HH, Lichtenstein P, Sundquist J, Sundquist K A Swedish population-based multivariate twin study of externalizing disorders, *Behav Genet* 2016; 46: 183–92. [PubMed: 26494460]
21. Larson JH, Thyne TR Marital attitudes and personal readiness for marriage of young adult children of alcoholics, *Alcohol Treat Q* 1999; 16: 59–73.
22. Burgess EW, Wallin P Engagement and Marriage Philadelphia, PA: J. B. Lippincott; 1953.
23. Schuckit MA, Tipp JE, Kelner E Are daughters of alcoholics more likely to marry alcoholics, *Am J Drug Alcohol Abuse* 1994; 20: 237–45. [PubMed: 8042605]
24. Yamaguchi K, Kandel DB On the resolution of role incompatibility: Life event history analysis of family roles and marijuana use, *Am J Sociol* 1985; 90: 1284–325.
25. Grant BF, Goldstein RB, Saha TD, Chou SP, Jung J, Zhang H et al. Epidemiology of DSM-5 alcohol use disorder: Results from the National Epidemiologic Survey on Alcohol and Related Conditions III, *JAMA Psychiatry* 2015; 72: 757–66. [PubMed: 26039070]
26. Kringlen E, Torgersen S, Cramer V A Norwegian psychiatric epidemiological study, *Am J Psychiat* 2001; 158: 1091–98. [PubMed: 11431231]
27. Kendler KS, Ji J, Edwards AC, Ohlsson H, Sundquist J, Sundquist K An extended Swedish national adoption study of alcohol use disorder, *JAMA Psychiatry* 2015; 72: 211–8. [PubMed: 25565339]
28. Lundin A, Hallgren M, Forsman M, Forsell Y Comparison of DSM-5 classifications of alcohol use disorders with those of DSM-IV, DSM-III-R, and ICD-10 in a general population sample in Sweden., *J Stud Alcohol Drugs* 2015; 76: 773–80. [PubMed: 26402358]
29. Organisation for Economic Co-Operation and Development. SF3.3. Cohabitation rate and prevalence of other forms of partnership; 2016.

Figure 1.
Relative risk of offspring marriage as a function of parental AUD, presented at different ages

(A)

(B)

Figure 2.

Relative risk of offspring marriage as a function of the number of parents affected with AUD, presented as hazard ratios at different ages for males (Panel A) and females (Panel B).

(A) Marriage to AUD-affected spouse as a function of parental AUD

(B) Marriage to AUD-affected spouse as a function of number of parents with AUD

Figure 3.

Offspring marriage to an AUD-affected spouse as a function of parental AUD (Panel A) and as a function of the number of parents with AUD (Panel B).

Table 1.

Descriptive statistics

	Males n = 602,047 (51.40%)	Females n = 569,023 (48.60%)
<i>Marital Outcomes</i>		
Marriage	313,794 (52.12%)	335,958 (59.04%)
Age at First Marriage	32.07 (<i>SD</i> = 5.67)	29.96 (<i>SD</i> = 5.91)
AUD-Affected Spouse	6,066 (2.07%)	14,121 (2.61%)
<i>Parental AUD</i>		
Any AUD parent	75,233 (12.50%)	71,528 (12.57%)
Number of AUD Affected Parents		
0	526,814 (87.50%)	497,495 (87.43%)
1	69,904 (11.61%)	66,505 (11.69%)
2	5,329 (0.89%)	5,023 (0.88%)
AUD-Affected Mother	20,892 (3.47%)	19,728 (3.47%)
AUD-Affected Father	59,670 (9.91%)	56,823 (9.99%)
<i>Covariates</i>		
Parental Education		
Level 1	108,977 (18.10%)	102,353 (17.99%)
Level 2	277,431 (46.08%)	263,592 (46.32%)
Level 3	215,639 (35.82%)	203,078 (35.69%)
Parental Marriage	586,769 (97.46%)	554,728 (97.49%)
Parental Divorce	205,679 (34.16%)	196,788 (34.58%)
Parental Criminal Behavior	82,816 (13.76%)	77,644 (13.65%)
Parental Drug Abuse	15,580 (2.59%)	14,477 (2.54%)
AUD (Self)	30,102 (5.00%)	10,232 (1.80%)
Year of Birth	1969.86 (<i>SD</i> = 3.18)	1969.87 (<i>SD</i> = 3.18)

Abbreviation. AUD = Alcohol Use Disorder.

Table 2.
Survival models predicting marriage as a function of parental AUD versus no parental AUD

	<i>Males</i>			<i>Females</i>		
	Model 1 Hazard Ratio [95% CI]	Model 2 Hazard Ratio [95% CI]	Model 3 Hazard Ratio [95% CI]	Model 1 Hazard Ratio [95% CI]	Model 2 Hazard Ratio [95% CI]	Model 3 Hazard Ratio [95% CI]
Covariates						
Birth year	--	0.99 [0.99, 0.99]	0.99 [0.99, 0.99]	--	0.99 [0.99, 0.99]	0.99 [0.99, 0.99]
Parent Education (2 vs. 1)	--	1.12 [1.11, 1.13]	1.11 [1.10, 1.13]	--	1.08 [1.07, 1.09]	1.08 [1.07, 1.09]
Parental Education (3 vs. 1)	--	1.29 [1.27, 1.30]	1.27 [1.26, 1.28]	--	1.16 [1.15, 1.17]	1.16 [1.17, 1.15]
Parental marriage	--	1.75 [1.70, 1.79]	1.70 [1.66, 1.75]	--	1.72 [1.68, 1.77]	1.71 [1.66, 1.75]
Parental divorce	--	0.89 [0.88, 0.89]	0.90 [0.90, 0.91]	--	0.90 [0.89, 0.90]	0.90 [0.90, 0.91]
Parental CB	--	0.93 [0.92, 0.94]	0.96 [0.95, 0.97]	--	0.97 [0.96, 0.98]	0.98 [0.97, 0.99]
Parental DA	--	0.98 [0.96, 1.00]	1.01 [0.98, 1.03]	--	1.00 [0.97, 1.02]	1.01 [0.99, 1.03]
Offspring AUD	--	--	0.36 [0.35, 0.37]	--	--	0.37 [0.36, 0.38]
Parental AUD (any vs none) - estimated hazard ratios [95% CI] at specific ages						
Age 18	1.37 [1.29, 1.45]	1.49 [1.40, 1.58]	1.53 [1.44, 1.62]	1.62 [1.54, 1.69]	1.72 [1.64, 1.80]	1.74 [1.66, 1.82]
Age 20	1.22 [1.17, 1.28]	1.33 [1.27, 1.39]	1.37 [1.31, 1.43]	1.34 [1.20, 1.39]	1.43 [1.38, 1.48]	1.44 [1.40, 1.49]
Age 25	0.96 [0.94, 0.98]	1.05 [1.03, 1.07]	1.08 [1.06, 1.10]	0.90 [0.89, 0.92]	0.96 [0.95, 0.98]	0.98 [0.96, 0.99]
Age 30	0.79 [0.78, 0.81]	0.86 [0.84, 0.88]	0.89 [0.87, 0.91]	0.82 [0.81, 0.84]	0.88 [0.87, 0.89]	0.89 [0.88, 0.90]
Age 35	0.85 [0.84, 0.86]	0.92 [0.91, 0.93]	0.96 [0.95, 0.98]	0.88 [0.87, 0.89]	0.93 [0.92, 0.95]	0.95 [0.94, 0.97]
Age 40	0.90 [0.88, 0.92]	0.98 [0.96, 1.00]	1.03 [1.01, 1.05]	0.93 [0.91, 0.95]	0.99 [0.96, 1.02]	1.01 [0.98, 1.03]

Notes. **Boldface** indicates $p < 0.05$.

Abbreviations. CB = Criminal Behavior, DA = Drug Abuse, AUD = Alcohol Use Disorder.

Table 3.

Cox models predicting marriage as a function of number of AUD-affected parents

	<i>Males</i>			<i>Females</i>		
	Model 1 Hazard Ratio [95% CI]	Model 2 Hazard Ratio [95% CI]	Model 3 Hazard Ratio [95% CI]	Model 1 Hazard Ratio [95% CI]	Model 2 Hazard Ratio [95% CI]	Model 3 Hazard Ratio [95% CI]
Covariates						
Birth year	--	0.99 [0.99, 0.99]	0.99 [0.99, 0.99]	--	0.99 [0.99, 0.99]	0.99 [0.99, 0.99]
Parent Education [2 vs. 1]	--	1.12 [1.11, 1.13]	1.11 [1.10, 1.13]	--	1.08 [1.07, 1.09]	1.08 [1.07, 1.09]
Parental Education [3 vs. 1]	--	1.29 [1.27, 1.30]	1.27 [1.26, 1.28]	--	1.16 [1.15, 1.17]	1.16 [1.17, 1.15]
Parental marriage	--	1.75 [1.70, 1.79]	1.70 [1.66, 1.75]	--	1.72 [1.68, 1.77]	1.71 [1.66, 1.75]
Parental divorce	--	0.89 [0.88, 0.89]	0.90 [0.90, 0.91]	--	0.90 [0.89, 0.90]	0.90 [0.90, 0.91]
Parental CB	--	0.93 [0.92, 0.94]	0.96 [0.95, 0.97]	--	0.97 [0.96, 0.98]	0.98 [0.97, 0.99]
Parental DA	--	0.98 [0.96, 1.00]	1.01 [0.98, 1.03]	--	1.00 [0.97, 1.02]	1.01 [0.99, 1.03]
Offspring AUD	--	--	0.36 [0.35, 0.37]	--	--	0.37 [0.36, 0.38]
Parental AUD (one vs none) - estimated hazard ratios [95% CI] at specific ages						
Age 18	1.33 [1.25, 1.41]	1.44 [1.35, 1.53]	1.47 [1.38, 1.57]	1.48 [1.42, 1.55]	1.58 [1.51, 1.65]	1.59 [1.52, 1.66]
Age 20	1.20 [1.14, 1.25]	1.30 [1.28, 1.30]	1.33 [1.27, 1.39]	1.27 [1.23, 1.32]	1.36 [1.21, 1.40]	1.37 [1.32, 1.42]
Age 25	0.96 [0.94, 0.98]	1.04 [1.02, 1.06]	1.07 [1.05, 1.09]	0.93 [0.91, 0.94]	0.98 [0.97, 1.00]	0.99 [0.98, 1.01]
Age 30	0.80 [0.79, 0.82]	0.87 [0.84, 0.89]	0.90 [0.88, 0.92]	0.81 [0.80, 0.83]	0.87 [0.85, 0.88]	0.88 [0.86, 0.89]
Age 35	0.86 [0.85, 0.87]	0.93 [0.91, 0.94]	0.97 [0.95, 0.98]	0.88 [0.87, 0.89]	0.93 [0.92, 0.95]	0.95 [0.93, 0.96]
Age 40	0.91 [0.89, 0.93]	0.98 [0.96, 1.00]	1.03 [1.01, 1.10]	0.94 [0.77, 0.85]	1.00 [0.97, 1.02]	1.01 [0.99, 1.04]
Parental AUD (two vs none) - estimated hazard ratios [95% CI] at specific ages						
Age 18	2.04 [1.68, 2.49]	2.29 [1.88, 2.80]	2.46 [2.02, 3.00]	2.37 [2.06, 2.72]	2.60 [2.26, 2.98]	2.66 [2.32, 3.06]
Age 20	1.62 [1.40, 1.88]	1.82 [1.57, 2.22]	1.97 [1.69, 2.29]	1.77 [1.60, 1.95]	1.94 [1.75, 2.14]	2.00 [1.81, 2.20]
Age 25	1.00 [0.93, 1.06]	1.12 [1.05, 1.20]	1.22 [1.15, 1.30]	0.95 [0.90, 1.00]	1.04 [0.99, 1.09]	1.08 [1.03, 1.14]
Age 30	0.79 [0.62, 0.72]	0.75 [0.70, 0.81]	0.83 [0.77, 0.89]	0.73 [0.69, 0.77]	0.80 [0.75, 0.84]	0.83 [0.79, 0.88]
Age 35	0.74 [0.71, 0.78]	0.83 [0.79, 0.87]	0.93 [0.88, 0.97]	0.81 [0.77, 0.85]	0.89 [0.84, 0.93]	0.93 [0.88, 0.98]
Age 40	0.81 [0.75, 0.87]	0.91 [0.84, 0.98]	1.02 [0.95, 1.10]	0.89 [0.82, 0.97]	0.97 [0.90, 1.05]	1.03 [0.95, 1.12]
Parental AUD (two vs one) - estimated hazard ratios [95% CI] at specific ages						
Age 18	1.54 [1.25, 1.89]	1.60 [1.30, 1.96]	1.68 [1.37, 2.06]	1.60 [1.39, 1.85]	1.65 [1.43, 1.90]	1.68 [1.45, 1.94]
Age 20	1.35 [1.16, 1.58]	1.41 [1.20, 1.64]	1.48 [1.27, 1.73]	1.39 [1.25, 1.54]	1.43 [1.29, 1.58]	1.46 [1.32, 1.62]

	Males			Females		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
	Hazard Ratio [95% CI]	Hazard Ratio [95% CI]	Hazard Ratio [95% CI]	Hazard Ratio [95% CI]	Hazard Ratio [95% CI]	Hazard Ratio [95% CI]
Age 25	1.04 [0.97, 1.11]	1.08 [1.01, 1.15]	1.14 [1.07, 1.22]	1.03 [0.98, 1.08]	1.06 [1.01, 1.11]	1.09 [1.04, 1.14]
Age 30	0.83 [0.77, 0.90]	0.87 [0.81, 0.94]	0.92 [0.86, 0.97]	0.89 [0.84, 0.94]	0.92 [0.87, 0.97]	0.95 [0.90, 1.01]
Age 35	0.87 [0.82, 0.91]	0.90 [0.86, 0.95]	0.96 [0.91, 1.01]	0.92 [0.88, 0.97]	0.95 [0.90, 1.00]	0.99 [0.94, 1.04]
Age 40	0.89 [0.83, 0.96]	0.93 [0.86, 1.00]	0.99 [0.92, 1.07]	0.95 [0.87, 1.03]	0.98 [0.90, 1.06]	1.02 [0.93, 1.10]

Notes. **Boldface** indicates $p < 0.05$.

Abbreviations. CB = Criminal Behavior, DA = Drug Abuse, AUD = Alcohol Use Disorder.

Table 4.

Multivariate logistic regression models predicting spouse AUD as a function of parental AUD versus no parental AUD (top) and as a function of number of parents with AUD (bottom)

	Male Offspring			Female Offspring		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
	Odds Ratio [95% CI]	Odds Ratio [95% CI]	Odds Ratio [95% CI]	Odds Ratio [95% CI]	Odds Ratio [95% CI]	Odds Ratio [95% CI]
Spouse AUD as a function of parental AUD						
Parental AUD [any vs. none]	1.47 [1.38, 1.57]	1.20 [1.12, 1.30]	1.18 [1.09, 1.27]	1.63 [1.56, 1.70]	1.29 [1.23, 1.35]	1.28 [1.22, 1.34]
Birth year	--	0.96 [0.96, 0.97]	0.97 [0.96, 0.97]	--	0.93 [0.93, 0.94]	0.93 [0.93, 0.94]
Parent Education [2 vs. 1]	--	0.94 [0.88, 1.00]	0.94 [0.88, 1.01]	--	0.91 [0.87, 0.95]	0.91 [0.87, 0.95]
Parental Education [3 vs. 1]	--	0.77 [0.72, 0.83]	0.78 [0.72, 0.84]	--	0.76 [0.73, 0.80]	0.76 [0.73, 0.80]
Parental marriage	--	0.93 [0.79, 1.08]	0.94 [0.81, 1.10]	--	0.98 [0.88, 1.09]	0.99 [0.89, 1.10]
Parental divorce	--	1.40 [1.32, 1.48]	1.38 [1.31, 1.46]	--	1.32 [1.28, 1.37]	1.32 [1.27, 1.37]
Parental CB	--	1.23 [1.15, 1.32]	1.21 [1.13, 1.30]	--	1.41 [1.34, 1.47]	1.40 [1.34, 1.47]
Parental DA	--	1.08 [0.94, 1.25]	1.06 [0.92, 1.23]	--	1.21 [1.11, 1.32]	1.21 [1.10, 1.32]
Offspring AUD	--	--	1.51 [1.38, 1.66]	--	--	1.32 [1.19, 1.46]
Spouse AUD as a function of number of parents with AUD						
One AUD parent vs. none	1.44 [1.34, 1.54]	1.19 [1.10, 1.28]	1.16 [1.07, 1.25]	1.59 [1.52, 1.66]	1.27 [1.21, 1.33]	1.26 [1.20, 1.33]
Two AUD parents vs. none	1.94 [1.58, 2.38]	1.50 [1.22, 1.85]	1.42 [1.15, 1.75]	2.15 [1.89, 2.46]	1.54 [1.34, 1.76]	1.52 [1.32, 1.74]
Birth year	--	0.97 [0.96, 0.97]	0.97 [0.96, 0.97]	--	0.93 [0.93, 0.94]	0.93 [0.93, 0.94]
Parent Education [2 vs. 1]	--	0.94 [0.88, 1.01]	0.94 [0.88, 1.01]	--	0.91 [0.87, 0.95]	0.91 [0.87, 0.96]
Parental Education [3 vs. 1]	--	0.77 [0.72, 0.83]	0.78 [0.72, 0.84]	--	0.76 [0.73, 0.80]	0.77 [0.73, 0.80]
Parental marriage	--	0.95 [0.80, 1.12]	0.97 [0.82, 1.15]	--	1.00 [0.89, 1.12]	1.01 [0.90, 1.13]
Parental divorce	--	1.49 [1.33, 1.48]	1.39 [1.31, 1.46]	--	1.33 [1.28, 1.38]	1.32 [1.28, 1.37]
Parental CB	--	1.22 [1.14, 1.31]	1.20 [1.12, 1.29]	--	1.41 [1.34, 1.47]	1.40 [1.34, 1.47]
Parental DA	--	1.08 [0.94, 1.25]	1.06 [0.92, 1.23]	--	1.20 [1.09, 1.31]	1.19 [1.09, 1.30]
Offspring AUD	--	--	1.50 [1.37, 1.65]	--	--	1.30 [1.17, 1.44]

Notes. **Boldface** indicates $p < 0.05$.

Abbreviations. CB = Criminal Behavior, DA = Drug Abuse, AUD = Alcohol Use Disorder.