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Alcohol use disorder and divorce: Evidence for a genetic correlation in a population-based Swedish sample

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Abstract

Aims—We tested the association between alcohol use disorder (AUD) and divorce; estimated the genetic and environmental influences on divorce; estimated how much genetic and environmental influences accounted for covariance between AUD and divorce; and estimated latent genetic and environmental correlations between AUD and divorce. We tested sex differences in these effects.

Design—We identified twin and sibling pairs with AUD and divorce information in Swedish national registers. We described the association between AUD and divorce using tetrachorics; and used twin and sibling models to estimate genetic and environmental influences on divorce, on the covariance between AUD and divorce, and the latent genetic and environmental correlations between AUD and divorce.

Setting—Sweden.

Participants—670,836 individuals (53% male) born 1940–1965.

Measurements—Lifetime measures of AUD and divorce.

Findings—AUD and divorce were strongly related (estimates [95% CIs]: males: r_{tet} = +0.44 [0.43, 0.45]; females r_{tet} = +0.37 [0.36, 0.38]). Genetic factors accounted for a modest proportion of the variance in divorce (males: 21.3% [7.6, 28.5]; females: 31.0% [18.8, 37.1]). Genetic factors accounted for most of the covariance between AUD and divorce (males: 52.0% [48.8, 67.9]; females: 53.74% [17.6, 54.5]), followed by nonshared environmental factors (males: 45.0% [37.5,

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54.9]; females: 41.6% [40.3, 60.2]). Shared environmental factors accounted for a negligible proportion of the covariance (males: 3.0% [−3.0, 13.5]; females: 4.75%, [0.0, 6.6].) The AUD-divorce genetic correlations were high (males: *r*A = +0.76 [0.53, 0.90]; females +0.52 [0.24, 0.67]). The nonshared environmental correlations were modest (males: *r*E = +0.32 [0.31, 0.40]; females: +0.27 [0.27, 0.36]).

Conclusions—Divorce and alcohol use disorder (AUD) are strongly correlated in the Swedish population, and the heritability of divorce is consistent with previous studies. Covariation between AUD and divorce results from overlapping genetic and nonshared environmental factors. Latent genetic and nonshared environmental correlations for AUD and divorce are high and moderate.

Alcohol use disorder (AUD) and divorce are relatively common, and are associated with one another (1–3). Cross-sectional studies show that divorced individuals consume more alcohol (4) and in more harmful patterns (5) than married individuals. Those who are divorced/separated/widowed are also more likely to have a lifetime or past 12-month AUD diagnosis (1) and to engage in alcohol-related risk behaviors such as drinking and driving (6) compared to marrieds. Individuals affected with AUD and couples where partners are discordant for heavy drinking are more likely to get divorced (7, 8), and divorce predicts longitudinal increases in drinking (4, 9). These associations have tangible costs, and alcohol-related mortality is higher among divorcees compared to their married counterparts (10). Identifying risk factors that predispose individuals to both AUD and divorce may inform avenues for prevention for these socially and personally costly behaviors (11–13). The goal of the present study was to clarify the nature and magnitude of the association between alcohol use disorder and divorce in a Swedish national sample.

Multivariate twin and sibling studies are one approach for decomposing the association between two outcomes into genetic and environmental components. Genetic factors influence AUD and divorce, and there is reason to believe that overlapping genetic factors may explain part of the association between the two. The heritability of AUD is 49% (14), and AUD is part of a spectrum of heritable externalizing disorders characterized by disinhibition and behavioral undercontrol (15–17). There are also genetic influences on divorce, with twin studies reporting heritability estimates of 15–53% (18–20). Several lines of evidence suggest that genetic factors may account for covariation between AUD and divorce. First, divorce is genetically correlated with a personality composite that includes low levels of constraint (i.e., higher behavioral disinhibition) as an indicator (21). Second, in a large sample of twins, latent genetic factors that contributed to AUD also contributed to earlier marital separation (22). Third, a history of divorce in biological mothers predicted AUD in adopted-away offspring in a Swedish national adoption study (23). Finally, alcohol use and problems are genetically correlated with indicators of romantic distress, such as conflict (24).

Nonshared environmental influences (i.e., environments or experiences to which only one twin or sibling is exposed) are also likely to contribute to the covariation between AUD and divorce. In particular, the characteristics of each twin or sibling's spouse—such as his or her personality or drinking habits—may be important nonshared environmental influences on alcohol and divorce outcomes (25–27). Although the spouses of twins are moderately

correlated for alcohol use (28), they are no more similar than random pairings on a number of other psychological traits (29) and may thus be a potential source of nonshared environmental influence. Furthermore, the bi-directional longitudinal associations between marital distress, divorce, and alcohol outcomes including consumption and AUD (4, 7–9, 30) suggest that the association between AUD and divorce may be in part causal. In multivariate twin models, causal effects are most typically captured by significant nonshared environmental covariation between the variables of interest (31). In practice it is difficult to identify the specific sources of nonshared environment; thus, nonshared environmental covariance would only be consistent with (but not proof of) causal effects (32).

Whether shared environmental factors (i.e., environmental factors that twins and siblings share, such as parental divorce) are likely to account for the covariation between AUD and divorce is less clear. Shared environmental influences account for 10% of the variance in AUD (14), and adoptive parents' divorce was a strong predictor of AUD in offspring in a Swedish national adoption study (23). However, twin and family studies have not yet found evidence of shared environmental influences on divorce, suggesting that the familial component of divorce is primarily genetic (18–20).

Our goal was to clarify the nature and magnitude of the association between AUD and divorce using a large population-based Swedish sample of twin and sibling pairs. We had four aims:

1. Describe the association between AUD and divorce.
2. Estimate the genetic and environmental influences on divorce given that this has not been reported previously from Sweden.
3. Estimate the degree to which genetic and environmental factors contribute to the covariance between AUD and divorce.
4. Estimate the latent genetic and environmental correlations between AUD and divorce.

We expected that there would be a positive association between lifetime AUD and divorce; that divorce would be moderately heritable; that genetic and nonshared environmental factors would explain a substantial portion of the covariation between AUD and divorce; and that there would be latent genetic and nonshared environmental correlations between AUD and divorce. We examined potential sex differences for all aims as recommended by Del Boca (33).

Methods

Design

We used Swedish national registers to identify a sample of twin and sibling pairs for whom there were data on AUD and divorce. We used tetrachoric correlations to investigate the association between AUD and divorce. We used twin and sibling models to estimate the genetic and environmental influences on divorce; to estimate the genetic and environmental sources of covariation between AUD and divorce; and to estimate the latent genetic and

environmental correlations between AUD and divorce. We examined sex differences in all analyses. For the twin and sibling analyses, we also tested for the inclusion of a unique twin environment parameter and age covariate.

Sample

We linked nationwide Swedish registers via the unique 10-digit identification number assigned at birth or immigration to all Swedish residents. The identification number was replaced by a serial number to ensure anonymity. The following sources were used to create our dataset: the Total Population Register, containing information about year of birth, sex, and yearly information on family and marital status from 1990; the Swedish Census, containing household information in 1960, 1965, 1970, 1975, 1980, 1985, and 1990; the Multi Generation Register, linking individuals born after 1932 to their parents; the Twin Register, including information of known zygosity with 95 % validity; the Hospital Discharge Register, containing hospitalizations for all Swedish inhabitants from 1964–2010; the Prescribed Drug Register, containing all prescriptions in Sweden picked up by patients from July 2005 to December 2010; the Outpatient Care Register, containing information from outpatient clinics from 2001 to 2010 (excluding Primary Health Care); the Primary Health Care Register, containing outpatient diagnoses from 2001–2007 for around 1 million patients from Stockholm and southern Sweden; the Crime Register that included national complete data on all convictions in lower court from 1973–2011; and the Swedish Suspicion Register that included national data on individuals strongly suspected of crime from 1998–2011. From the twin register we identified twin pairs with known zygosity, and from the Multi Generation Register we derived full and half siblings born within five years from each other. We included same-sex pairs where both individuals were born between 1940 and 1965, alive and living in Sweden at age 35. As detailed elsewhere (34), zygosity in the same-sex pairs from the twin registry was assigned using standard self-report items from mailed questionnaires. This is an indirect screening for level of cooperation because at least one of the pair had to return a questionnaire to the twin registry and cooperation was lower in subjects with AUD thus the prevalence is lower in this group compared to twins pairs not returning the questionnaires.

We assessed, using the Swedish national census and total population registries, the cohabitation status of the sibling pairs as the proportion of possible years lived in the same household until the oldest turned 18, the age of majority in Sweden. We defined pairs as “reared together” when this proportion was ≥ 80%, which was the full- and half siblings used in this report. We included all possible pairs, meaning some individuals were part of more than one sibling pair, for a total of 393,972 unique pairs made up of 670,836 unique individuals.

Measures

Individuals’ lifetime AUD was identified from Swedish medical registries by the following ICD codes: ICD8: 571.0, 291, 303, 980; ICD9: V79B, 305A, 357F, 571A, 571B, 571C, 571D, 425F, 535D, 291, 303, 980; and ICD 10: E244, G312, G621, G721, I426, K292, K700, K701, K702, K703, K704, K709, K852, K860, O354, T510, T512, T511, T513, T518, T519, F101, F102, F103, F104, F105, F106, F107, F108, F109; and from the

Prescribed Drug Register if retrieved disulfiram (Anatomical Therapeutic Chemical (ATC) Classification System N07BB01), acamprosate (N07BB03), or naltrexone (N07BB04). In addition, we identified AUD as convicted for or suspected of at least two alcohol-related crimes according to law 1951:649, paragraph 4 and 4A and law 1994:1009, Chapter 20, paragraph 4 and 5 from the Swedish Crime Register and code 3005 and 3201 in the Suspicion register.

We identified lifetime divorce by the married status variable in the Total Population Register. Unmarried individuals who ever cohabited with children were also included and defined as divorced if separated from the other parent. Individuals never married, as defined by the marital status variable, or never cohabiting with children, were excluded.

Statistical methods

We utilized an extension of the classical twin modelling, assuming a liability threshold model with three sources of liability: additive genetic (A), shared environment (C), and nonshared environment (E). In addition to twins we included full and half siblings. We assume that MZ twins share all their genes while DZ twins and full siblings share on average half of their genes, and half siblings share only a quarter of their genes identical by descent. The shared environment, reflecting family and community experiences, is the same within each pair although we examine the presence of a unique twin environment (T). The unique twin environment includes random developmental effects, environmental experiences not shared by siblings, and random error.

We began with a univariate model of divorce in view of the limited heritability information for this measure from previous studies. Following this, we conducted a bivariate analysis of AUD and divorce, which is based on the idea of an underlying liability to AUD and divorce assessed as binary outcomes. We set up a bivariate model using the Cholesky decomposition where the first factor loads on both AUD and divorce while the second loads only on the latter. Although the method can handle missing items we excluded pairs where one twin or sibling was never married and thus missing information on divorce. Preliminary analyses indicated that the prevalence of AUD was different in these individuals owing to the fact that the missingness pattern is not completely at random. To account for the variation in age in the sample, associated with prevalence of AUD and divorce we tested whether to allow the threshold to linearly depend on birth year, which we refer to as the ‘age regression’. To account for potential quantitative sex differences, we tested whether to allow the paths to vary across sex. Models were fit in OpenMx (35). We present estimates from the full ACE models, as recommended by Sullivan and Eaves (36).

We used the results from the best-fitting bivariate Cholesky model to quantify the results in two ways. First, we decomposed the covariance between AUD and DIV into A, C and E components by dividing the genetic, shared environmental and nonshared environmental covariances by the overall covariance. Second, we algebraically transformed the Cholesky model results into latent genetic and environmental correlations. These decomposed sources of covariance and latent genetic and environmental correlations provide an intuitive way to conceptualize the source and magnitude of the association between AUD and DIV.

Results

Descriptive statistics and association between AUD and divorce

The prevalence of lifetime AUD and divorce (DIV) and the twin/sibling pair tetrachoric correlations are presented in Table 1. For both sexes, the MZ twin pair correlations for AUD and DIV were higher than the corresponding DZ, FS, and HS pair correlations. The only exception was that the male-male FS pair correlation for DIV was higher than the male-male MZ pair correlation. The tetrachoric correlations [95% CIs] between lifetime AUD and DIV were +0.44 [0.43, 0.45] for males and +0.37 [0.36, 0.38] for females.

Univariate analysis of DIV

The results of the model fitting procedure are summarized in Table 2. The baseline ACE model (Model 1) included the unique twin environment (T) and age regression parameters. Dropping the T parameters (Model 2) resulted in no deterioration in model fit compared to Model 1, $\Delta AIC = -2.5$, $\chi^2(2) = 1.53$, $p = 0.47$. Constraining the male and female A, C, and E paths to be equal (Model 3) resulted in significant deterioration in fit compared to Model 2, $\Delta AIC = 24.9$, $\chi^2(2) = 28.82$, $p = 6 \cdot 10^{-7}$. Dropping the age regression parameters (Models 4 and 5) resulted in significant deterioration in fit, $\Delta AIC = 64.1$, $\chi^2(6) = 76.10$, $p = 2 \cdot 10^{-14}$ and $\Delta AIC = 64.5$, $\chi^2(6) = 76.42$, $p = 2 \cdot 10^{-14}$, respectively. Thus, the best fitting model was an ACE model with age regression where the parameters were allowed to vary across sex (Model 2). For males, variance components [95% CIs] for this model were: A = 21.3% [7.6, 28.5]; C: 2.7% [0.00, 9.7]; E = 76.1% [71.5, 82.8]. For females, variance components [95% CIs] were: A = 31.0% [18.8, 37.1]; C = 2.1% [0.0, 8.5]; E = 66.9% [62.9, 72.9].

Bivariate analysis of AUD and DIV

The results from the model fitting procedure are summarized in Table 2. The baseline ACE model (Model 1) included the unique twin environment (T) and age regression parameters. Dropping the T parameters (Model 2) resulted in no deterioration in model fit compared to Model 1, $\Delta AIC = -3$, $\chi^2(6) = 8.61$, $p = 0.19$. Constraining the male and female A, C, and E paths to be equal (Model 3) resulted in significant deterioration in fit compared to Model 2, $\Delta AIC = 239$, $\chi^2(7) = 253.39$, $p = 7 \cdot 10^{-51}$. Dropping the age regression parameters (Models 4 and 5) resulted in significant deterioration in fit compared to Models 1 and 2, $\Delta AIC = 490$, $\chi^2(12) = 513.63$, $p = 3 \cdot 10^{-102}$ and $\Delta AIC = 490$, $\chi^2(12) = 514.26$, $p = 2 \cdot 10^{-102}$, respectively. In sum, the best fitting model was an ACE model with age regression where the parameters were allowed to vary across sex (Model 2). The parameter estimates and 95% CIs for this best fitting model are presented in Figure 1.

Sources of covariation—As summarized in Figure 2, genetic factors accounted for the majority of the covariance between AUD and DIV (52.0% [48.8, 67.9] for males and 53.7% [17.6, 54.5] for females), followed by nonshared environmental factors (45.0% [37.5, 54.9] for males and 41.6% [40.3, 60.2] for females). Shared environmental factors accounted for only a trivial amount of the covariance (3.0% [−3.0, 13.5] for males and 4.8% [0.0, 6.6] for females).

Latent correlations—As summarized in Figure 3, the genetic correlations [95% CIs] between AUD and DIV were positive and large for males (rA = +0.76 [0.53, 0.90]) and females (rA = +0.52 [0.24, 0.67]). The nonshared environmental correlations between AUD and DIV were positive and moderate for males (rE = +0.32 [0.31, 0.40]) and females (rE = +0.27 [0.27, 0.36]). The shared environmental correlations for AUD and DIV were not different from zero for males (rC = +0.34 [−1.00, 0.46]) or females (rC = +0.76 [−1.00, 1.00]).

Discussion

We addressed three questions to further understand the magnitude and nature of the association between lifetime AUD and divorce in a nationally representative Swedish twin and sibling pair sample. We examined the association between AUD and divorce at the population level; conducted a univariate analysis of divorce; and then used a bivariate model to estimate the degree to which genetic and environmental factors accounted for the association between AUD and divorce. Consistent with previous epidemiological findings (1), we found in the Swedish general population born 1940–1965 that lifetime divorce and AUD were positively associated (r_{tet}: +0.44 for males and +0.37 for females). Thus, those with a lifetime history of divorce are more likely to have an AUD diagnosis (and vice versa), and this association is stronger in males than females. The stronger association between lifetime AUD and divorce for males compared to females diverges from previous work where the association was of similar magnitude across sexes (22).

The best fitting univariate model of divorce indicated that paths should be allowed to vary across sexes. According to this model, genetic factors accounted for a moderate proportion of the variance in divorce for males and females (estimates [95% CIs]: males 21.3% [7.6, 28.5] and females 31.0% [18.8, 37.1]). These estimates are consistent with two prior divorce heritability studies (estimates [95% CIs]: 15% [5, 19] and 32% [26, 38]) (19, 20) but are lower than the heritability estimate of 52.5% [41.9, 63.1] reported by McGue et al. (18). Nonshared environmental factors accounted for the majority of the variance in divorce for males and females (76.1% and 66.9%, respectively). Shared environmental factors were modest (2.7% and 2.1% of the variance in males and females) and did not differ from zero.

The best fitting bivariate model of AUD and DIV also indicated that paths should be allowed to vary across sexes. Genetic factors accounted for 52% of the AUD-DIV covariance in males and 54% in females. This suggests that there is a set of genetic factors that contribute to both AUD and divorce, and these genetic factors account for half of the observed association between AUD and divorce. Nonshared environmental influences accounted 45% of the AUD-DIV covariance in males and 41% in females, which indicates that there are events and experiences that twins and siblings don’t share that account for just under half of the observed association between AUD and divorce. Shared environmental influences accounted for a modest proportion of the AUD-DIV covariance in males (3%) and females (5%). The latent genetic correlation between AUD and divorce for males (rA = +0.76) and females (rA = +0.52) suggests that there is substantial—but incomplete—overlap between the genes that predispose individuals to AUD and divorce. The genetic correlation between AUD and divorce is consistent with previous work showing that heritable personality factors

related to behavioral undercontrol account for some of the heritability of AUD and divorce (21, 37). Furthermore, this genetic correlation is consistent with two potential interpretations: the first being that the association between AUD and divorce is attributable to non-causal genetic factors; the second being that genetic factors influence AUD, and that AUD in turn causes divorce (or vice versa). Whether this reflects non-causal and causal processes cannot be determined from a bivariate Cholesky model.

The nonshared environmental correlations between AUD and divorce for males (r_E = +0.32) and females (r_E = +0.27) further suggest that there are factors that differ between twins and siblings that predispose to AUD and divorce. Partners may be a meaningful nonshared environment (29) that contributes to the propensity to develop AUD or become divorced. Romantic partners influence one another's alcohol use (26), and discordance in partners' heavy drinking habits predicts divorce (27). Furthermore, the significant nonshared environmental correlation is also consistent with prior evidence of bidirectional causal relationships between AUD and divorce (38, 39).

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 (1, 40). However, there is a high degree of concordance among the registries (23) and the prevalence of ICD AUD in Sweden is similar to that observed here (41). Whether the pattern of genetic and environmental covariance for AUD and divorce generalizes to AUD cases identified through other ascertainment strategies is unknown. Second, because we could not disambiguate the relationship status of individuals who were cohabiting (unmarried) without children they were excluded from our analyses. Third, we included cohabiting couples with children together in our analyses, which may bias the AUD-divorce association. We addressed this possibility in supplementary analyses using strict definitions of marriage and divorce as defined by the Total Population Register. The latent genetic and environmental correlations were very similar to those obtained using the less strict definitions (results available upon request from the first author). Fourth, we limited our analyses to same-sex pairs, which precluded examination of qualitative sex differences (42), because attempts to include opposite-sex pairs resulted in unstable estimates. Finally, we included all possible same-sex pairs, which may artificially reduce the confidence intervals for the estimates. However, the impact of this is likely minimal given the large sample size.

Conclusions

Divorce and AUD were strongly correlated in the Swedish population, and divorce was moderately heritable. Genetic and nonshared environmental factors each accounted for approximately half of the covariance between AUD and divorce. Shared environmental factors accounted for minimal covariance. Latent genetic and nonshared environmental correlations for AUD and divorce were high and moderate, respectively. Questions remain about the specific factors and mechanisms that contribute to these latent genetic and environmental correlations, and the degree to which they reflect causal versus non-causal

processes. Ultimately, we hope that this knowledge will help refine marital interventions that address risk factors that predispose individuals to both AUD and divorce (43, 44).

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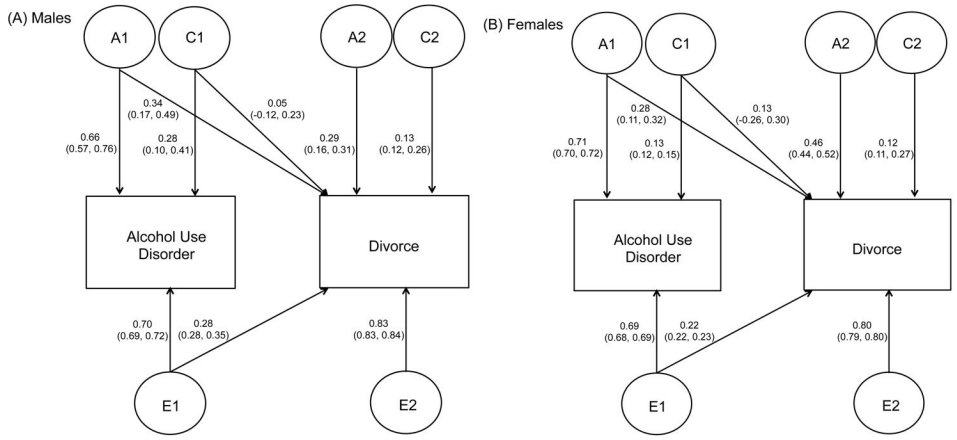

Figure 1.

Parameter estimates and 95% confidence intervals from the full bivariate Cholesky decomposition model for alcohol use disorder (AUD) and divorce (DIV) for males (Panel A) and females (Panel B). ‘A’ denotes additive genetic effects, ‘C’ denotes shared environmental effects, and ‘E’ denotes nonshared environmental effects. Genetic, shared environmental, or nonshared environmental factors contribute to the association between AUD and DIV when the 95% confidence intervals for the cross-paths from the A1, C1, and E1 latent factors to DIV do not include zero

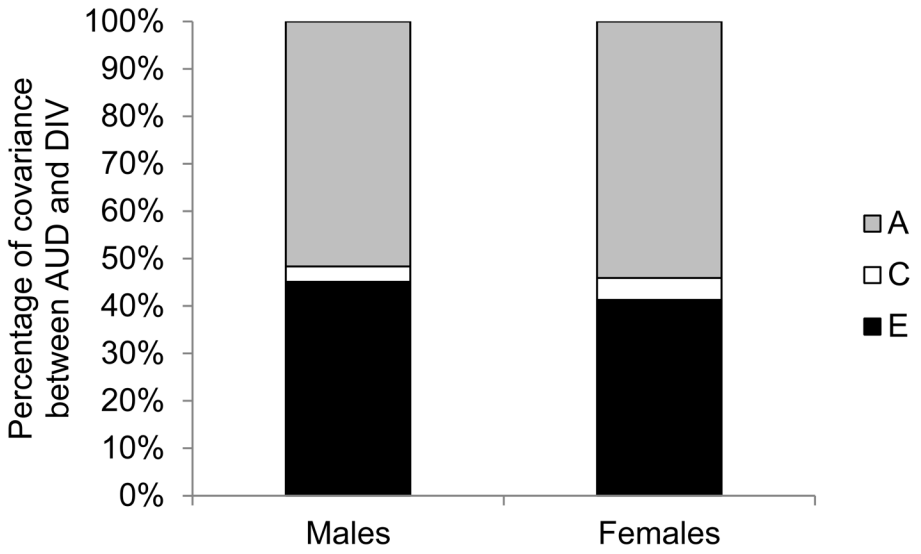

Figure 2.

Genetic and environmental sources of covariation between AUD and DIV for males and females. Percentages represent the degree to which covariance between AUD and DIV accounted for by additive genetic (A), shared environmental (C), and nonshared environmental (E) factors.

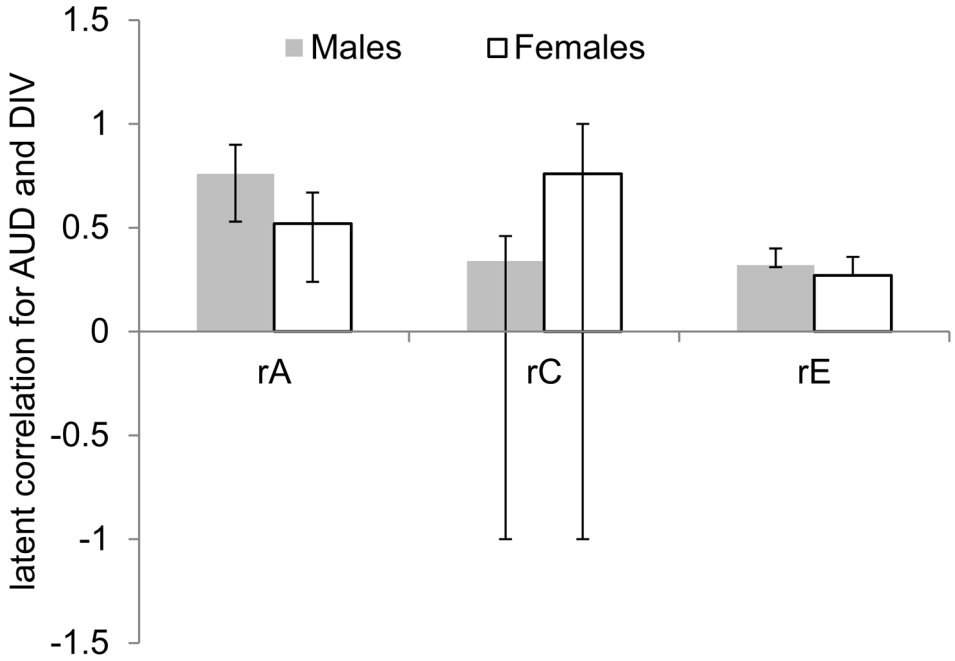

Figure 3.
Latent genetic (*r*A), shared environmental (*r*C), and nonshared environmental (*r*E) correlations between AUD and DIV. Bars define the 95% confidence intervals for the estimates.

Table 1

Prevalence and twin/sibling tetrachoric correlations for AUD and DIV.

Twin/Sibling type	*N* pairs	*N* unique individuals	Birth year, mean (SD)	AUD	DIV	Twin/sibling correlation (SE) AUD	Twin/sibling correlation (SE) DIV
MZ males	1,893	3,786	1951.2 (7.2)	6.1%	31.7%	0.56 (0.06)	0.26 (0.04)
DZ males	2,740	5,480	1951.0 (7.1)	7.2%	33.5%	0.32 (0.06)	0.14 (0.03)
MZ females	2,312	4,624	1951.6 (7.4)	2.8%	34.6%	0.64 (0.06)	0.34 (0.03)
DZ females	3,125	6,250	1950.9 (7.2)	3.5%	33.6%	0.24 (0.08)	0.21 (0.03)
FS male-male	199,650	338,792	1951.8 (6.9)	7.4%	33.0%	0.30 (0.01)	0.30 (0.01)
HS male-male	4,576	7,972	1953.4 (6.5)	12.1%	41.7%	0.27 (0.01)	0.17 (0.00)
FS female-female	170,972	296,765	1952.0 (6.8)	2.5%	33.0%	0.24 (0.01)	0.11 (0.00)
HS female-female	3,996	7,167	1953.5 (6.4)	4.7%	43.5%	0.27 (0.03)	0.11 (0.02)

Abbreviations. MZ = monozygotic; DZ = dizygotic; FS = full sibling; HS = half sibling; SE = standard error; AUD = alcohol use disorder; DIV = divorce

Table 2

Model fitting results

Model	−2 logL	AIC	Number of parameters	Comparison vs. model	Δ 2logL	Δ df	p
Univariate analysis of divorce							
1. ACTE, incl. age regression	389,161.1	−219,938.9	18				
2. ACE, incl. age regression	389,162.6	−219,941.4	16	1	1.53	2	0.47
3. ACE, incl. age regression, no quantitative effects	389,191.5	−219,916.5	14	2	28.82	2	$6 \cdot 10^{-7}$
4. ACTE	389,237.2	−219,874.8	12	1	76.10	6	$2 \cdot 10^{-14}$
5. ACE	389,239.1	−219,876.9	10	2	76.42	6	$2 \cdot 10^{-14}$
Bivariate analysis of Alcohol Use Disorder and Divorce							
1. ACTE, incl. age regression	1,270,277	−1,843,747	44				
2. ACE, incl. age regression	1,270,286	−1,843,750	38	1	8.61	6	0.19
3. ACE, incl. age regression, no quantitative effects	1,270,539	−1,843,511	31	2	253.39	7	$7 \cdot 10^{-51}$
4. ACTE	1,270,791	−1,843,257	32	1	513.63	12	$3 \cdot 10^{-102}$
5. ACE	1,270,800	−1,843,260	26	2	514.26	12	$2 \cdot 10^{-102}$

Abbreviations. ACE = Cholesky model with additive genetic (A), shared environmental (C), and nonshared environmental (E) factors; ACTE = ACE Cholesky model plus unique twin environment (T) factor.