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Shared Environmental Contributions to Substance Use

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Abstract

The current study examined the association between substance use in the household during childhood, parental attitudes towards substance use and lifetime substance use in males. Subjects included 1081 monozygotic and 707 dizygotic twins from the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders. Retrospective reports of substance use and features of the family environment (adult household substance use and parental attitudes towards substance use) were obtained using a life history interview. A trivariate Cholesky decomposition was conducted using the program Mx to decompose common shared environmental variance. Findings suggest that family environmental factors accounted for a large proportion of the shared environmental effects for illicit drug use. Results illustrate an important way of extending behavior genetic research to reveal specific etiological environmental mechanisms.

Keywords

Substance use; Shared environment; Family environment; Twins; Twin study

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Introduction

Until recently, researchers argued that shared (or family) environmental influences on psychological and behavioral outcomes were minimal (McGue and Bouchard 1998; Plomin et al. 2001; Plomin and Daniels 1987). This is because behavior genetic research often showed that environmental influences impacting on individual differences were largely non-shared or individual-specific in nature (Plomin and Daniels 1987). Non-shared environment refers to experiences that are unique to individuals within the same family which produce within-family differences while shared environment refers to experiences common to individuals raised within the same family that produce within-family similarities. This trend has been revealed with a multitude of phenotypes, including substance use and misuse (Agrawal and Lynskey 2008; Li et al. 2003; Prescott and Kendler 1999; van den Bree et al. 1998).

Importantly however, research examining the genetic and environmental risk factors for substance use and misuse are often drawn from adult samples and focus on substance use disorders. Recent research utilizing genetically informative samples of adolescents tells a different story indicating that shared environmental factors contribute substantially to individual differences in substance use and to psychological factors in general (Agrawal and Lynskey 2006; Burt 2009; Hopfer et al. 2003; Rende and Slomkowski 2009; Rhee et al. 2003). Several studies have also examined the change in genetic and environmental variance across development. These studies reveal that, in general, shared environmental influences are more important than genetic influences in childhood and adolescence yet almost disappear by adulthood (Baker et al. 2011; Hicks et al. 2007; Kendler et al. 2008; Pagan et al. 2006; Young et al. 2006). Examining substance initiation in adults provides similar results. Shared environment impacts substance initiation while this impact approaches zero for substance misuse (i.e. abuse and dependence). This is shown for a number of substances including cannabis, stimulant, cigarette, alcohol, and cocaine initiation (Kendler et al. 1999; Kendler and Prescott 1998a; Kendler and Prescott 1998b; Koopmans et al. 1999; Stallings et al. 1999; van den Bree et al. 1998).

A large number of studies have examined environmental risks for substance initiation, use, and other externalizing behaviors in adolescence and/or childhood from a phenotypic perspective. One of the most consistent findings is the importance of the peer group to initiation (Bauman and Ennett 1996; Hawkins et al. 1992; Hops et al. 2000). However, recent research shows the causal influence of peers may be overestimated and due to assortative friendship (Bauman and Ennett 1996; Heath and Martin 1988; Hill et al. 2008; Kandel 1996). Certain family environments also appear to play a role. For example, low levels of parental attachment and low parental monitoring predict initiation while proactive parents and clear parental communications discouraging use decrease the likelihood of initiation (Chilcoat and Anthony 1996; Kosterman et al. 2000; Sargent and Dalton 2001; Stice and Barrera 1995).

To date, however, few behavior genetic studies have sought to identify the sources of shared environmental variance on substance use. Decomposing this latent influence into specifics is an important extension of behavior genetic research and is often carried out when specific genes are examined to account for the heritability of a phenotype. It is equally important to decompose latent environmental variance into specific mechanisms. One such study showed that 77% of the shared environmental variance in early substance use was accounted for by peer deviance and parent–child relationship problems (Walden et al. 2004). Research examining additional externalizing phenotypes provides similar results (Burt et al. 2003; Pike et al. 1996; Rende and Slomkowski 2009). For example, approximately 15% of the

shared environmental variance in adolescent delinquency is accounted for by the parent–child relationship (Burt et al. 2007).

The current investigation sought to decompose the shared environmental variance of lifetime substance use utilizing retrospective reports on childhood environments obtained from an adult sample of male–male twins. We examined the sources of the association between licit and illicit substance use on the one hand and adult household substance use and parental attitudes toward substance use on the other. Both operate at the family-level and have significant phenotypic associations with substance use (Hops et al. 2000). We also aim to provide further evidence of the significance of shared environmental factors on the important trait of substance use.

Methods

Participants

This report is based on the third wave of interviews for adult males within the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (Kendler and Prescott 2006). Detailed descriptions of this sample can be found elsewhere (Gillespie et al. 2007; Kendler et al. 2003). Twins were selected from the population based Virginia Twin Registry and born between 1940 and 1974. The third interview wave (MM3) took place between 1998 and 2004 and was completed solely by members of male–male twin pairs. Twins were eligible for MM3 if they were a member of a male–male twin pair and both members had been interviewed in Wave 2. Responses rates for all twins at the previous two interview waves were 72 and 83%. Seventy-five percent of those twins contacted to participate in MM3 were interviewed. This includes 1081 monozygotic (MZ) and 707 dizygotic (DZ) twins aged 24–62 years (mean = 40.3, s.d. = 9.0) Zygosity was determined using a combination of self-report measures, photographs, and DNA analyses (Kendler et al. 2000).

Most participants were interviewed by telephone. However, a small number were interviewed in person. Interviewers had a Master's degree in a mental-health related field or a Bachelor's degree in this area and two years of clinical experience. Twin pairs were interviewed by different interviewers who were blind to information about the co-twin. This project was approved by Virginia Commonwealth University's IRB and twins provided informed consent before interviews.

Assessment

Information for the current project was obtained through retrospective assessment. Lifetime licit and illicit substance use was assessed by asking participants if ever in their lifetime they had used alcohol, cigarettes, marijuana, sedatives, cocaine, hallucinogen, and stimulants. For parental attitudes, participants were asked whether, during the time they were growing up, their parents would strongly agree, agree, disagree, or strongly disagree to their use of seven different substances. Agreement was rated by participants on a four point scale with four indicating strongly agree and one indicating strongly disagree. However, items were recoded so that the lowest possible item score was zero, which corresponds to strongly disagree, and a highest possible score of three which corresponds to strongly agree. Substances asked about included: smoking cigarettes, smokeless tobacco, drinking of coffee, drinking of alcohol, alcohol intoxication, marijuana use, and the use of “other drugs like cocaine or LSD”. The following phrasing was used to ask participants about their parent's attitudes toward substance use for each substance noted above: “It is OK for a teenager to smoke cigarettes?” A parental attitudes factor was created by summing the seven parental attitude items (one for each substance) together. Scores ranged from 0 to 14 (mean = 3.5, s.d. = 2.4) with higher scores indicating more agreement.

Household adult substance use was assessed in two ways. Approximately 60% of the sample was directly asked about their family members' (including mother, father, non-twin siblings, and other adults living in the household) substance use during childhood. Participants were asked how often these adults used six different substances including: cigarettes, smokeless tobacco, marijuana, alcohol, became intoxicated by alcohol use, and "other drugs such as cocaine or LSD". Participants were originally asked to score Items on a five point scale with scoring possibilities including: never, once or twice a year, a few times a month, once or twice a week, and nearly every day. A score of five corresponded to nearly every day and a score of one corresponded to never. However, items were recoded so that the lowest possible item score was zero, which corresponds to never, and a highest possible score of four which corresponds to nearly every day.

All participants were initially asked about household substance use in this fashion. However, due to privacy issues in asking twins about specific individual's substance use that arose in the middle of our study, questions were modified in the following way: participants were asked how often adults, in general, in their household used the six substances noted above. If the question applied to more than one person in the household, twins were asked to answer the questions for the individual who used most often. Questions, scoring possibilities, and item creation were identical to those previously described. To make responses comparable across assessments, the responses for the adult (mother, father, sibling, or other) described as most deviant within the initial assessment method was merged together with the more general household use assessment method.

No participant completed both household use question sets so we are unable to directly compare them. However, the means for the two assessment types are strikingly similar. For those participants asked directly about each family member's substance use, for which the most deviant response was used, the mean score was 7.16 (s.d. = 4.13). For the follow-up inquiry when twins were asked to consider the person who uses the most, the mean score was 6.58 (s.d. = 4.18).

An adult household substance use factor was created by summing the six household substance use items for each substance together. Scores ranged from 0 to 22 (mean = 7.0, s.d. = 4.2) with higher scores indicating more frequent use. Internal reliability was examined and Cronbach's alphas were 0.73 and 0.57 for parental attitudes and for the merged assessments of household substance use, respectively.

Analysis

Because of the very low frequency of participants indicating strong parental agreement with substance use and very frequent household use, which created missing cells, the four most deviant response sets for parental attitudes and the seven most deviant response sets for household substance use were merged together into one response category. For household substance use, sum scores 17–22 were merged into sum score 16, making 16 the highest possible score. For parental attitudes, sum scores 11–14 were merged into sum score 10, making 10 the highest possible score. These cut-offs were chosen because there were less than ten participants who endorsed these high sum scores. A combined "any illicit drug use" variable was also created to indicate any lifetime use of marijuana, cocaine, sedatives, stimulants, or hallucinogens.

Analyses were conducted in the structural equation modeling program Mx (Neale 1997). Data were treated as ordinal, and a raw data approach was used which allows results from both complete and incomplete twin pairs to be utilized. It is assumed that thresholds delineating the different categories overlay a normally distributed continuum of liability. The data were treated as ordinal because we were forced to combine the most deviant

response sets of the household substance use and parental attitudes towards use variables which eliminated the true continuous nature of the variables by eliminating possible response options and placing them all into one “category”. This also made the size of the difference between responses inconsistent (e.g. a true sum score of 10 for parental attitudes versus a true score of 16 forced to be 10). Dichotomization was additionally necessary due to the dichotomous nature of the substance use variables.

Genetic-modeling

First, standard biometric model fitting analyses were conducted to examine the genetic and environmental influences on the parental attitudes and household use variables. The sources of variation revealed in this design include additive genetic effects (A), which are the sum of allelic effects within and across genes, shared environmental effects (C), which refers to environments shared by the twins, and unique environmental effects (E), which refers to variance not shared by the twins and also includes measurement error. The basic principle of twin studies is that MZ twins are genetically identical while DZ twins are presumed to share, on average, 50% of their genes. Another important assumption of this model is that shared environmental effects correlate equally in MZ and DZ twin pairs while non-shared environmental effects are uncorrelated. Our expectation was that individual differences in the family-level factors, at least to a substantial degree, would reflect shared environmental influences. However, because twins are the same age this can also present its self as shared environmental variance. Therefore, an age corrected biometric model was also implemented, in order to determine the amount of shared environmental effects on the family-level variables after partitioning out the effects of age.

Cross-twin cross-trait correlations allow the covariance between traits to be delineated into additive genetic and environmental influences as is done with single phenotypes. A trivariate Cholesky decomposition was used to decompose the amount of shared environmental covariance between household substance use, parental attitudes towards substance use, and substance use (Fig. 1). Substance use was the downstream or dependent variable as we wanted to determine what proportion of the shared environmental effect on use was shared with the household adult use and parental attitude measures. From this model, the variance in substance use is parsed into components attributable to the genetic and environmental effects on household use and parental attitudes and substance specific genetic and environmental effects. For all analyses, we chose to only examine the full ACE model because we were specifically interested in examining shared environmental effects.

Results

Descriptive statistics for lifetime substance use and the family environment variables by zygosity are provided in Table 1. A majority of the sample had smoked or used alcohol in their lifetime. The mean scores for the family environment variables were similar in MZ and DZ twins. Before conducting twin modeling analyses, the rank biserial correlation macro for SAS 9.2 was used to assess the phenotypic correlation between the family environment and lifetime substance use by zygosity (SAS 2008). As can be seen in Table 2, phenotypic correlations were small to moderate. The polychoric correlation between parental attitudes and household substance use was moderate and significant at 0.29 ($P < 0.01$).

Age-corrected cross-twin, within-trait correlations for each phenotype were also examined (Table 3). In general, for lifetime substance use, MZ correlations were greater than the DZ correlations, with DZ correlations being estimated at slightly above half the MZ correlation. However, for lifetime alcohol use MZ and DZ correlations were quite similar suggesting that shared environmental factors are more important than genetic factors. Shared environmental effects were also suggested for the family environment variables as the DZ

correlations were greater than half the MZ correlations. MZ and DZ adult household substance use correlations were strikingly similar indicating that these measures reflected shared environmental factors much more than they indexed genetic factors.

Biometric model fitting

As can be seen in Table 4, and as predicted, both the household use and parental attitudes factors exhibit shared environmental variance. Before accounting for the effects of age, the shared environmental variance on both variables was substantial with shared environment accounting for 70% (95% CI: 60; 77) and 87% (95% CI: 81–89) of the variance in attitudes toward substance use and adult household substance use, respectively. However, these estimates decreased after accounting for age and, for parental attitudes, the confidence interval included zero. In the age corrected model shared environment accounted for 22% (95% CI: 0; 43) of the variance in parental attitudes toward substance use and 72% (95% CI: 60; 80) of the variance in adult household substance use.

Cholesky decomposition

Trivariate Cholesky decompositions were conducted with all substance use variables. Paths for the Cholesky decomposition are squared and summed to index the percentage of variance accounted for by shared environment. These results are presented in Table 5.

Results revealed that household substance use and parental attitudes toward substance use account for a proportion of the variance of shared environmental effects for lifetime substance use. With the exception of hallucinogen use, the family-level variables accounted for more of the variance for the illicit drugs than smoking or alcohol use. For cocaine, stimulant, and sedative use approximately 100% of shared environmental variance was accounted for. This is calculated by $\left(C_{31}^2+C_{32}^2\right) / \left(C_{31}^2+C_{32}^2+C_{33}^2\right)$ from Fig. 1 (this ratio is known with low precision because it is influenced by errors of estimation of all the parameters in the equation). Forty-four percent of the shared environmental variance was accounted for in any illicit drug use. For alcohol and smoking family environmental factors accounted for 23 and 14% of the shared environmental variance, respectively.

Importantly however, as shown in Table 5, the shared environmental influences for several of the substances were low and confidence intervals included zero. Additionally, several of the 95% confidence intervals were wide. This could explain the large range of estimates between substances. Taken together, results provide the greatest evidence for the influence of household substance use and parental attitudes toward substance use on lifetime illicit drug use, specifically marijuana, cocaine, and any illicit drug use. Each of these substances had significant shared environmental effects estimated.

Discussion

The aim of the present study was to begin to understand shared environmental processes that may contribute to lifetime substance use. Several results are noteworthy. First, the contribution of the shared environment to adult household substance use was substantial while the contribution to parental attitudes was moderate and nonsignificant after accounting for age. The small to moderate genetic and nonshared environmental effects likely reflect the fact siblings will not always see the same parenting behavior nor will the parental attitudes be expressed in the same way in different offspring. Additionally, the twin's view of their parents is inevitably shaped by their own personality and way of interacting. This is especially true for parental attitudes toward substance use, which had a much lower shared environmental estimate, given this variable is likely subject to more participant

interpretation. However, in general, our measures were successful at detecting environmental experiences shared by twins.

Secondly, results suggest that perceived substance use in the household during childhood and parental attitudes towards substance use may influence later substance use, specifically use of illicit drugs. Of note however, with the exception of alcohol use, the shared environmental effects on the substances were small to moderate. To our knowledge, there is only one previous report examining the covariance of parenting behaviors on substance use from a behavior genetics perspective (Walden et al. 2004). This report examined the covariance among peer deviance, parent–child relationship problems, and substance use and found that peers and parents account for 77% of the shared environmental variance in early substance use in adolescence (Walden et al. 2004). Our results are similar, suggesting household use and parental attitudes towards use account for a portion of the shared environmental variance in lifetime illicit substance use.

While we did not examine age at first use, results are consistent with previous studies showing parental monitoring and parenting behaviors as important factors for substance initiation as well as frequency of use (Andrews et al. 1993; Andrews et al. 1997; Chassin et al. 1993; Doherty and Allen 1994; Li et al. 2002; Nash et al. 2005; Rai et al. 2003; White et al. 2000). In fact, in past decades, parental substance use was thought to be the most powerful influence on adolescent initiation of the same substance (Glynn 1980). Importantly though, our “use” variables reflect any use in the household and not just among parents. Including any adult household substance use is an important strength of our study and reveals the importance of modeling use not only at the parental level. Interestingly however, more recent research reveals several parental behaviors mediate and/or moderate the relationship between parental modeling and adolescent substance use (Chassin et al. 1993; Doherty and Allen 1994; Farrell et al. 1995; Nation and Heflinger 2006). For example, parents who are problem drinkers may provide less support to children or may be unable to monitor their behaviors which might lead to substance use by the offspring (Chassin et al. 1993).

Additionally, several studies report that the importance of parental substance use and parenting behaviors varies relative to type of substance (Kosterman et al. 2000; Li et al. 2002). However, our results imply that household substance use and parental attitudes toward substance use are associated with lifetime substance use at a more general level. This is indicated by the fact our factors accounted for a moderate amount of the shared environmental variance in lifetime substance use for most substances, especially those of the illicit variety, despite the fact we did not examine household substance use and parental attitudes for each specific substance. This is consistent with research showing that parental influences generalize across adolescent use of cigarettes, alcohol and marijuana (Chassin et al. 1993).

Finally, the fact only a minimal amount of shared environmental variance was accounted for in alcohol use was somewhat surprising given the impact of parental use and parenting behaviors shown previously on adolescent alcohol use (Kosterman et al. 2000; Nash et al. 2005; Simons-Morton et al. 2001; Webster et al. 1994). As noted, confidence intervals for this covariance were wide. However, this may also suggest a difference between alcohol use and other substances with regard to shared environmental influences. Several possibilities may account for this. A previous study examining both parent and peer variables showed that peer variables tend to have a stronger association with alcohol use (Allen et al. 2003). Our all male sample could also be impacting findings. It is possible the impact of household substance use and parental attitudes towards use on lifetime alcohol use are different between the sexes and has less of an impact for males. Moreover, given widespread alcohol

advertising, individuals are likely to be exposed to norms that assert the acceptability of alcohol use compared to most of the other substances examined. Finally, the fact that we examined lifetime use in an adult sample rather than timing of alcohol initiation could also account for the minimal amount of shared environmental covariance exhibited. These family-level factors may only be important during the time period in which initiation occurs.

There are limitations to this study that warrant discussion. First, we are unable to determine the exact nature of the impact our family-level variables have on lifetime substance use. For example, a passive gene by environment correlation, which is the association between a child's inherited genotype and the environment in which the child was raised, could account for these findings. Second, adult household substance use and parental attitudes towards substance use were examined through twin report. Our factors therefore, are more accurately a measure of perceived behavior and attitudes. In regard to peer influence, research shows that perceived friend's use has a stronger association with substance use compared to peer's self-reported use of substances (Bauman and Ennett 1996; Hill et al. 2008; Iannotti et al. 1996; Kandel 1996). A similar influence was shown for child report of maternal drug use (Kandel 1996). Therefore, our results may only reflect perception of the environment rather than actual use and attitudes. Additionally, twin reports of parenting behaviors produce more genetic effects while parental self-reports produce lower nonshared and higher shared environmental estimates (Kendler 1996).

Third, the use of these family-level variables in twin modeling also has limitations, namely that these models cannot provide estimates of the environment that are unbiased by genetic (or unique environmental) effects (Turkheimer et al. 2005). In other words, the methods used in the present study indicate an association between parental attitudes/household substance use and substance use, but cannot distinguish with completely certainty whether the association is due to environmental mediation or common genetic factors (Purcell and Koenen 2005). Fourth, our data are retrospective. Subjects were asked to recollect household substance use and parental attitudes towards use when growing up. This significant time lag between childhood and assessments could impact the reliability and accuracy of the reports. However, we utilized a life history calendar in our assessments and a substantial body of evidence suggests that such methods, which reflect the structure of autobiographical memory and promote sequential retrieval within memory networks, can substantially improve the completeness and accuracy of retrospective reporting (Belli 1998; Cook et al. 2003; Freedman et al. 1988; Yoshihama et al. 2002).

Similarly, conclusions about causation cannot be made in the absence of longitudinal data. Findings reported elsewhere suggest a reciprocal relationship between parenting behaviors and substance use. Stice and colleagues (1995) report that deficits in parental support and control predicted adolescent substance use, and adolescent substance use was related to lower levels of parental support and control. We also did not examine the age of substance initiation in the current report. Therefore, it is possible our results may not directly generalize to studies specifically examining initiation within adolescent samples. Parental influences have also been shown to change with age (Allen et al. 2003) so our variables may differ with importance dependent on the age of initiation.

Fifth, our subjects are restricted to white males born in Virginia. However, this sample does not differ from the general population in rates of psychopathology, including illicit substance use (Kendler et al. 2000). A sixth limitation is the way our household use factors were created. As previously discussed, two separate assessment strategies were used, and no subjects completed both forms of assessment. We also suspect that the two measures examined here (household drug use and parental attitudes) reflect a broader set of familial variables that would include religious, social and community values. Finally, research also

suggests that MZ twins experience more similar environments than DZ twins, which could affect results (e.g. Martin et al. 1997). Further research in our and other data sets will hopefully clarify the structure of these familial-environmental influences.

Despite these limitations the current report provides significant evidence for the importance of family-level influences on lifetime illicit drug use. Prevention and intervention efforts should consider the importance of the parents and parental modeling when establishing programs as these influences likely combine with other factors (e.g. peer relationships) to influence lifetime substance use. This is an especially important consideration for prevention given the role of early use in later substance abuse and other externalizing behavior problems.

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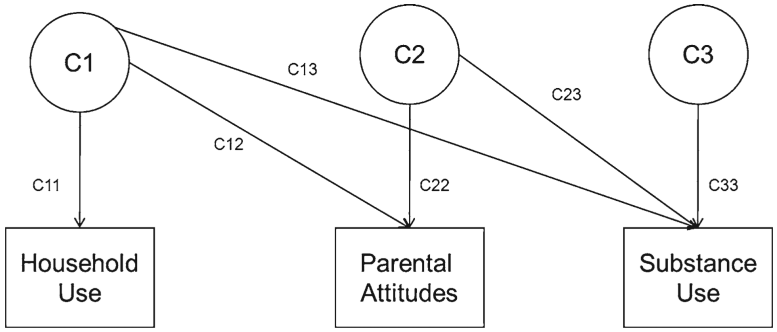

Fig. 1.

Shared environmental portion of trivariate Choleksy model of adult household substance use, parental attitudes towards substance use, and lifetime substance use. *C1* shared environmental factors common to household use, parental attitudes and substance use, *C2* shared environmental factors common to parental attitudes and substance use, *C3* shared environmental factors unique to substance use, *C11* shared environmental path for household use, *C12* shared environmental path between household use and parental attitudes, *C13* shared environmental path between household use and substance use, *C22* shared environmental path for parental attitudes, *C23* shared environmental path between parental attitudes and substance use. *C33* shared environmental path for substance use

Table 1

Frequency of lifetime substance use and mean scores for family environment variables by zygosity

	Smoking (%)	Alcohol (%)	Marijuana (%)	Cocaine (%)	Stimulant (%)	Sedative (%)	Hallucinogen (%)	Any illicit drug (%)	Parental attitudes mean (SD)	Household use mean (SD)
MZ	85	94	58	20	21	12	17	59	3.41 (2.3)	6.74 (4.0)
DZ	87	96	64	24	24	15	20	65	3.63 (2.3)	7.10 (4.1)

Frequency of lifetime substance use indicates the percentage of those respondents ever having used the substance in their life *SD* standard deviation

Table 2

Individual phenotypic correlations between lifetime substance use and family environment variables by zygosity

Substance	Parental attitudes MZ (DZ)	Household use MZ (DZ)
Smoking	0.11 (0.14)	0.21 (0.19)
Alcohol	0.33 (0.05)	0.31 (0.16)
Marijuana	0.14 (0.02)	0.30 (0.20)
Cocaine	0.09 (0.06)	0.26 (0.20)
Stimulant	0.15 (0.08)	0.29 (0.20)
Sedative	0.13 (0.05)	0.30 (0.25)
Hallucinogen	0.18 (0.08)	0.30 (0.16)
Any illicit drug	0.15 (0.02)	0.31 (0.21)

MZ monozygotic twins, *DZ* dizygotic twins

Table 3

Age-corrected cross-twin, within-trait correlations for lifetime substance use and family environment variables

	Smoking	Alcohol	Marijuana	Cocaine	Stimulant	Sedative	Hallucinogen	Any illicit drug	Parental attitudes	Household use
MZ	0.41	0.44	0.58	0.58	0.46	0.34	0.48	0.59	0.34	0.75
DZ	0.21	0.42	0.30	0.29	0.23	0.19	0.28	0.32	0.24	0.72

MZ monozygotic twins, *DZ* dizygotic twins

Table 4

Univariate biometric model fitting of parental attitudes toward substance use and household adult substance use

	a^2	95% CI	c^2	95% CI	e^2	95% CI	-2LL
Age corrected model							
Parental attitudes	22	0; 48	22	0; 43	56	50; 64	6822.7
Household use	6	0; 19	72	60; 80	22	19; 26	7639.2
Non-age corrected model							
Parental attitudes	7	0; 20	70	60; 77	22	20; 27	7948.0
Household use	3	2; 3	87	81; 89	10	8; 12	8357.3

a^2 additive genetic variance, *95% CI* 95% confidence interval, c^2 shared environmental variance, e^2 unique environmental variance, *–2LL* log likelihood. Sums may not equal 100% due to rounding error

Table 5

Genetic and environmental contributions to lifetime substance use

Substance	% Variance[a] (95% CI)			% of C variance in substance use accounted for by household use	% of C variance in substance use accounted for by parental attitudes	% of Total C variance in substance use accounted for by family environment (95% CI)
	a^2	c^2	e^2			
Smoking	62 (12; 83)	10 (0; 56)	27 (17; 40)	13	10	23 (0;100)
Alcohol	11 (0; 56)	62 (19; 82)	26 (15; 42)	5	9	14 (0;100)
Marijuana	42 (11; 75)	36 (5; 63)	22 (16; 31)	19	11	30 (2; 100)
Cocaine	47 (18; 76)	31 (6; 58)	21 (14; 31)	10	90	100 (11;100)
Stimulant	54 (22; 73)	12 (0; 40)	34 (23; 46)	25	75	100 (2; 100)
Sedative	41 (1; 69)	16 (0; 54)	42 (29; 60)	13	73	87 (0; 100)
Hallucinogen	43 (6; 80)	29 (0; 61)	28 (18; 40)	7	3	10 (0;100)
Any illicit drug	41 (10; 75)	37 (5; 64)	22 (15; 30)	27	17	44 (7; 100)

a^2 additive genetic variance for substance, c^2 shared environmental variance for substance, e^2 unique environmental variance for substance. The % of C variance columns represent the proportion of shared environmental variance in lifetime substance use that is accounted for by household substance use, parental attitudes, and the total percent accounted for by both family environmental variables. *95% CI* 95% confidence intervals for % C variance accounted for by family environment

[a]Proportion of total genetic and environmental variance for each substance from trivariate Cholesky. Sums 100% allowing for rounding error