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Genetic and Environmental Overlap Between Substance Use and Delinquency in Adolescence: An Analysis by Same-Sex Twins

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Abstract

During adolescence, many teens begin to experiment with substances and engage in delinquent behavior. The current study seeks to examine whether and to what extent genetic and environmental factors contribute to the association between substance use (i.e., marijuana and alcohol) and different forms of delinquent offending (i.e., violent and nonviolent) across males and females. Analyses were based on same-sex twins ($N = 1{,}072$) from the sibling subsample of the National Longitudinal Study of Adolescent to Adult Health (Add Health). The results revealed moderate to large genetic overlap between substance use and delinquent behavior for males. Much of the covariation between alcohol use and offending behavior for females was attributable to common environmental factors, while common genetic factors explained a large portion of the overlap between marijuana use and offending in males and females. The implications of these findings for sex differences in prevention and intervention efforts are discussed from a biosocial perspective.

Keywords

alcohol, biosocial, delinquency, genetics, marijuana, sex

During adolescence, substance use and delinquency often co-occur in a reciprocal manner, with delinquency predicting substance use and substance use increasing one's risk for engaging in delinquent behavior (D'Amico, Edelen, Miles, & Morral, 2008). Part of this reciprocal relationship may be explained by a variety of risk factors common to both behaviors. For instance, research has

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found that low self-control, exposure to deviant peers, and punitive parenting are positively associated with property crime, violent delinquency, and drug use (Alarid, Burton, & Cullen, 2000; LaGrange & Silverman, 1999). At the same time, parallel lines of research have provided evidence suggesting that individual differences across each of these phenotypic outcomes (e.g., self-control, peer affiliation) are heavily influenced by genetic factors (Beaver et al., 2009). Put differently, correlates common to both substance abuse and delinquency are heritable outcomes (Korhonen et al., 2012). This raises the possibility that genetic factors common to both outcomes may be partially responsible for the covariation between substance use and offending behavior.

Behavioral and molecular genetic studies have shown that genetic and environmental factors contribute to the co-occurrence of various forms of substance use (e.g., alcohol, cigarettes, drugs) and antisocial behaviors, including conduct disorder, aggression, externalizing behaviors, and delinquency (Cerdá, Sagdeo, Johnson, & Galea, 2010; Knopik, Heath, Bucholz, Madden, & Waldron, 2009; McAdams, Rowe, Rijsdijk, Maughan, & Eley, 2012). An examination of these relationships by sex has found mixed results, with some studies showing that genetic and environmental factors explaining comorbidity are similar for males and females (Knopik et al., 2009; Rose, Dick, Viken, Pulkkinen, & Kaprio, 2004; von der Pahlen et al., 2008), while others contend that shared genetic factors explain comorbidity among females and that comorbidity among males is best explained by shared environmental effects (Miles, van den Bree, & Pickens, 2002). In an effort to shed light on these mixed findings and to further our understanding of the co-occurrence of substance use and delinquent behaviors in adolescence, the current study examines the genetic and environmental overlap between substance use (e.g., alcohol and marijuana) and delinquency (e.g., violent and nonviolent) in same-sex twin adolescents.

Substance Use and Delinquent Involvement

Adolescence is a time when many teenagers begin to experiment with substances, including alcohol and marijuana, and start to engage in other forms of delinquent acts as well. Results from the National Surveys on Drug Use and Health (2015) suggest that approximately 2.4 million adolescents between the age of 12 and 17 (roughly 1 in 10 adolescents in the United States) report consuming alcohol and that approximately 1.8 million adolescents between the age of 12 and 17 experiment with marijuana. Findings from the Monitoring the Future Study (2016) also show that the prevalence of marijuana use within the past month increases from 5.4% in the 8th grade to 22.5% in the 12th grade (Johnston, O'Malley, Bachman, Schulenberg, & Institute for Social Research, 2016). Similar results are reported for alcohol consumption with 7.3% of 8th graders reporting consumption within the past 30 days compared to 33.2% of 12th graders (Johnston, O'Malley, Bachman, Schulenberg, & Institute for Social Research, 2016). In addition to drug and alcohol use, adolescence is a period in which teens are more likely to engage in delinquent behaviors. Studies have shown that the frequency of delinquent behaviors significantly increases from late childhood (age 10–11) to late adolescence (age 17–18; Jennings, Maldonado-Molina, & Komro, 2010; van Lier, Vitaro, Barker, Koot, & Tremblay, 2009; Wiesner & Windle, 2004).

Perhaps not surprisingly, given the concomitant increase in substance use and delinquent behaviors during adolescence, Newman and colleagues' (1996) analysis of youths from the Dunedin Multidisciplinary Health and Development Study revealed that substance abuse and conduct disorder tend to co-occur. Specifically, 43% of respondents who had a substance use disorder at age 21 also had a history of conduct disorder. Overall, studies on this topic seem to converge to demonstrate that substance use and delinquent involvement tend to co-occur, particularly in adolescence (Dembo, Pacheco, Schmeidler, Fisher, & Cooper, 1998; Felson, Savolainen, Aaltonen, & Moustgaard, 2008; White, Loeber, Stouthamer-Loeber, & Farrington, 1999). There is evidence that the co-occurrence of substance use and antisocial behavior during adolescence is the result of reciprocal

causal effects. For instance, Young, Sweeting, and West (2008) found that approximately 20% of the variation in antisocial behavior was accounted for by concurrent alcohol use and that antisocial behavior predicted subsequent increases in alcohol use.

Behavioral Genetic Research on Substance Use and Delinquent Involvement

Studies using behavioral genetic methodologies have shown that, beyond reciprocal causality, the comorbidity of substance use and antisocial behavior is also accounted for by common genetic influences. To arrive at this conclusion, behavioral genetic methods use information from families, particularly twins and siblings, to partition the variance and covariance of behaviors into genetic, shared environment, and nonshared environment components. Genetic components provide heritability estimates for the behavior or phenotype and reveal the extent to which additive and (sometimes) dominant effects of genes impact behavior. Shared environments are social experiences shared between children within the home, neighborhood, or school environment and function to promote behavioral similarities between siblings. Nonshared environments, on the other hand, are experiences and events that are unique to each child residing in the home and function to make siblings different from one another. These can include different experiences at school, dissimilar involvement in activities, individualized parental treatment, and unique friendship networks. Researchers often take a univariate genetic approach to the study of antisocial behavior and substance use by examining the genetic and environmental influences on these phenotypes separately. A bivariate genetic approach, however, allows investigators to examine whether genetic and environmental factors contribute to the co-occurrence of these two behaviors. In other words, the extent to which these two behaviors overlap as a result of common genetic and environmental factors can be estimated. Evidence that genetic factors partly account for the covariance between antisocial behavior and substance use would suggest that there is a genetic vulnerability common to both behaviors.

Research has shown that there are common genetic influences on antisocial behavior and substance use/dependence, suggesting that these behaviors share a genetic etiology (Cerdá et al., 2010; Kendler, Ohlsson, Sundquist, & Sundquist, 2015; Malone, Taylor, Marmorstein, McGue, & Iacono, 2004). That is, individuals who are genetically at risk or vulnerable for developing antisocial behavior are also at risk for developing problematic substance use behaviors. For instance, Krueger et al. (2002) found that a latent variable of externalizing behavior adequately captured the covariation between antisocial behavior, conduct disorder, alcohol dependence, drug dependence, and a lack of constraint in adolescence. Krueger et al. show a common genetic influence on substance use and antisocial behavior in finding that latent additive genetic effects accounted for 81% of the variation in externalizing behavior. Similarly, Young, Stallings, Corley, Krauter, and Hewitt's (2000) biometric factor analysis demonstrated that conduct disorder, substance experimentation, novelty seeking, and attention deficit hyperactivity disorder shared a common underlying genetic risk factor. Korhonen et al.'s (2012) analysis of adolescent twin pairs revealed that about half of the covariance between externalizing behavior and drug use was due to common additive genetic factors, with the remaining variation due to common shared environmental factors. These results align with findings from a study of adolescent twins from the United Kingdom, which revealed that common additive genetic factors explained 78% of the phenotypic covariation between marijuana use and delinquency, 82% of the association between marijuana use and aggression, 58% of the alcohol–delinquency correlation, and 47% of the covariation between alcohol use and aggression (McAdams et al., 2012). Other studies have also confirmed that common genetic factors are important in the etiology of both antisocial behavior and substance use, and that the covariation between both phenotypes may be due to common genetic sources (McGue, Iacono, & Krueger, 2006;

Pickens, Svikis, McGue, & LaBuda, 1995; Slutske et al., 1998; except see Rose et al., 2004; True et al., 1999).

What should not be overlooked, however, is that involvement in antisocial and delinquent behavior is not equally distributed among members of the general population. The distribution of antisocial behavior and substance use varies across sex. This suggests that the magnitude of genetic and environmental contributions to the comorbidity between substance use and antisocial behavior may also vary across males and females. Given that males often report greater involvement in antisocial behavior and substance use across the life course, some studies have investigated whether sex moderates the effects of genetic and environmental influence on antisocial behavior and substance use. Specifically, researchers have examined whether there are sex differences in the magnitude of the heritability coefficients for antisocial behavior and substance use, the types of genetic and environmental factors that contribute to both phenotypes, and the genetic and environmental contributions to the phenotypic covariation between antisocial behavior and substance use.

A review of the behavioral genetic literature on substance use and antisocial behaviors reveals four key findings. First, a number of behavioral genetic studies have shown that the size of the heritability coefficient for delinquency, aggression, conduct disorder, externalizing behavior, and antisocial behavior is statistically the same for males and females, with genetic factors explaining between 40% and 60% of the variance in these phenotypes (Arseneault et al., 2003; Button et al., 2006; Gelhorn et al., 2005; Maes, Silberg, Neale, & Eaves, 2007; Miles et al., 2002; Rhee & Waldman, 2002; Van Hulle, Rodgers, D'Onofrio, Waldman, & Lahey, 2007). Second, studies have found that the same genetic and environmental factors are relevant to the etiology of antisocial behavior for males and females (Boisvert, Vaske, Wright, & Knopik, 2012; Jacobson, Prescott, & Kendler, 2002; Van Hulle et al., 2007; von der Pahlen et al., 2008). However, other studies have reported sex differences in environmental and genetic influences on substance use and abuse. Specifically, studies have found that environmental factors (especially nonshared environmental influences) play a larger role in the etiology of alcohol and drug use/dependence for females rather than for males (Hicks et al., 2007; Knopik et al., 2009; Maes et al., 2016; McGue et al., 2006; van den Bree, Johnson, Neale, & Pickens, 1998).

Lastly, several behavioral genetic studies have reported that the types of genetic and environmental contributions to the comorbidity between antisocial behavior and substance use do not vary across sex (Button et al., 2006; Knopik et al., 2009; Rose et al., 2004; von der Pahlen et al., 2008). However, a few studies have found significant sex differences in the genetic and environmental effect on the comorbidity between antisocial behavior and substance use. For instance, some studies have reported that only environmental factors account for the phenotypic correlation between drug use and antisocial behavior in both males and females (Knopik et al., 2009; Rose et al., 2004), while others have reported that genetic, shared environmental, and nonshared environmental influences explain the overlap between conduct disorder and drug use in both males and females (Button et al., 2006). Other work has found that genetic factors explain the comorbidity of adolescent antisocial behavior and substance use for females, while the comorbidity for males was a function of shared environmental effects (Miles et al., 2002).

Purpose of the Current Study

While studies have explored the magnitude of genetic and environmental influence on the association between substance use and antisocial behavior in general, to our knowledge, no study has examined the genetic and environmental effects on the association between different forms of substance use and different forms of offending across males and females. As such, the current study aims to add to the existing body of literature on substance use and offending by examining the genetic and environmental contributions to the co-occurrence between substance use, specifically

alcohol use and lifetime marijuana use, and nonviolent and violent offending in males and females. Evidence that environmental (or genetic) factors are relevant to the co-occurrence of these phenotypes for both sexes suggests that a single intervention could influence a broad array of maladaptive behaviors for a variety of individuals and be more cost-effective than developing interventions that are targeted to only one phenotype and one sex.

Method

Sample

Data for the current study are from the National Longitudinal Study of Adolescent to Adult Health (Add Health), which has been described in detail elsewhere (see Harris, 2009; Kelley, Peterson, & Peterson, 1997). Briefly, Add Health is a nationally representative longitudinal study of American youth. To date, four waves of data collection have been completed, spanning a time period of 14 years. The first wave, which began in September 1994, occurred in the classroom ($N = 132$ schools) and over 90,000 students in Grades 7 through 12 participated. From this sample, a subsample of adolescents ($N = 20{,}745$) were randomly chosen to complete an in-home interview. A distinctive feature of the Add Health is that the researchers oversampled for siblings at Wave I. Specifically, study respondents were asked whether they had a sibling residing within the same household who was also enrolled in Grades 7 through 12. These identified sibling pairs, including twins, full-siblings, half-siblings, cousins, and nonrelated siblings, were then included in the Wave I in-home interview. The second wave of data collection occurred approximately 1 year later (1995–1996) when most participants were still adolescents and enrolled in Grades 8 through 12 ($N = 14{,}738$). Six years later (2001–2002), the third wave of data collection included over 15,000 participants who were young adults between the ages of 18 and 26. The most recent wave of data was collected in 2007–2008, when participants were between the ages of 24 and 34 ($N = 15{,}701$).

The current study included a subsample of same-sex twin pairs from Wave I when most respondents were in early adolescence. This resulted in a final analytical sample of 288 monozygotic (MZ) males, 290 MZ females, 262 dizygotic (DZ) males, and 232 DZ females totaling 536 twin pairs.[1]

Measures

Alcohol use. Alcohol use was measured by asking respondents the following question:

> Think of all the times you have had a drink during the past 12 months. How many drinks did you usually have each time? (A "drink" is a glass of wine, a can of beer, a wine cooler, a shot glass of liquor, or a mixed drink.).

Responses to this question ranged from 0 to 55, with few respondents (2%) reporting more than 12 drinks per drinking episode. As such, those who reported 12 or more drinks were given a score of 12. Over half of the respondents (57%) indicated that they did not consume any alcohol within the past 12 months. Due to the positively skewed distribution of the variable, the measure was log transformed ($\log(x + 1)$) prior to being included in the analyses. The descriptive statistics for this measure is provided in Table 1. As shown, males reported, on average, significantly higher levels of alcohol use compared to females ($t = 4.88$, $p < .001$).

Marijuana use. Marijuana use was measured by asking participants the following question: "During your life, how many times did you use marijuana?" Responses ranged from 0 to 900 times. A small number of respondents (3%) indicated using marijuana more than 50 times in their lifetime. As such, these respondents received a value of 51 for the measure of marijuana use. Most respondents (74%)

Table 1. Descriptive Statistics for Substance Use and Offending.

Variables	Males Mean (*SD*)	Females Mean (*SD*)	*t* test
Alcohol use	2.32 (3.56)	1.40 (2.48)	4.88**
Marijuana use	5.03 (13.00)	2.71 (8.76)	3.39**
Nonviolent offending	1.91 (3.24)	1.08 (2.23)	4.85**
Violent offending	1.42 (.77)	.77 (1.60)	5.22**

Note. Descriptive statistics for measures prior to log transformation.
**$p < .001$.

indicated never having used marijuana in their lifetime. Because this measure was also positively skewed, it was log transformed before being included in the analysis. Table 1 summarizes the descriptive statistics for this measure. Males reported significantly higher levels of lifetime marijuana use compared to females ($t = 3.39$, $p < .001$).

Nonviolent offending. Nonviolent offending was assessed by asking respondents to indicate how often, in the past 12 months, they had participated in eight different forms of nonviolent delinquent behavior, such as damaging property and stealing (see Appendix A for a complete list of items). The response categories for all items ranged from 0 (*never*) to 3 (*five or more times*). Item responses were then summed together to create a nonviolent offending scale, where higher scores indicate a greater involvement in nonviolent delinquent activities (Cronbach's $\alpha = .77$). About 59% of the respondents reported having never engaged in any form of nonviolent offending behavior within the past year. As presented in Table 1, males reported, on average, significantly higher levels of nonviolent offending compared to females ($t = 4.85$, $p < .001$).

Violent offending. Similar to the measure of nonviolent offending, participants self-reported their involvement in violent offending. Specifically, respondents indicated how often, in the past year, they had engaged in six violent behaviors, such as shooting or stabbing someone or pulling a knife on someone (see Appendix A for a complete list of items along with their response categories). The responses to these six questions were summed together to create a measure of violent offending, with higher scores indicating greater levels of violent delinquent involvement (Cronbach's $\alpha = .75$). About 58% of the participants indicated that they had not engaged in any form of violent offending behavior within the previous year. Table 1 shows that males reported, on average, significantly higher levels of violent offending compared to females ($t = 5.22$, $p < .001$).

Plan of Analysis

The analysis for the current study were conducted in three stages. First, cross-twin and cross-twin/cross-trait correlations were calculated by sex to preliminarily examine whether genetic factors influenced substance use and offending independently as well as the covariation between the two. Cross-twin correlations were calculated[2] separately for all male and female MZ and DZ twin pairs for each measure of substance use and offending. If the cross-twin correlation for MZ twin pairs was greater than the cross-twin correlation for DZ twin pairs, this suggests that genes were influencing the behavior. Second, the cross-twin/cross-trait correlations were also calculated[3] separately for all male and female MZ and DZ twin pairs. If the cross-twin/cross-trait correlation for MZ twin pairs was greater than that of DZ twin pairs, this suggests that genes were influencing the covariation between the two variables (Neale & Cardon, 1992).

Table 2. Phenotypic Correlations Between Substance Use and Offending.

Variables	Males	Females
Alcohol use and nonviolent offending	.30**	.30**
Alcohol use and violent offending	.20**	.07†
Marijuana use and nonviolent offending	.40**	.34**
Marijuana use and violent offending	.18**	.15*

*$p < .01$. **$p < .001$. †$p < .05$.

Table 3. Cross-Twin Correlations for MZ and DZ Twin Pairs.

Variables		MZ	DZ
Alcohol use	Males	.65**	.34**
	Females	.57**	.51**
Marijuana use	Males	.63**	.28*
	Females	.69**	.43**
Nonviolent offending	Males	.45**	.30*
	Females	.47**	.45**
Violent offending	Males	.52**	.30*
	Females	.47**	.40**

Note. MZ = monozygotic; DZ = dizygotic.
*$p < .01$. **$p < .001$.

Third, univariate and bivariate genetic analyses were performed in the statistical program Mx[4] to estimate the degree to which additive genetic (A), shared environmental (C), and nonshared environmental (E) factors explained variance in substance use and offending as well as the covariance between the two. The estimates used to calculate the proportion of the covariance between substance use and offending that were due to the same genetic and environmental factor were derived from the best fitting models (Neale & Cardon, 1992). Determining the best fitting model was accomplished by comparing the fit statistics of submodels (i.e., AE or CE[5]) to those of the full saturated ACE model. A submodel was considered a better fitting model if there was a nonsignificant difference in χ^2 statistic and a lower Akaike Information Criterion (AIC) value compared to the ACE model. The estimates from the best fitting model were then used to calculate the proportion of the phenotypic correlation (r_p) between the two variables that were due to common genetic, shared environmental, and nonshared environmental factors (Neale & Cardon, 1992).

Results

Table 2 presents the phenotypic correlations between the measures of substance use and offending for males and females. Three noteworthy findings are derived from these analyses. First, for both males and females, the relationship between substance use (alcohol and marijuana) and nonviolent offending (.30–.40) appears to be stronger than the relationship between substance use and violent offending (.07–.18). Second, the strongest relationship appears to be the association between marijuana use and nonviolent offending for both males (.40) and females (.34). Lastly, the weakest relationship appears to be between alcohol use and violent offending among females (.07).

Table 3 presents the cross-twin correlations for male and female MZ and DZ twin pairs. The results show that, in general, MZ twin pair correlations (MZ*r*) are higher than DZ twin pair correlations (DZ*r*) for alcohol use (MZ*r* = .57–.65, DZ*r* = .34–.51), marijuana use (MZ*r* = .63–.69,

Table 4. Genetic Analysis of Substance Use and Offending.

	Fit Statistics				Trait 1			Trait 2		
Variables	−2lnl	*df*	AIC	*p*	A	C	E	A	C	E
					Alcohol Use			Nonviolent Offending		
Males	730.45	1,071	−1,411.55	.81	.67	—	.33	.45	—	.55
					(.57–.74)	—	(.26–.43)	(.32–.55)	—	(.45–.68)
Females	324.88	1,016	−1,707.12	.97	—	.55	.45	—	.46	.54
					—	(.45–.63)	(.37–.55)	—	(.35–.55)	(.45–.65)
					Alcohol Use			Violent Offending		
Males	585.50	1,073	−1,560.50	.51	.66	—	.34	.50	—	.50
					(.57–.74)	—	(.26–.43)	(.39–.60)	—	(.40–.62)
Females	180.01	1,015	−1,849.99	.50	—	.55	.45	—	.44	.56
					—	(.45–.63)	(.37–.55)	—	(.34–.54)	(.46–.66)
					Marijuana Use			Nonviolent Offending		
Males	1,019.95	1,062	−1,104.05	.14	.65	—	.35	.44	—	.56
					(.55–.73)	—	(.27–.45)	(.32–.55)	—	(.45–.68)
Females	553.86	1,005	−1,456.15	.62	.74	—	.26	.51	—	.49
					(.65–.80)	—	(.20–.35)	(.40–.61)	—	(.39–.60)
					Marijuana Use			Violent Offending		
Males and females	1,602.23	970	−1,035.08	.83	.70	—	.30	.41	—	.59
					(.59–.82)	—	(.18–.41)	(.31–.59)	—	(.41–.69)

Note. Best fitting models are presented. AIC = Akaike information criterion; A = additive genetic; C = shared environmental; E = nonshared environmental.

DZ*r* = .28–.43), nonviolent offending (MZ*r* = .45–.47, DZ*r* = .30–.45), and violent offending (MZ*r* = .47–.52, DZ*r* = .30–.40). This suggests that additive genetic factors are influencing the measures of interest. In addition, the results from the cross-twin/cross-trait correlations (available upon request) show a general pattern of higher MZ correlations compared to DZ correlations, particularly for the male subsample. This suggests that, at least for males, similar genetic factors may be operating on both behaviors. For females, though, it appears that environmental factors may better explain the co-occurrence between the behaviors of interest. These preliminary findings indicate that both genetic and environmental factors may be contributing to both the variance in substance use and offending as well as their covariance. As such, bivariate genetic analyses were conducted to more accurately estimate the extent to which the same genetic, shared environmental, and nonshared environmental factors can explain the covariation between substance use and offending in early adolescence across males and females.

Table 4 presents the results from the best fitting models from the genetic analyses of substance use and delinquency by sex.[6] To determine whether it was appropriate to examine the bivariate association between substance use and delinquency separately for males and females, a series of preliminary analyses were estimated. First, we estimated a series of bivariate models to determine whether there were sex differences in the structure of genetic and environmental effects on substance use and offending. For each bivariate model, an additional model was estimated that allowed all parameters to vary between males and females, but the underlying influences were the same across the sex (i.e., a sex-limitation model). Model fit for the sex-limitation model was then compared to a scalar sex-differences model where all parameters were constrained to be equal between males and females, but the total variation/covariation for males was allowed to be the same as the total variation/covariation for females. The results indicated that the quantitative sex-differences model for the alcohol use–nonviolent offending, alcohol use–violent offending, and marijuana

Table 5. Percentage of Covariance Between Substance Use and Offending Due to Common Additive Genetic (A), Shared Environmental (C), and Nonshared Environmental (E) Factors.

Variables	A	C	E
Alcohol use and nonviolent offending			
Males	40%	—	60%
Females	—	83%	17%
Alcohol use and violent offending			
Males	35%	—	65%
Females	—	29%	71%
Marijuana use and nonviolent offending			
Males	58%	—	42%
Females	84%	—	16%
Marijuana use and violent offending			
Males and females	71%	—	29%

use–nonviolent offending associations fit significantly better than the scalar sex-differences model, suggesting that a quantitative sex difference in the magnitudes of genetic and environmental influences on the association supported the decision to separately analyze male and female twins. However, the results revealed that the scalar sex-differences model was a significantly better fit than the quantitative sex-differences model for marijuana use and violent offending, suggesting that there was not a significant difference in the magnitude of genetic and environmental effects on this association. As such, a Cholesky model with both male and female twins combined was estimated to examine the association between marijuana use and violent offending.

As can be seen in Table 4, an AE submodel was the best fitting model for males across all estimated bivariate models for substance use and offending. This included models for alcohol use and nonviolent offending ($\Delta\chi^2 = .98$, $\Delta AIC = -5.02$, $p = .81$), alcohol use and violent offending ($\Delta\chi^2 = 2.31$, $\Delta AIC = -3.70$, $p = .51$), and marijuana use and nonviolent offending ($\Delta\chi^2 = 5.51$, $\Delta AIC = -.49$, $p = .14$). For females, a CE model was the best fitting model for alcohol use and both nonviolent ($\Delta\chi^2 = .23$, $\Delta AIC = -5.77$, $p = .97$) and violent offending ($\Delta\chi^2 = 2.34$, $\Delta AIC = -3.66$, $p = .50$). With regard to marijuana use for females, the AE submodel was the best fitting model for nonviolent offending ($\Delta\chi^2 = 1.78$, $\Delta AIC = -4.22$, $p = .62$). The best fitting model for marijuana use and violent offending for males and females was also an AE model ($\Delta\chi^2 = 2.81$, $\Delta AIC = -7.39$, $p = .83$).

Using the information presented in Table 4, the correlation between substance use and offending (see Table 2) was decomposed into the percent that is due to common genetic, shared environmental, and nonshared environmental factors. The results from this segment of the analysis are presented in Table 5. As an example, the correlation between marijuana use and nonviolent offending for males is .40, the univariate genetic estimate for marijuana use and nonviolent offending in males is .65 and .44, respectively, and the genetic correlation between the two variables for males is .43.[7] The proportion of the correlation between the two variables due to common genetic influences is then calculated ($\sqrt{.65} \times \sqrt{.44} \times .43)/.40$), revealing that 58% of the correlation between marijuana use and nonviolent offending in males was accounted for by common genetic characteristics contributing to both behaviors. These calculations are then repeated with the estimates of the nonshared environment.[8]

Several noteworthy findings are presented in Table 5. For males, a moderate to large amount of genetic overlap existed between substance use and offending, particularly for marijuana use and nonviolent offending (58%). For females, much of the covariation between alcohol use and nonviolent offending was attributable to common shared environments (83%), while common

nonshared environmental factors appeared to explain much of the overlap between alcohol and violent offending (71%) in females. This is particularly interesting, given that there is moderate genetic overlap between alcohol and offending in males, but not for females. With regard to marijuana use for males and females, common genetic factors help to explain a large portion of the overlap between marijuana use and both nonviolent and violent offending.[9]

Discussion

The current study examined the relationship between substance use and offending across male and female adolescents using behavioral genetic methods. Not surprisingly, males reported significantly more substance use and criminal offending compared to females. Given the limited amount of behavioral genetic research on substance use and different forms of criminal behavior across sex, the current study set out to estimate the magnitude of genetic and environmental overlap between alcohol use, marijuana use, nonviolent offending, and violent offending for male and female adolescent twins. The results highlight the importance of examining the association between alcohol use and delinquency for males and females, separately. For instance, results from the current study revealed a moderate amount of genetic overlap between substance use (alcohol and marijuana) and delinquency (violent and nonviolent) for males, while much of the covariation between alcohol use and delinquency was attributed to common environmental factors for females (see also Knopik et al., 2009; Rose et al., 2004). Moreover, common additive genetic influences explained a large portion of the overlap between marijuana use and offending for males and females. Overall, the reported findings suggest that different degrees of biological and environmental influence operate to explain the association between substance use and delinquency during adolescence for males and females.

Variation in genetic and environmental influences on the covariation between substance abuse and antisocial behavior are not surprising given gender differences in substance use. Across both alcohol use and marijuana use, males show increased rates of abuse and dependence (Substance Abuse and Mental Health Services Administration, 2016). Interestingly, the results from the current study indicate that there are both consistencies and differences in the etiology of substance use across gender. The results from the bivariate models considering the covariance between marijuana and offending are relatively consistent across males and females. These models show that for both males and females, covariance between substance use and offending is explained by additive genetic factors and nonshared environmental factors, while shared environmental factors do not contribute to covariance between marijuana use and offending for either males or females. Within these models, nonshared environmental influences do appear particularly strong for the covariance between marijuana use and nonviolent offending among males. Stronger nonshared environmental influences on the covariance between marijuana use and nonviolent offending among males may be explained by the increased salience of delinquent peer influence for both marijuana use (Andrews, Tildesley, Hops, & Li, 2002) and delinquency (Piquero, Gover, MacDonald, & Piquero, 2005).

Gender differences in the etiology of substance use and offending are clearly indicated from the results of models partitioning the covariance of alcohol use and offending. These models show that additive genetic factors are not as important for the explanation of the covariation between alcohol use and offending for females, while both shared and nonshared environmental factors contribute to the covariation between alcohol use and offending. In contrast, additive genetic factors and nonshared environmental factors are important for the explanation of the covariation between alcohol use and offending for males, while shared environmental factors are not. Heightened environmental influences among females may reflect increased monitoring and disapproval of alcohol use and offending for females. Previous research has found that females tend to report higher levels of perceived parental control (Stattin & Kerr, 2000) and increased parental monitoring (Svensson, 2003) during adolescence. Taken in the context of increased social and parental disapproval of

alcohol use among females (Brady & Randall, 1999; Kelly et al., 2011; Nolen-Hoeksema, 2004), this suggests that gender differences in the influence of environmental factors on the covariation between alcohol use and offending may work through differences in parental supervision and disapproval of alcohol use between males and females. It is also possible that variation across gender in the modeling of alcohol use behaviors contributes to the salience of environmental factors for females.

Understanding the causes of the co-occurrence of substance use and delinquency in adolescence can assist in the development of effective treatment strategies targeted at each behavior (either alone or together) for males and females. In fact, research has shown that sex differences can have an impact on the effectiveness of substance abuse and delinquency prevention and treatment programs (Kaminer, 1994; Schinke, Botvin, & Orlandi, 1991; Semlitz & Gold, 1986). Our results suggest that intervention efforts would benefit from focusing on individual characteristics and environmental influences when attempting to target modifiable risk factors associated with marijuana use and delinquent behavior in adolescent males and females. For alcohol use and delinquency, a more focused effort on environmental factors may prove to be most effective at reducing both behaviors in females. Research has suggested, for example, that environmental factors which are most influential for females, relative to males, include relationships with peers (e.g., peer pressure) and romantic partners (Cauffman, Farrugia, & Goldweber, 2008; Giletta et al., 2012). As such, interventions which focus on how girls navigate their social environments (such as Covington's *Voices* program) may be more effective than programs that ignore the contexts in which females make decisions.

Based on contemporary evidence from offender treatment research, targeting individual and environmental factors to reduce both substance use and delinquent involvement has shown some promise (Cuijpers, 2002; Ennett et al., 2003; Farrington & Welsh, 2003; Landenberger & Lipsey, 2005; Lipsey, 1992; Pearson, Lipton, Cleland, & Yee, 2002). Environmental risk factors that are dynamic in nature, such as parenting behaviors and antisocial peers, have been targeted to help reduce substance use and/or delinquent behaviors in adolescents (Mason, Kosterman, Hawkins, Haggerty, & Spoth, 2003). However, what is important to keep in mind is that these environmental risk factors are also heritable. Evidence from a review of 55 studies examining several different measures of social environments (e.g., parenting, social support, marital quality, and peer interactions) revealed an overall weighted heritability of .27, meaning that genetic characteristics accounted for about 27% of the variance in exposure to different environments (Kendler & Baker, 2007).

The influence of genetic factors on environmental measures can best be explained through gene–environment correlations (i.e., passive, evocative, and active; Scarr & McCartney, 1983). For example, the relationship between parenting behavior and child behavior may operate through passive gene–environment correlations where the parent(s) provide both the genetic material and the rearing environment to the child. Recent research has found a strong degree of genetic overlap between exposure to family conflict and future antisocial behavior in children (Connolly & Beaver, 2015). As such, intervention efforts aimed at reducing youth substance use and/or offending behavior should also consider the parent(s), as it is possible that they are also at an increased level genetic risk (or susceptibility) for antisocial behavior and are also providing a toxic environment that is conducive to the expression of antisocial behaviors among their children. Indeed, intervention programs that target the family unit as an agent for change have shown moderate success in reducing offspring behavioral problems (Henggeler, Melton, Brondino, Scherer, & Hanley, 1997; Sawyer & Borduin, 2011).

With regard to delinquent peer association, it has been suggested that individuals actively chose their peers based on their own genetic predispositions (i.e., active gene–environment correlations). Individuals who are delinquent will be more likely to seek out others like themselves, making the delinquent peer association also a heritable characteristic (Beaver et al., 2009; Boardman,

Domingue, & Fletcher, 2012; Fowler, Settle, & Christakis, 2012; Wright, Beaver, DeLisi, & Vaughn, 2008). Intervention and prevention programs should consider this body of research, as the risk of delinquent peer association seems to point back to the individual. Programs that simply focus on severing ties with delinquent peers may not be as effective at reducing problem behaviors. Taken together, the intertwined relationship between genes and the environment seems to suggest that changes to individual characteristics (and perhaps the characteristics of immediate family members in the home) may have the added benefit of changing environmental risks for substance use and delinquent behavior.

Antisocial behaviors and substance use may also co-occur because they are both manifestations of a common latent variable, which is also highly heritable (Krueger et al., 2002). For example, behavioral disinhibition (what many refer to as low self-control in criminology) may underlie a wide range of behavioral problems in adolescents. Studies have shown a moderate amount of genetic overlap between low self-control and substance use (Boisvert, Boutwell, Barnes, & Vaske, 2013) as well as low self-control and delinquency (Boisvert, Wright, Knopik, & Vaske, 2012). Future research should extend these studies to examine whether common genetic influences overlap between behavioral disinhibition/self-control, substance use, and offending behavior to better understand the underlying mechanisms at play in these robust relationships. From a policy perspective, findings flowing from this body of future research may help to explain why intervention efforts that focus on individual characteristics, such as cognitive behavioral therapies, tend to be effective at reducing problem behavior (Wilson, Gottfredson, & Najaka, 2001). More specifically, programs that focus on improving heritable constructs, such as low self-control and neuropsychological deficits, might be most effective at decreasing antisocial behaviors, including substance use and offending behaviors (Diamond, Barnett, Thomas, & Munro, 2007; Young, Chick, & Gudjonsson, 2010).

Limitations

There are at least three noteworthy limitations to the current study. First, related to the measures of substance use, we included single items that tap the amount of consumption per drinking episode as well as lifetime frequency of marijuana use. Yet, substance use and abuse are complex behaviors and it is possible that these results may vary in replication studies based on how substance use/abuse is conceptualized and operationalized. For instance, the current study did not differentiate between use and abuse, nor did it include the age of onset of substance use, individual and familial substance use history, or the inclusion of other legal and illegal drug abuse (e.g., crystal meth, prescription drugs, cocaine). There is no consensus in the literature, however, on how best to measure substance use and abuse, which may contribute to the inconsistencies in the results reported across studies (Knopik et al., 2009; McAdams et al., 2012; Miles et al., 2002; Rose et al., 2004; von der Pahlen et al., 2008).

Second, the current study, along with most behavioral genetic models, assumes that the genetic and environmental effects on substance use and offending behaviors act independently from one another. As mentioned, however, it is more likely that genetic and environmental effects are intertwined. Referred to as gene–environment interactions, the influence of genetic factors on substance use and delinquency may be moderated by the environment. For example, studies have shown that the heritability of alcohol use is higher in urban areas compared to rural areas (Rose, Dick, Viken, & Kaprio, 2001), environments with higher alcohol sales (Dick, Rose, Viken, Kaprio, & Koskenvuo, 2001), and homes where parents drink alcohol (Cleveland & Wiebe, 2003). These environmental conditions may "trigger" a genetic liability to substance use. Conversely, other environmental conditions have been shown to buffer the genetic liability to substance use, such as marriage (Heath, Jardine, & Martin, 1989) and religious upbringing (Koopmans, Slutske, van Baal, & Boomsma, 1999). Research has also shown that heritability estimates for serious and violent delinquency tend to increase when exposure to risk factors, such as delinquent peer association, neuropsychological

deficits, alcohol consumption, residing in a broken home, and having weak school attachments increase (Beaver, 2011). As such, it is likely that gene–environment interactions are operating on our measures of interest, but our behavioral genetic models did not estimate their specific effects. Rather, the effects of gene–shared and gene–nonshared environmental interactions were subsumed in our reported genetic and nonshared environmental estimates, respectively (Heath et al., 2002).

Lastly, twin studies have been criticized more generally with regard to (1) the generalizability of the results derived from them back to the population and (2) the violation of the equal environment assumption (EEA; i.e., MZ twins are treated more similarly compared to DZ twins). With regard to the first criticism, one of the main arguments leveled by critics is that twins possess unique characteristics, due to their shared prenatal and postnatal environments, that singletons may not, which could later impact their behaviors. For example, twins are at a higher risk for premature births, resulting in lower birth weight, which has been found to be a predictor of later criminal involvement (Tibbetts & Piquero, 1999). Research by Barnes and Boutwell (2013), however, addressed the twin-singleton generalizability issue using the Add Health data specifically. Their results showed that few significant differences emerged across key variables of criminological interest, such as delinquency, when comparing twins and singletons. With regard to the second criticism, Barnes and colleagues (2014) assessed how violating the EEA, at varying degrees, would impact heritability and shared environmental estimates. Results from a series of simulation models aligned with a long line of behavioral genetic research showing that even when the EEA is violated, it has a minimal impact on generated heritability and environmental estimates. While the results from this study, and others, provides further supporting evidence for the robustness and reliability of the twin-based design, it is recommended that future studies replicate these analyses using a larger number of twin pairs, given the moderately sized confidence intervals reported within the current study.

Conclusion

In conclusion, our study contributes to prior research on the co-occurrence of substance use and antisocial behavior. While the findings from this body of literature are mixed, our results point to a need to disaggregate measures of adolescent delinquency by seriousness of offense and sex. This suggests that a "one-size-fits-all" strategy for prevention programming for adolescent substance use and delinquency may not yield much in the way of effective outcomes when applied indiscriminately to both males and females. What may yield larger dividends is a targeted approach for males and females guided by the results of genetically sensitive research designs. By using analytical strategies, such as the ones employed in the current study, future research may support the development of individually tailored intervention/prevention strategies that are likely to produce the most positive outcomes.

Appendix A

Nonviolent Criminal Behavior

In the past 12 months, how often did you:

1. paint graffiti or signs on someone else's property or in a public place,
2. deliberately damage property that didn't belong to you,
3. take something from a store without paying for it,
4. drive a car without its owner's permission,
5. steal something worth more than US$50,
6. go into a house or building to steal something,

7. sell marijuana or other drugs, and
8. steal something worth less than US$50.

Response Categories: 0 = *never*, 1 = *one or two times*, 2 = *three or four times*, 3 = *five or more times*

Violent Criminal Behavior

In the past 12 months, how often did you:

1. get into a serious physical fight,[a]
2. hurt someone badly enough to need bandages or care from a doctor or nurse,[a]
3. use or threaten to use a weapon to get something from someone,[a]
4. take part in a fight where a group of your friends were against another group,[a]
5. pull a knife or a gun on someone, and[b]
6. you shot or stabbed someone.[b]

Response Categories: [a]0 = *never*, 1 = *one or two times*, 2 = *three or four times*, 3 = *five or more times*. [b]0 = *never*, 1 = *once*, 2 = *more than once*.

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Notes

1. Twins with unknown zygosity were not included in the analyses.
2. A cross-twin correlation is calculated by correlating the score of one twin to that of his or her co-twin. For example, correlating the nonviolent offending scores for twin 1s with the nonviolent offending scores of twin 2s.
3. A cross-twin/cross-trait correlation was calculated by correlating the score of one twin on one behavior to that of his or her co-twins' score on a second behavior (and vice versa). For example, correlating the marijuana use scores of twin 1s with the nonviolent offending scores of twin 2s and correlating the marijuana use scores of twin 2s with the nonviolent offending scores of twin 1s.

4. Analyses were also replicated using Mplus and were virtually identical to those estimated in Mx (and are available upon request).
5. Estimates of E cannot be set to zero in a submodel as it contains measurement error.
6. Results from all models fitting analyses are available upon request.
7. All estimates for genetic, shared environmental, and nonshared environmental correlations are available upon request. It is important to note that the confidence interval for the environmental correlations between alcohol use and violent offending in females spanned zero. Future research should use larger samples of female twin pairs to better pinpoint these estimates.
8. Since AE is the best fitting model for substance use and nonviolent offending in males, the C parameters are set to zero.
9. Bivariate models were also run using Mplus Version 8 (Muthén & Muthén, 1998–2012) to check the robustness of the reported findings. Parameter estimates generated in Mplus were almost identical to parameter estimates generated in Mx. Results from all estimated models from analyses using M*plus* and Mx are available upon request.

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