Modeling the genetic and environmental association between peer group deviance and cannabis use in male twins

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ABSTRACT

Background Peer group deviance (PGD) is linked strongly to liability to drug use, including cannabis. Our aim was to model the genetic and environmental association, including direction of causation, between PGD and cannabis use (CU). **Method** Results were based on 1736 to 1765 adult males from the Mid-Atlantic Twin Registry with complete CU and PGD data measured retrospectively at three time-intervals between 15 and 25 years using a life-history calendar. **Results** At all ages, multivariate modeling showed that familial aggregation in PGD was explained by a combination of additive genetic and shared environmental effects. Moreover, the significant PGD–CU association was best explained by a CU→PGD causal model in which large portions of the additive genetic (50–78%) and shared environmental variance (25–73%) in PGD were explained by CU. **Conclusions** Until recently PGD was assumed to be an environmental, upstream risk factor for CU. Our data are not consistent with this hypothesis. Rather, they suggest that the liability to affiliate with deviant peers is explained more clearly by a combination of genetic and environmental factors that are indexed by CU which sits as a 'risk indicator' in the causal pathway between genetic and environmental risks and the expression of PGD. This is consistent with a process of social selection by which the genetic and environmental risks in CU largely drive the propensity to affiliate with deviant peers.

Keywords Cannabis, drugs, genes, peers, risks.

INTRODUCTION

A substantial body of literature has linked peer group deviance (PGD) and liability to drug use [1–12]. In a meta-analysis of 2700 papers, Allen and colleagues [11] found that increasing PGD predicts drug use in general ($r = 0.30$) and cannabis use (CU) even more strongly ($r = 0.38$).

Although most reports do not test alternate causal models, the general consensus is that peers influence the risk of drug use [13]. However, among those who have explored competing models, Farrell and colleagues found that associations between peer deviance and drug use are better explained by a reciprocal interaction [14]. An alternative explanation is that the association is the result of correlated liabilities which increases both the risk of deviant peer affiliation and drug use. These might be environmental risks, such as low parental monitoring, or biological, such as a prefrontal cortex dysfunction leading to behavioral disinhibition [15–17].

Although PGD as a risk factor has been considered typically 'environmental', a number of behavior genetic studies have revealed that variation in PGD is attributable to a combination of environmental and genetic factors [18–26]. Unfortunately, the relative contribution of genes and environment varies across studies, due perhaps to variations in measurement, age of sample, study design and low statistical power. Recently, Kendler and colleagues [27], using a large population-based sample of male twins, showed that genetic effects on PGD increase steadily from ~30% to ~50% between the ages of 8 and 25 years, while shared environmental influences decline. This suggests that as adolescents mature and create their own social worlds genetic factors become

increasingly important in peer affiliation, while common or shared environmental become progressively less influential.

More is known about the etiology of drug use. For instance, a number of twin studies support the hypothesis that both genetic and environmental effects generate variation in drug use, abuse and dependence for a variety of licit and illicit substances, including cannabis [28–44]. Our knowledge of the etiological mechanisms influencing the transition from initiation to regular use and abuse is also improving [29,42,44]. Recently, Gillespie and colleagues [45] have estimated that nearly half the total genetic variation in the symptoms of cannabis abuse can be explained by genetic effects underpinning variation in the liability to initiate cannabis.

The challenge for twin studies now is to move beyond estimating heritabilities and begin to identify the causal pathways to drug use and other complex behaviors. Rather than being entirely attributable to 'within the skin' genetic effects (via for example, brain neurochemical systems), a proportion of the observed genetic risk to CU may be mediated by 'outside the skin' genetic pathways via active genotype by environment correlations. Such correlations arise when individuals create or evoke environments as a result of their genetically influenced dispositions [46]. In particular, individuals at risk for drug problems may seek out and help to create deviant social environments which, in turn, exacerbate the risk of substance use.

To date, no twin studies have examined the nature of the association between PGD and quantitative measures of CU. Our prediction is that if genetic risk for CU is mediated through self-selection into deviant peer groups then we would expect to see significant genetic contributions in the PGD–CU association. In order to test this hypothesis, as well as determine the nature of the causal relationship, we model the PGD–CU association using data from three epochs between the ages of 15 and 25 years. The key issues are: (i) to what extent is the covariance between PGD and CU explained by shared genetic and environmental liabilities; (ii) how does the relative contribution of these shared genetic and environmental liabilities change over time; and (iii) what is the direction of causation between PGD and CU?

METHODS

Subjects

As part of on ongoing study of adult male twins from the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (VATSPSUD), this report is based on data collected from a second and third wave of interviews between 1994 and 2004. The VATSPSUD is described in detail elsewhere [47]. Briefly, twins were eligible for participation in this study if one or both twins were matched successfully to birth records, were a member of a multiple birth with at least one male, were Caucasian, and were born between 1940 and 1974. Of 9417 eligible individuals for the first wave (1993–96), 6814 (72.4%) completed the initial interviews. At least 1 year later, we contacted those who had completed the initial interview to schedule a second interview. The second interview (1994–98) was completed by 5629 individuals, or 82.6% of those who had completed the first interview. The third interview wave (1998–2004) was completed solely by members of male–male twin pairs. Individuals were eligible for this study if they came from a male–male pair, and if both had been interviewed in wave 2.

The third interview included measures of retrospectively assessed peer group deviance (PGD) at five age-periods of 8–11, 12–14, 15–17, 18–21 and 22–25 years. The importance of these time-periods is underscored by observations that: (i) the mean onset or initiating age for most drugs use in the US general population is between 12 and 20 years [45,47,48]; (ii) cessation of drug use occurs normally by 29 years, while initiation rarely occurs after this age [49]; and (iii) incidence of drug use, abuse and dependence peaks from age 15 to 25 [45,50,51]. The PGD data were based on 10 items obtained from two validated instruments [52,53] which assessed the proportion of the respondent's friends, at each particular epoch, who engaged in specific behaviors. Friends were defined as '. . . people who you would have seen regularly and spent time with in school and outside of school'. The 10 items were: (i) smoked cigarettes; (ii) drunk alcohol; (iii) got drunk; (iv) had problems with alcohol; (v) been in trouble with the law; (vi) stole or damaged property on purpose; (vii) smoked marijuana; (viii) used inhalants; (ix) used other drugs such as cocaine, downers or lysergic acid diethylamide (LSD); and (x) sold or gave drugs to other people. The five response options were: (i) none; (ii) a few; (iii) some; (iv) most; and (v) all. As an alternative to using raw sum scores, and because measurement error and item-specific variance are known to produce biased estimates in causal modeling [54], we estimated individual maximum likelihood factor scores for the PGD items at each time-point based on the factor loadings and item thresholds calculated under a unidimensional factor structure in the Mx [55] software package.

Cannabis use (CU) data were based on average monthly use. CU was measured in individual drug units and one joint was considered one dose. Therefore, if a subject smoked three joints per day every day, then monthly use was recorded as 90. In order to coincide with the fixed PGD measures, average monthly CU was calculated for the same five PGD age-periods.

In order to improve the quality of the PGD and CU retrospective measures which have the potential for recall bias and telescoping effects [56], the interview utilized a Life History Calendar format developed by Thornton [57]. This method has been shown empirically to improve the accuracy of retrospective reporting by providing multiple cues to improve the chances of accurate recall [57,58]. This makes the task more akin to the accurate and well-retained process of recognition than to the less reliable task of free recall.

Due to the sparseness in the cannabis use data at earlier ages, we limited our analyses to data between 15 and 25 years. In order to correct for skew, both the CU and the latent factor PGD scores at each time-period were recoded onto three- and five-point ordinal scales, respectively.

There were 1738, 1768 and 1761 male twins with complete latent factor PGD scores at times 1–3, respectively, which represented 73–75% of the eligible sample from the previous interview ($n = 2368$). Complete CU data were available from 1781 subjects at each time-period. Ages ranged from 24 to 62 years ($\mu = 40.3$ years, $\sigma^2 = 9.1$ years). There were 1736, 1765 and 1758 subjects with complete PGD and CU data times 1 through 3, respectively. Standardized Cronbach's alpha coefficients for the PGD items at times 1–3 were 0.90, 0.87 and 0.87, respectively. As reported elsewhere [27], PGD test–retest correlations for the three time-periods (based on 141 subjects interviewed on average of 29 days apart) were 0.81, 0.78 and 0.73, respectively. Age-adjusted retest correlations for CU were 0.97, 0.94 and 0.94 at times 1–3, respectively.

Zygosity and interview protocol

Zygosity was diagnosed using a combination of self-report measures, photographs and DNA analysis [33]. In both interviews, most subjects (~90%) were interviewed by telephone. A small number were interviewed in person because of subject preference, residence in an institutional setting (usually jail) or not having a telephone. The project was approved by the Virginia Commonwealth University institutional review board. Subjects were informed about the goals of the study and provided informed consent before interviews. Interviewers had a Master's degree in a mental health-related field or a Bachelor's degree in this area plus 2 years of clinical experience. The two members of a twin pair were each interviewed by different interviewers.

Statistical analyses

We used the raw ordinal analysis method in Mx [55] to analyze the twin data. This approach is based on the Central Limit Theorem, which assumes that ordered categories reflect an imprecise measure of an underlying, normal liability distribution, and that this distribution has one or more threshold values which discriminate between categories [59,60]. All analyses were corrected for the linear effects of age at interview to remove age and cohort effects which are confounded in these data.

In studies of monozygotic (MZ) and dizygotic (DZ) twins reared together, phenotypic variation can be explained by additive genetic (A), common environment (C) and random environment (E) variance components. With multivariate analysis, the additional information in the cross-twin cross-trait correlations can allow us to determine the extent to which genetic and environmental influences are shared in common or are variable-specific [61].

Decomposing the covariance

Multivariate analysis makes use of the information in the cross-twin cross-trait correlations to permit us to determine the extent to which two or more measured phenotypes can be explained by common genetic and environmental influences [61]. As our first aim was to determine how much of the covariance between PGD and CU can explained by shared genetic and environmental liabilities, we fitted a Cholesky decomposition to the data [54]. Illustrated in Fig. 1, this is a method of triangular decomposition where the first variable is assumed to be caused by a latent factor that can also explain some or all of the variance in the remaining variable(s). This pattern continues until the final observed variable is explained by a latent variable, which is uncorrelated with all preceding factors and influences only one variable (i.e. a factor specific to one variable). The same factor structure is repeated for the sources of variance described above (A, C and E). In order to estimate the variance in CU explained by PGD, we first entered PGD followed by CU.
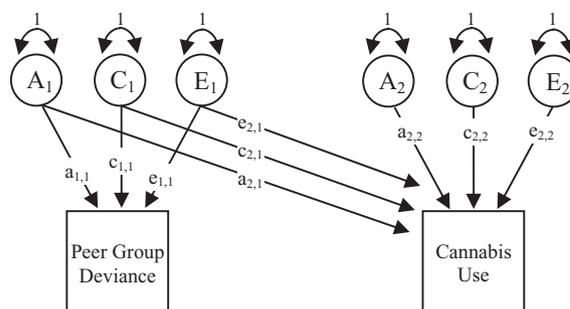

Figure 1 Cholesky decomposition to model the association between peer group deviance (PGD) and cannabis use (CU) by decomposing the source of covariance between PGD and CU into shared genetic ($a_{2,1}$) and environmental ($c_{2,1}$ and $e_{2,1}$) effects. This approach also models the genetic ($a_{2,2}$) and environmental ($c_{2,2}$ and $e_{2,2}$) effects which are unique to CU. A, C and E: latent additive genetic, shared and non-shared environmental effects for PGD and CU
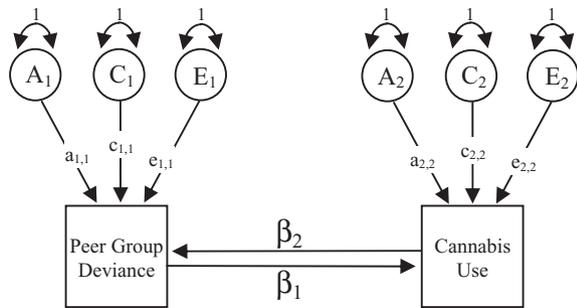
Figure 2 Modeling direction of causation between peer group deviance (PGD) and cannabis use (CU). This approach predicts the relationship between PGD and CU is explained by a reciprocal interaction at the phenotypic level. In the unidirectional PGD→CU and CU→PGD models, the β_2 and β_1 pathways are set to zero. A, C and E: latent additive genetic, shared and non-shared environmental effects for PGD and CU

Table 1 Comparison of monozygotic (MZ) and dizygotic (DZ) cross-twin cross-trait peer group deviance and cannabis use polychoric correlations with 95% confidence intervals for each time-period at 15–17 years, 18–21 years and 22–25 years.

	15–17 years	*18–21 years*	*22–25 years*
MZ	0.52 (0.45 0.59)	0.48 (0.41 0.55)	0.44 (0.36 0.51)
DZ	0.38 (0.27 0.47)	0.36 (0.25 0.45)	0.29 (0.18 0.39)

Modeling direction of causation

Under the Cholesky decomposition model any covariance between PGD and CU is attributable to unmeasured correlated, latent liabilities. This model is agnostic, in so far as it makes predictions about the direction of causation. It is possible to use the same cross-twin cross-trait correlations to test hypotheses about the direction of causation at the phenotypic level between variables measured at the same time. This form of modeling assumes that [62] sibling cooperation or rivalry is absent, the relationship between PGD and CU is equivalent for twin 1 and twin 2, the twin-pair correlations are different for PGD and CU [63] and there are no unmeasured variables which influence both measures, thereby inflating the correlations arising through the causal influence of one variable on the other. Based on the methods described elsewhere [44,62,64], we fitted a series of unidirectional and reciprocal causation models to the twin data illustrated in Fig. 2. Heath *et al.* [62] have shown that the unidirectional and reciprocal causation models are nested within the Cholesky decomposition, which permits model comparisons using goodness-of-fit statistics.

We chose a priori to retain all parameters in our best-fitting multivariate model. Sullivan & Eaves [65] have reported that in analyses based on discreet traits, estimates from the full ACE model will be more accurate and that attempts at parsimony result in oversimplification of the models, rather than a simpler and more accurate representation of the data. This will probably occur in cases such as ours, which involve more complex multivariate modeling and where the sample is not large enough to make definitive conclusions. Removing all parameters with lower bounds spanning zero, including parameters with small point estimates, i.e. <0.10, assumes that the

component of variance is known to be zero without any error variance, and if this argument is incorrect then future research might ignore an important source of variance [65].

RESULTS

Phenotypic and cross-twin cross-trait correlations

The cross-trait correlations between the latent factor PGD scores and CU at 15–17 years, 18–21 years and 22–25 years were 0.65, 0.64 and 0.61, respectively. The monozygotic (MZ) and dizygotic (DZ) cross-twin cross-trait correlations at each epoch are shown in Table 1. Based on the 95% confidence intervals (CI), all correlations were significantly different from zero, suggesting that familial aggregation accounts for some of the covariance between PGD and CU. All the DZ twin pair cross-trait correlations were greater than half the MZ twin pair cross-trait correlations, which suggests that a combination of genetic and shared environmental probably explains the familial covariance.

Multivariate analyses

We next compared the fit the Cholesky decomposition to the two unidirectional (PGD→CU and CU→PGD) and reciprocal causation (PGD→CU) models. As shown in Table 2, the PGD→CU model could be rejected at all ages. The causal CU→PGD and reciprocal PGD→CU models both provided a good fit to the data, as judged by the non-significant changes in log-likelihood and lowest sample size-adjusted Bayesian information criterion (BIC) (which has been shown to outperform the more traditionally used Akaike information criterion [66]). In the reciprocal interaction model, the standardized causal pathways (β_1) from PGD→CU at each time-period were negative and therefore more likely to be artifactual than substantive. They were also small (−0.14, −0.27 and −0.17), which meant that PGD explained very little variance in CU, so the results resemble more closely the CU→PGD model. Therefore, the more parsimonious CU→PGD causal model was chosen as the best-fitting.

Standardized path coefficients and their 95% CI for all parameters in the CU→PGD causal model are shown in

Table 2 Comparison of the (i) full Cholesky decomposition, (ii) unidirectional peer group deviance causes cannabis use (PGD→CU), (iii) unidirectional cannabis use causes peer group deviance (CU→PD) and (iv) reciprocal causation (PGD↔CU) models at 15–17 years, 18–21 years and 22–25 years.

	−2LL	df	Δ-2LL	Δdf	P	BIC
15–17 years						
Cholesky	6921.31	3546				−3214.81
PGD→CU	6935.70	3548	14.39	2	<0.001	−3211.38
CU→PGD	6923.23	3548	1.92	2	0.38	−3217.62
PGD↔CU	6921.34	3547	0.03	1	0.86	−3216.68
18–21 years						
Cholesky	7722.99	3541				−2804.56
PGD→CU	7752.65	3543	29.66	2	<0.001	−2793.50
CU→PGD	7726.97	3543	3.98	2	0.14	−2806.33
PGD↔CU	7723.28	3542	0.29	1	0.59	−2806.30
22–25 years						
Cholesky	7725.38	3542				−2805.25
PGD→CU	7744.32	3544	18.94	2.00	<0.001	−2799.54
CU→PGD	7729.08	3544	3.70	2.00	0.16	−2807.16
PGD↔CU	7728.00	3543	2.62	1.00	0.11	−2805.82

−2LL: log-likelihood; Δ-2LL: change in log-likelihood which is asymptotically distributed as a χ^2; BIC: sample size-adjusted Bayesian information criterion. All PGD and CU thresholds are adjusted for the linear effects of age at interview.

Table 3. Although we have retained all parameters in the model, the 95% CIs for the additive genetic and shared environmental pathways from PGD span zero at all ages. The same table also includes the CU→PGD causal parameters which are large, significant and range from 0.60 to 0.67.

Based on the path coefficients in Table 3, standardized variance components were estimated and appear in Table 4. The proportion of total variance in CU attributable to additive genetic effects was steady at 32–33% between 15 and 21 years but then increased to 54% between 22 and 25 years. By contrast, the proportion of shared environmental variance over the same period declined from 45% to 16%. Although the standardized additive genetic and shared environmental variance in PGD appeared stable over time, the proportion of additive genetic variance explained by CU increased; it ranged from 50% to 58% between 15 and 21 years and 78% between 22 and 25 years. This coincided with a decline in the proportion of shared environmental variance explained by CU, which equaled 73% between 15 and 17 years and 25% by 22 and 25 years. Most of the non-shared environmental variance (74–79%) in PGD was unique and not attributable to CU.

Because secular trends in the use of cannabis are unlikely to be linear, we re-ran our models with linear and quadratic age adjustments on the PGD and CU thresholds. We found an identical pattern of results; the unidirectional PGD→CU model was rejected while the CU→PGD causal model provided the best fit to the data.

DISCUSSION

This is the first study to examine the nature of the genetic and environmental association between the liability to affiliate with deviant peers and cannabis use. Our prediction was that part of the genetic risk for CU would be mediated through selection into deviant peer groups. Although there was a significant genetic contribution in the PGD–CU association, we found no evidence that genetic or environmental risks in PGD increase or mediate the risk of cannabis use. Instead, our results support the hypothesis that the association arises because of a causal pathway from CU to PGD. Between the ages of 15 and 25 years, CU explained between one-half and three-quarters of the genetic variance in PGD. Although declining over time, large proportions of the shared environmental variance in PGD were similarly attributable to CU. CU can therefore be understood as the 'risk indicator' for the liability to affiliate with deviant peers, because it appears to sit in the causal pathway between genetic and environmental risks, on one hand, and the expression of PGD on the other hand.

The significant association between PGD and CU is consistent with the seminal research by Dishion [67], who found that although early problem behaviors, poor peer relations and family management practices were correlated with drug use, these effects were non-significant when deviant peer affiliation was included. The question is whether socialization or

Table 3 Standardized path coefficients and 95% confidence intervals from the latent genetic, shared and non-shared environmental factors based on the best-fitting cannabis use (CU) to peer group deviance (PGD) causal model. Also included are the standardized causal pathways (β_2 in Fig. 1) at each time-period.

	Peer group deviance				Cannabis use		
	a_1	c_1	e_1	$\leftarrow \beta_2 -$	a_2	c_2	e_2
15–17 years	0.33 (−0.50 0.50)	0.27 (−0.46 0.46)	0.61 (0.56 0.67)	0.67 (0.62 0.71)	0.57 (0.27 0.78)	0.67 (0.44 0.82)	0.47 (0.40 0.55)
18–21 years	0.37 (−0.54 0.54)	0.30 (−0.49 0.49)	0.58 (0.53 0.64)	0.66 (0.61 0.69)	0.57 (0.25 0.78)	0.63 (0.37 0.79)	0.53 (0.47 0.60)
22–25 years	0.23 (−0.53 0.53)	0.42 (−0.53 0.53)	0.63 (0.57 0.68)	0.61 (0.56 0.65)	0.73 (0.47 0.87)	0.40 (−0.65 0.65)	0.55 (0.48 0.63)

a_1, c_1 and c_1: path coefficients from latent genetic environmental latent effects which explain variance in PGD: a_2, c_2 and c_2: path coefficients from latent genetic environmental latent effects which explain variance in CU.

social/self-selection provides a better explanation for the observed PGD–CU association.

The dominant socialization model [68–70] is well supported in the literature and has been considered by some to be responsible primarily for the relationship between PGD and liability to drug use [7,71–75]. However, this hypothesis, captured by our PGD→CU causal and reciprocal interaction models, is not well supported by our findings. Our results are also inconsistent with Wills' transactional model [70], which predicts that childhood temperament, family environment and peer effects all influence, and precede, the development of self-control, which in turn mediates the liability to drug initiation, regular use and abuse.

Our findings are, instead, more consistent with social or self-selection processes which drive and underpin deviant peer affiliation [76]. Snyder [6] has argued that when operating in open environments offering elective relationships, individuals select and affiliate with others who are behaviorally similar. A number of mechanisms, including temperament and maladaptive externalizing behaviours, have been proposed to make individuals more likely to affiliate with deviant peers [5,77,78] and biometrical modeling has also shown that variation in the sorts of friends we choose and with whom we affiliate can be explained partly by genes [79,80], which is consistent with our data.

Social and self-selection processes are related closely to genotype–environmental correlations in biology, which describe non-random distributions of environments among different genotypes. In other words, 'at-risk' genotypes gain or create more than their fair share of 'at-risk' environments. If expanded to include at-risk 'phenotypes', then this concept is in line with the causal CU→PDG model in which genetic and environmental risks in CU also increase the risk of individuals seeking out and affiliating with similarly inclined peers.

We still do not know if the 'A', 'C' and 'E' in CU are the causal components in the relationship with CU or whether they index more distal phenotypic or broader genetic risks which predispose individuals to drug use which, in turn, mediates the risk of affiliating with deviant peers. However, we do know that the 'A' and 'C' risks are shared in common with the genetic and shared environmental risk for using and abusing cocaine, hallucinogens, sedatives, stimulants and opiates [81,82], and that a number of other risks which CU might index have also been shown to elevate the risk of affiliating with deviant and drug-using peers [5,76,78,83]. Environmental variables such as family structure [84–86] and adverse family environments [78] are predictive of PGD, but the extent to which these are correlated with 'A', 'C' and 'E' in CU remains unclear. Combined, our findings support the interpretation that the environmen-

Table 4 Standardized proportions of additive genetic (A), shared (C) and non-shared (E) environmental variance in cannabis use (CU) and peer group deviance (PGD) including the proportions of variance in PGD which can be explained by CU as well as variance which is unique to PGD.

	15–17 years		18–21 years		22–25 years	
	PGD	CU	PGD	CU	PGD	CU
Total A variance (%) →	25	33	28	32	25	54
A explained by CU	58%	–	50%	–	78%	–
A unique to PGD	42%	–	50%	–	22%	–
Total C variance (%) →	27	45	26	39	24	16
C explained by CU	73%	–	66%	–	25%	–
C unique to PGD	27%	–	34%	–	75%	–
Total E variance (%) →	47	22	46	28	51	30
E explained by CU	21%	–	26%	–	22%	–
E unique to PGD	79%	–	74%	–	78%	–

Results are based on the best-fitting CU→PGD causal model path coefficients in Table 3 using Wright's [91] tracing rules.

tal and genetic risks explaining average cannabis use also mediate the risk of affiliating with deviant peers. This conclusion has implications for intervention and harm reduction strategies. If PGD is a 'downstream' consequence of CU, then earlier targeted interventions based on data which have clearly identified the 'A', 'C' and 'E' risks in CU are required in order to reduce mean levels and variation in maladaptive forms of both CU and PGD.

Limitations

Our findings must be interpreted in the context of three potential limitations. First, our data were drawn from white Virginian males. Males have a higher prevalence of drug use [87–90], and although previous analyses using the same data suggest that it is broadly representative of US males and does not differ from the general population in rates of psychopathological conditions, including illicit substance use, abuse and dependence [33], our results cannot be extrapolated to females. Secondly, we did not model cannabis initiation. However, because there is converging evidence showing how initiation, regular use and progression to abuse and dependence can be explained by common genetic and environmental processes [29,45], we would expect to see a similar pattern of results using binary measures of initiation. Finally, the latent PGD factor scores were assumed to take a unidimensional factor structure. The first three eigenvalues at 15–17 years (6.95, 1.12, 0.86), 18–21 years (5.56, 1.35, 0.80) and 22–25 years (5.85, 1.31, 0.73), followed by a comparison of the one- and two-factor solutions, suggested that a two-factor solution provides a marginally better fit to the data. These two factors were interpreted as 'general peer group deviance' and 'peer alcohol and cigarette use' dimensions. Between 18 and 25 years, the general peer group deviance factor also included illicit drug items. The peer alcohol and cigarette use factor included items for

alcohol-related problems at all ages, as well as peers' use of cannabis at 21–25 years. Modeling the two latent factor scores separately revealed an almost identical pattern of results. Regardless of whether the PGD construct was divided into peers' licit versus illicit drug use, or modeled as a predominately delinquency factor, the CU→PGD causal model still provided the best fit to the data.

CONCLUSION

Although our modeling was not exhaustive, we have demonstrated how genetic and environmental risks in CU and PGD are related. Until recently PGD was assumed to be an environmental [27] upstream risk factor for CU. The current data are not consistent with this hypothesis. Rather, the liability to affiliate with deviant peers was better explained by a combination of genetic and environmental factors for which CU could be understood as a causal 'risk indicator'. This is consistent with a social or self-selection process by which the genetic and environmental risks in CU largely underpin and drive the likelihood of affiliating with deviant peers.

Declarations of interest

None.

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