Genetic factors contribute to the association between peers and young adults smoking: Univariate and multivariate behavioral genetic analyses

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Abstract

This present study investigated the genetic and environmental influences on the associations between adolescents' peer characteristics (i.e., peer college orientation, and peer delinquency) and smoking in young adulthood. We used longitudinal data from the Nonshared Environment and Adolescent Development (NEAD) project. Parents’ reports on adolescents' peer characteristics and adolescents' self-reports on smoking in young adulthood were examined. Genetic and environmental influences on each construct as well as on the association between the two were analyzed. Findings showed that genetic and nonshared environmental influences contributed to peer college orientation and smoking status. Genetic, shared and nonshared environmental influences contributed to peer delinquency. Further, genetic and nonshared environmental influences contributed to the association between adolescents' peer college orientation and smoking in young adulthood. Genetic and shared environmental influences contributed to the association between adolescents' peer delinquency and smoking in young adulthood. In conclusion, the present study showed that genetic influences contributed to adolescents' peer characteristics and, in addition, genetic factors mediated the association between peer characteristics and smoking.

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1. Introduction

Tobacco use is widely recognized as an important public health issue with a broad impact (WHO, 2005). We know from previous research that most smokers start using tobacco products during adolescence and continue to smoke into adulthood. There are several factors contributing to adolescents' initiation and continuation of smoking, with peers as major one (Avenevoli & Merikangas, 2003). The amount of time children spend with peers increases with age and becomes especially important during adolescence (O'Loughlin, Paradis, Renaud, & Gomez, 1998). Evidence implies that certain peer characteristics such as smoking (e.g., Avenevoli & Merikangas, 2003), substance use, delinquency, school related problems and lack of college orientation (Dishion & Owen, 2002; Simons-Morton et al., 1999) increase the risk for smoking in adolescents and young adults. Based on evidence that an individual’s engagement with deviant peers may explain why individuals continue smoking (Avenevoli & Merikangas, 2003; Kobus, 2003) we expect that the peers with whom adolescents engage can have a lasting effect on their smoking. There are three possible explanations for this association: selection processes, influence processes and demographic propinquity. Selection processes suggest that adolescents affiliate with peers who are similar to themselves. Influence processes suggest that adolescents...
become similar to their peers by interacting with them. Finally, Rose (2002) suggests a third process, ‘demographic propinquity’. Adolescents have more opportunities to interact with peers who are in the same region, community or neighborhood and this may create similarities in adolescent affiliations that would not be present in other situations. Although all three processes may play a role in similarity among peers, findings from prospective studies focusing on influence and selection processes have found that selection plays a greater role (e.g., Bauman & Ennett, 1996). However, it is difficult to unravel these three processes in a survey design. In this present study, we use a genetically informative design to examine the association between adolescent peer characteristics and young adult smoking. The use of this type of a design allows us to examine the possible processes and mechanisms involved in associations between peer groups and smoking behavior.

Genetically informative studies, such as twin studies, estimate genetic, shared environmental (i.e., nongenetic factors that make siblings similar to one another), and nonshared environmental (i.e., nongenetic factors that make siblings different from one another) influences contributing to individual variation. The genetic and environmental influences underlying the association between peers and smoking are usually not considered. Estimating genetic and environmental influences on these associations, however, may provide important clues to how these associations arise. For example, finding genetic influences for the association between peer characteristics and smoking supports the selection process: individuals selecting and being selected by others because of genetically influenced characteristics. However, if environmental influences were found to be the most important explanation for this association the influence process and demographic propinquity explanations would be supported. Such findings would suggest that individuals are affected by others through imitation or modeling or even by the characteristics of the neighborhood they live in. In conclusion, by examining these associations within the framework of a genetically-informed design, we will gain additional insight into the possible mechanisms involved which may then contribute to improving prevention programs. If genetics indeed play a role in the association between peers and individual’s smoking, it may be that individuals will profit more from a tailored prevention program than from the current ‘one size fits all’ prevention programs.

1.1. Peers

Rose (2002) found clear genetic influences on friend selection with monozygotic (MZ) twins choosing friends more similar to one another than dizygotic (DZ) twins. Findings from other studies examining genetic and environmental influences on peer relationships and peer group characteristics have been mixed. An early report from the Nonshared Environment in Adolescent Development (NEAD) project showed large and significant genetic influences for parent reports of adolescent peer group characteristics (i.e., peer college orientation, peer delinquency, and peer popularity) (Manke, McGuire, Reiss, Hetherington, & Plomin, 1995). Findings from adolescent self-reports on the same measure of peer group characteristics for a later assessment of the NEAD sample and from the Colorado Adoption Project (CAP) indicated a somewhat different pattern of findings that varied by construct and sample (Iervolino et al., 2002). Specifically, when just same sex siblings were included, only nonshared environmental influences were significant for peer delinquency in NEAD and CAP sample and for peer group popularity in CAP. Genetic and nonshared environmental influences were significant for college orientation and popularity for NEAD, although genetic influences accounted for less variance than found by Manke, McGuire, Reiss, Hetherington and Plomin (1995). For the CAP sample both genetic and nonshared environmental influences were significant for college orientation when all siblings were included (same and opposite sex).

Another study showed that adolescents’ exposure to friends’ substance use (i.e., tobacco and alcohol use) was primarily explained by genetic influences and to a lesser extent by nonshared environmental influences (Cleveland, Wiebe, & Rowe, 2005). Finally, a longitudinal study on peers’ smoking using an Australian twin sample (White, Hopper, Wearing, & Hill, 2003) indicated that genetic, shared and nonshared environmental influences contributed to peer smoking at age 13–18. However, at age 16–21 only shared and nonshared environmental influences were significant, and at age 18–25 only genetic and nonshared environmental influences contributed to peers’ smoking. Although the sample examined by White et al. (2003) was assessed longitudinally, the findings are cross-sectional and did not examine how genetic and environmental influences contributed to stability or change in peer smoking. In sum, the general pattern of findings suggest that individual differences in peer characteristics can be explained by genetic, shared environmental and nonshared environmental influences, with some variation due to construct and reporter.

1.2. Smoking

Behavioral genetic studies on smoking have shown that genetic influences contribute to smoking behavior, including smoking initiation, nicotine dependence, quantity of cigarettes smoked, smoking persistence, and current smoking status (Heath & Madden, 1995). Studies suggest that the genetic contribution is higher for nicotine dependence, smoking persistence and heavy smoking than for smoking initiation (e.g., Kendler et al., 1999; True et al., 1999). This suggests that genetic influences are strongest in the later phases of the smoking process (White et al., 2003).

1.3. The association between peers and smoking

To our knowledge there has been only one behavioral genetic study investigating the association between peer characteristics and smoking from adolescence to young adulthood. White et al. (2003) examined the cross-sectional associations between peers’ smoking and smoking in adolescence and young adulthood longitudinally from middle adolescence to early/emerging adulthood.
Their findings showed that when the adolescents were 13–18 years old, genetic and nonshared environmental influences were significant, while during later adolescence and early/emerging adulthood (16–21 years; and 18–25 years) only shared and nonshared environmental influences contributed to the cross-sectional association between peer group smoking and participant smoking (measured concurrently). This finding suggests that the factors influencing associations between peer group smoking and participant smoking change from middle adolescence to early/emerging adulthood. The authors interpreted this as implying that during adolescence genes contributed to smoking indirectly by influencing the choice of peers.

1.4. Present study

The current study extends previous work by White et al. (2003) by investigating, using longitudinal data from the NEAD project, whether the longitudinal association between other adolescent peer characteristics (i.e., peer delinquency, and peer college orientation) and smoking in young adulthood is ‘purely’ environmental or involves some degree of genetic mediation. In the current study we examined the following research questions: 1) do genetic, shared and nonshared environmental influences contribute to adolescent peers’ characteristics?; 2) do genetic, shared and nonshared environmental influences contribute to smoking during young adulthood?; and 3) do genetic, shared and nonshared environmental influences contribute to the association between adolescent peers’ characteristics and young adults’ smoking?

2. Method

2.1. Participants

Data used in these analyses were from the first and third assessments of NEAD. The first NEAD assessment occurred in 1988 with 720 families participating (see Reiss, Neiderhiser, Hetherington, & Plomin, 2000 for more information on the recruitment procedures and detailed sample characteristics). Families consisted of nondivorced and step families with two parents and two same-sex adolescent children from 9–18 years of age. Siblings needed to be within four years of age of one another and were comprised of five different types: MZ twins, DZ twins and full siblings in nondivorced families and full, half and step-siblings in stepfamilies. At this first assessment the nondivorced families experienced no divorce since the birth of the oldest child participating in this study and the stepfamilies were required to be in existence for at least 5 years. In the case of dual custody in stepfamilies, these families were only included in this study when the adolescent resided in the household at least half of each week.

At Time 3 the participating siblings ranged from 20–35 years of age. The mean age of the older child at Time 1 was 13.5 ± 2.0 and for the younger child the mean age was 12.1 ± 1.3. At Time 1, 48% of the sibling pairs were female, and 52% male. The majority of the participating families were Caucasian (93%) and middle class (average family income $25,000 to $35,000). The social class skew of the sample was a direct consequence of the inclusion criteria described above. The average years of education were 13.6 years for mothers and 14.0 years for fathers. At Time 3, at least one family member from 413 families participated. The primary reason that families did not participate at Time 3 was because the families could not be reached (N=204). Of the 516 families who could be contacted 16% refused to participate and 4% did not return their questionnaires. There were in general no mean differences in demographic characteristics (gender of the siblings, antisocial behavior of siblings, peer college orientation, peer delinquency, parents’ age, parents’ education, and family income) for families who participated only at Time 1 versus families who participated at both waves (Time 1 and Time 3). However, there were two exceptions. Mothers’ who participated only at Time 1 were significantly younger compared to the mothers who participated at both times, F(df=340, N=712)1.44, p=.0006. Further, mothers’ report on family income indicated that families who participated only at Time 1 had a significantly lower income compared to the families who participated at both times, F(df=361, N=697)1.27, p=.0283.

For the current report we examine only those families where we have self-report data on the young adult. Thus, the nondivorced families included data from 55 MZ twin pairs, 49 DZ twin pairs, and 39 full sibling pairs (FI). The stepfamilies included 53 full sibling pairs (FS), 34 half sibling pairs (HS), and 39 unrelated sibling pairs (US). The full range of genetic relatedness is encompassed by these six groups. MZ twins share 100% of their segregating genes; DZ twins and full siblings (FI & FS) share 50%, on average; HS sibling pairs share 25% on average; and US sibling pairs share 0%.

2.2. Procedure

Interviewers visited the families at home for the Time 1 assessment. During the home visits by the interviewers each family member filled in questionnaires individually. Recruitment and data collection for Time 3 participation was conducted entirely via telephone and mail-out questionnaires. All participants who had participated in NEAD were included in the sampling pool for the Time 3 assessment.

2.3. Measures

2.3.1. Peer characteristics

We used parent reports of adolescent peer group characteristics. The measure consisted of 28 items and is called “Parents’ Perceptions of Children’s Peer Groups” (PCP; Reiss et al., 1994). This measure was derived from the peer subscales of the SIDE...
Daniels & Plomin, 1985) and included two additional items on the number of adolescents’ friends that drink alcohol and use marijuana. Parents used a 4-point Likert scale (1=very much like, 4=very much unlike) to rate each adolescent’s peer groups on 28 different adjectives. The scores for these adjectives were combined to form three subscales (for more information on these subscales see Iervolino et al., 2002; Manke et al., 1995). In this study, we examined peer college orientation and peer delinquency subscales. Peer college orientation included items measuring aspects of the peer group such as ambitiousness, intelligence, and school achievement and peer delinquency included items measuring peers’ rebelliousness, drug-taking behavior, and unconformity. However, in this study peers college orientation was recoded so that higher scores indicated that the peer is less college oriented.

2.3.2. Smoking

The current smoking status of young adults at the third wave was assessed with self-reports. Young adults were asked "Do, or did, you smoke tobacco?", response category ranged from 0 'no, never' to 3 'yes currently, every day'.

2.4. Analyses

All analyses were conducted after controlling for age, gender, age difference, and age by gender effects (McGue & Bouchard, 1984). All variables were then normalized using the PROC RANK procedure in SAS (1999–2001).

2.4.1. Genetic model-fitting analyses

We examined genetic and environmental influences on peer characteristics, smoking and the associations between adolescent peer characteristics and young adult smoking using maximum-likelihood model fitting analyses with Mx (Neale, Boker, Xie, & Maes, 2002). In our study, data may be missing from one measurement occasion or from one twin. Therefore, to make use of all the data available, we used the raw data function in Mx (e.g. Bartels et al., 2004). This allows for the testing of the relative goodness of fit for nested models, which is what will be reported for the current study. Model-fitting also allows for the testing of alternative models and estimation of all genetic, shared environmental and nonshared environmental influences simultaneously.

2.4.2. Assumptions

A number of assumptions are implied in Fig. 1. First, the Equal Environments Assumption (EEA) assumes that the environmental influences relevant for the trait of interest are the same for MZ and DZ twin pairs. The EEA has been tested in the NEAD sample and found to be tenable (Pike, McGuire, Hetherington, Reiss, & Plomin, 1996). Another assumption of the model is that shared and nonshared environmental effects are the same across sibling types and gene-environment interaction is negligible. Finally, we assume that there was no selective placement of the stepsiblings and no assortative mating. See Pike, McGuire, Hetherington, Reiss and Plomin (1996) and Neiderhiser et al. (2004) for more information and detailed discussion on these assumptions for the NEAD sample.

1 Peer popularity was not correlated with smoking, and therefore we did not analyze this in the current study. There should be at least a moderate correlation (r>.20) between variables of interest to ensure that decomposing covariance into its genetic and environmental components is meaningful.

2 Peer college orientation was negatively correlated with young adults’ smoking, and because this may cause problems when analyzing with MX we recoded the variable peer college orientation.

3 SAS software. Version 8.02 of the SAS System for Windows. Copyright 1999–2001 SAS Institute Inc. SAS and all other SAS product or service names are registered trademarks or trademarks of SAS Institute Inc., Cary, NC.
2.4.3. Univariate analyses

The path diagram for this model is depicted in Fig. 1. This model was used to estimate genetic, shared and nonshared environmental influences on each construct (peer college orientation, peer delinquency, young adult's current smoking status). In Fig. 1, A represents genetic influences, C represents shared environmental influences, and E represents the nonshared environmental influences for each sibling in a pair. In the figure it is shown that the nonshared environmental factors are uncorrelated (0), and the shared environmental factors are correlated at 1.0. The correlation of the genetic factors varies with the degree of genetic relatedness of the sibling pair. The estimates from the latent to the observed variables are indicated by the paths labeled a, c, and e.

First the full model was run, followed by a series of nested models. In the first submodel the path a was dropped in order to test a model including only shared and nonshared environmental influences. In the second submodel the path c was dropped to test a model including only the genetic and nonshared environmental influences. For simplicity, only the full and best fitting models will be reported. To determine whether the submodels yielded an improved fit, we subtracted the log likelihood (−2LL) of the full model from the best fit model, which can be interpreted as a change in χ². We took the same steps for the change in Akaike’s Information Criterion (AIC). The AIC is an indicator of the most parsimonious model and is used to select the most parsimonious model in cases where the change in χ² is not significant. The best fitting model is indicated by the smallest AIC.

2.4.4. Bivariate analyses

A Cholesky model was used to examine genetic and environmental influences on the association between peer characteristics and smoking (Fig. 2). The latent variables A, C and E represent the genetic, shared and nonshared environmental influences on the association between peer characteristics and young adult smoking. This model also estimates genetic and environmental influences on smoking that are uncorrelated with adolescent peer characteristics in the latent variables a, c and e. The parameter estimates for genetic, shared, and nonshared environmental contributions to the association between the two measures are represented by a11 and a12, c11 and c12, and e11 and e12, respectively. The parameter estimates for the unique contributions to young adult smoking are represented by a22, c22, and e22. We used the same strategy to compute best fitting models as we did for the univariate analyses. First the full model was run, followed by a series of nested models. In the first submodel the path a12 from the genetic component was dropped to test the significance of genetic influences on the covariation between the two variables. It is only necessary to drop this path because this is where the genetic covariance between the two variables loads. In the second
submodel the path c12 from the shared environment component was dropped to test the significance of shared environmental contributions to the covariance. Finally, the third submodel the path e12 from the nonshared environment component was dropped in order to test the significance of nonshared environmental contributions to the covariance.

3. Results

3.1. Prevalence of smoking

Smoking status of the young adults indicated that 53.3% of the older siblings and 48.9% of the younger siblings had never smoked, 19.9% of the older siblings and 22.1% of the younger siblings used to smoke but quit, 9.6% of the older siblings and 11.4% of the younger siblings were currently smoking some of the time but not daily, and 15.7% of the older siblings and 16.9% of the younger siblings were currently smoking every day at the time of data collection. In addition, 1.5% of the older and 0.7% of the younger siblings had missing data on this specific item.

3.2. Correlational results

The phenotypic correlations between adolescent’s peer college orientation and delinquency with young adult’s smoking are .28 (p < .05) for both peer constructs. The more parents report at Time 1 that the peers of their offspring were involved in delinquency or were less college-oriented, the more likely that their offspring were smoking in young adulthood (Time 3).

3.3. Model fitting results

The standardized parameter estimates and confidence intervals for the full univariate models for peer and smoking variables are shown in Table 1. The model-fitting analyses showed that the AE model represented the best fit for the peer college orientation and smoking status. The full (ACE) model represented best the univariate model for peer delinquency. The model-fitting analyses replicate the findings reported by Manke et al. (1995). Specifically, genetic and nonshared environmental influences explained the variance in peer college orientation while genetic, shared environmental and, to a much lesser extent, nonshared environmental influences were found for peer delinquency. The influence on smoking status consisted of primarily genetic and non-shared environmental factors although there were small and nonsignificant shared environmental influences.

Table 2 contains the parameter estimates for each of the bivariate models, with the columns mapping onto the paths in Fig. 2. For example, the estimates reported in column a11 is the estimate for the path marked a11 in Fig. 2 and so forth. The full and best fitting models are presented in Table 2, with the full models presented first. The first set of model-fitting estimates (a11–e12) represent genetic, shared environmental and nonshared environmental contributions to the covariance between adolescent peer...
group characteristics and young adult smoking. The last set of model-fitting estimates (a22–e22) represent genetic, shared environmental and nonshared environmental influences that are unique to young adult smoking once the variance shared with peer group characteristics has been accounted for. The model-fitting statistics are also reported in Table 2 (columns 2–5). The Chi-square (which should be non-significant) and the lowest number for AIC indicate which model is the best-fitting model. Table 2 shows that the AE model best represented the association between adolescent’s peer college orientation and smoking in young adulthood. However, the AC model best represented the association between adolescent’s peer delinquency and smoking in young adulthood.

The proportion of covariance accounted for by each latent factor is presented in Table 3. The proportion of covariance accounted for was obtained by multiplying together the standardized paths leading to each latent variable (for example, paths a11 and a12 leading to A in Fig. 2) to obtain the parameter’s contribution to the phenotypic association. Adding the genetic and environmental contributions together results in an approximation of the estimated phenotypic correlation between peer characteristics and young adults’ smoking; any discrepancies are the result of rounding. To better compare the measures, we calculated the percentage of covariance accounted for by a particular parameter by dividing the contribution to the estimated correlation between peer characteristics and young adults’ smoking (shown in Table 3). The contributions to the covariance are reported in Table 3 only for the best fitting model. Genetic influences accounted for the largest percentage for the covariance between peer characteristics and young adults’ smoking. A modest percentage of the covariance between peer delinquency and young adults’ smoking was accounted for by shared environmental influences. In contrast, the association between peer college orientation and smoking status was accounted for by nonshared environmental influences in addition to genetic influences.

As noted above, the bivariate cholesky model also estimates genetic and environmental influences unique to young adult smoking after covariance with peer group characteristics has been accounted for. In all models, genetic and nonshared environmental influences uniquely contributed to smoking status independent of the influences of peer group characteristics.

Table 2
Standardized parameter estimates for the contributions to the association between parents’ report on peer characteristics and young adults’ self-report on smoking and for the unique variance of smoking

<table>
<thead>
<tr>
<th>Measure</th>
<th>Parameter estimates</th>
<th>A</th>
<th>C</th>
<th>E</th>
<th>Estimated correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peer college orientation</td>
<td>-2LL (df)</td>
<td>Δχ²</td>
<td>AIC</td>
<td>a11</td>
<td>a12</td>
</tr>
<tr>
<td>Full</td>
<td>5026.091 (1904)</td>
<td>.94</td>
<td>.28</td>
<td>.00</td>
<td>.06</td>
</tr>
<tr>
<td>Best-fitting Model 1</td>
<td>5041.010 (1905)</td>
<td>.92</td>
<td>.18</td>
<td>.51</td>
<td>.36</td>
</tr>
<tr>
<td>Model 2</td>
<td>5026.091 (1905)</td>
<td>.94</td>
<td>.28</td>
<td>.00</td>
<td>.33</td>
</tr>
<tr>
<td>Model 3</td>
<td>5027.148 (1905)</td>
<td>.94</td>
<td>.21</td>
<td>.00</td>
<td>.33</td>
</tr>
<tr>
<td>Peer delinquency</td>
<td>Full</td>
<td>4793.662 (1908)</td>
<td>.76</td>
<td>.31</td>
<td>.60</td>
</tr>
<tr>
<td>Best-fitting Model 1</td>
<td>4793.790 (1909)</td>
<td>.70</td>
<td>.14</td>
<td>.45</td>
<td>.52</td>
</tr>
</tbody>
</table>

Note: -2LL=Log likelihood; AIC=Akaike’s information criteria; The Chi-squares with an * indicate that the p < .05; A=genetic influence; C=shared environmental influence; E=nonshared environmental influence. Confidence intervals are reported in parentheses. Three best fit models were tested. In the first model only the path a12 from the genetic component was dropped. In the second model only path c12 from the shared environment component was dropped. And in the third model only path e12 from the nonshared environment component was dropped. We looked at Δχ² and AIC whether these three models were significantly improved.
4. Discussion

In the present study we used a genetic design to investigate genetic, shared and nonshared environmental influences on the association between peer characteristics during adolescence and young adults’ smoking behavior.

4.1. Peers

The adolescent peer group characteristics (i.e. peer college orientation and peer delinquency) on which we focused in our study were also investigated in two previous studies using the questionnaire. Our findings were similar to the findings of Manke et al. (1995), but were in contrast with the study of Iervolino et al. (2002) who used the Time 2 NEAD sample and the CAP (Colorado Adoption Project) sample. Iervolino et al. (2002) found only shared and nonshared environmental and no genetic influences while Manke et al. (1995) and the present study found genetic, shared and nonshared environmental influences. Further, Iervolino et al. (2002) found that peer college orientation was due to genetic, shared and nonshared environmental influences, although in the study of Manke et al. (1995) and our study heritability was higher and both found no evidence of shared environmental influences. One explanation for these differences is that in our study and in Manke et al. (1995) parent reports from NEAD at Time 1 were used to assess adolescent peer group characteristics, as child reports were not available. The Iervolino et al. (2002) study, in contrast, used child reports from NEAD Time 2 (late adolescence) and from a sibling/adoption study. Thus, it is likely that the difference in findings can be explained by the use of parent reports vs. adolescent reports and/or by age differences in peer group characteristics.

Our findings suggest that siblings within a family who are more genetically similar are engaged with more similar peer groups, at least for the constructs of college orientation and delinquency. These findings are consistent with previous findings that at least part of the reason adolescents are engaged with specific peer groups is because of their own genetically influenced characteristics (e.g., Cleveland et al., 2005; Pike, Manke, Reiss, & Plomin, 2000).

4.2. Smoking

Both genetic and nonshared environmental influences contributed to smoking status. Our findings on smoking are in line with previous findings which have found heritabilities ranging from 35% in young adults (White et al., 2003) to 50% in adults (Heath & Madden, 1995).

4.3. Peers and smoking

Genetic influences accounted for most of the association between adolescent peer group characteristics and young adults’ smoking. The genetic factors contributing to this association are likely due to a correlation between the genotype and the environment. Two types of genotype-environment correlation may explain the high heritability in peer groups in our study: evocative genotype-environment correlation and active genotype-environment correlation (Manke et al., 1995). Evocative genotype-environment correlation occurs when the environment (in this case, peers) react to genetically-influenced characteristics that contribute to the risk of early substance use (e.g., temperament). Second, the characteristics of the adolescent might change the group. In sum, adolescents may engage with certain peers and also certain peer groups may solicit the membership of certain individuals because of personality characteristics, cognitive skills and other heritable characteristics.

Nonshared environmental influences did not contribute to the covariation between adolescent peer group delinquency and young adult smoking. These findings are not consistent with those reported by White et al. (2003) which found that only shared and nonshared environmental factors influenced the association between peer smoking and smoking in young/emerging adulthood, although genetic and nonshared environmental factors did influence the association between adolescent peer characteristics and smoking in adolescence. White et al. (2003) suggest that when adolescents grow older the heritability contributing to the association between peer characteristics and smoking disappears. This is in contrast to our study where heritable characteristics still account for the association between adolescent peer characteristics and smoking in young adulthood. However, the difference in findings may be due to measure and analytic differences in the two studies. First, the relative contributions of genetic and environmental factors depends, in part, on the type of peer characteristics investigated. We examined peer delinquency and college orientation, while White et al. (2003) investigated peers’ smoking. It is therefore not entirely surprising, given the different peer constructs used in the two studies, that the estimates vary. A second explanation is that we looked at the association between peer characteristics during adolescence and smoking 11–13 years later. In contrast, White et al. (2003) examined cross-sectional associations during young/emerging adulthood. The mechanisms underlying contemporaneous and longitudinal associations may not be the same, resulting in differences in genetic and environmental influences.

4.4. Limitations

The present study has some limitations. First, the limitation of using parent reports is that parents may not know the covert behavior of the peers of their offspring. On the other hand, adolescents’ subjective self-reports of their peers’ characteristics have
other limitations (see Bauman & Ennett, 1996). In future research, it would be especially informative to explore these questions using multiple perspectives (including peer reports) to see if the pattern is different depending on who (i.e., peers, parents, or adolescents) is reporting adolescent’s peer characteristics. However, findings from other studies using different assessment strategies for measuring adolescent peers’ substance use and peer characteristics were in line with our findings that genetic influences contribute to peer characteristics (Cleveland et al., 2005; Daniels & Plomin, 1985).

Second, the present study was conducted on a primarily Caucasian US sample. The patterns for smoking behavior may be different in other ethnic groups and for populations in other countries. In addition, Lerman and Berrettini (2002) suggest that different ethnic groups may exhibit different allele frequencies for specific genes (e.g., Maes et al., 2004). Therefore, the genetic and environmental patterns found for the association between peer group characteristics and smoking may also differ for different ethnicities and/or populations.

Third, the number of sibling pairs within each sibling/family type did not permit us to look at gender-specific effects. Finally, we were unable to control for smoking at Time 1 or peer influences at Time 3 as these data were unavailable in the NEAD sample. If we had been able to do so, the phenotypic association would probably be lower but we expect that the genetic and environmental pattern would be the same.

4.5. Implications

The present study showed that genetic influences rather than environmental influences contribute to adolescents’ peer characteristics and to the association between peer characteristics and smoking. These findings support the selection process. We found less support for the influence process or demographic propinquity as the contributions of environmental influences were small. Thus, this suggests that selection processes are likely to play an important role in explaining why peers can have a long-term effect on their smoking. Thus, the content of prevention programs may need to be reconsidered. In addition to teaching adolescents to resist peer pressure to smoke, prevention programs might consider focusing on the selection processes in friendships, and make adolescents aware of their own role in these processes and the implicit choices they make with regard to engaging and selecting new friends (e.g., Engels, Knibbe, Drop, & De Haan, 1997).

The present study showed that genetic influences mediated the relationship between adolescent peer characteristics and young adult smoking. However, this report does not provide information, from the perspective of smoking, on how the genetic effects operate. There are two plausible mechanisms on how the genetic effects might operate. First, genes act on the central nervous system making some people more vulnerable to the addictive properties of nicotine. Second, genes might exert their influence by increasing the likelihood that adolescents will enter risky environments. For example, some individuals might be more vulnerable to environmental influences than others, depending on the individual's personality characteristics. More research is needed to understand how these two mechanisms might explain why peers with whom adolescents engage can have a long-term effect on their smoking. Better understanding of the genetic and environmental processes involved in smoking status may lead to improvement in cessation aids for individuals who smoke in young adulthood. The importance of individual genetically influenced characteristics indicated in our study implies that a successful approach for smoking prevention and cessation programs is a program or smoking treatment which is developed specifically to the characteristics of an individual. Therefore, Lerman and Berrettini (2002) suggest that a favorable approach might be to consider genetic research to tailor the intensity and type of smoking treatment (“treatment matching”). Such an approach may be more effective for individuals than the current “one size fits all” smoking treatment.

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References


