Adolescent Peer Choice and Cigarette Smoking: Evidence of Active Gene-Environment Correlation?

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Abstract

Both peer groups and genetics have been associated with adolescent smoking behavior. Recently, Loehlin (2010) reported that twin differences in alcohol use were associated with differences in the number of common friends. Twins with more friends in common were more similar in drinking, but only for DZ pairs. Using the same sample as Loehlin (the National Merit twins), we replicated all of these findings for a composite cigarette smoking measure and for smoking initiation, but not persistence. The pattern of results is most consistent with homophily, or the tendency to associate with individuals that are like oneself. If peer influence occurs in the presence of homophily, then active genotype-environment correlation will be induced.

Keywords

Peers; peer influence; cigarette smoking; tobacco; nicotine; genetics; homophily; gene-environment correlation

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Understanding the mechanisms behind our choices to undertake actions detrimental to health is one key to developing ways to effectively prevent these choices. The hazards of cigarette smoking are numerous and well documented (Prasad et al., 2009). Yet, why some individuals partake in this activity while others do not is still a topic of debate. The present study focused on the interplay between two known suspects, peer relationships in adolescence and genetic influences (Kobus, 2003; Munafò & Johnstone, 2008).

Peer Relationships

Association with cigarette smoking peers has been shown to increase the likelihood that one will also smoke (Alexander et al., 2001; Hoffman et al., 2006; Holliday et al., 2010; Pollard et al., 2010; Vink et al., 2003). For example, belonging to peer networks composed of at least half smokers or having best friends who smoked made individuals twice as likely to smoke (Alexander et al., 2001). This observed homogeneity in smoking behavior between individuals and their peer groups was reviewed by Kobus (2003) who summarized the work done to pinpoint the specifics of *how* peers are related to smoking behavior, but concluded

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that further work should be done to illuminate the 'subtleties' behind the social dynamics of this contribution.

Two potential mechanisms may influence the correlation in smoking among peers—*homophily* and peer influence. Homophily is defined as the propensity to associate with more similar than dissimilar individuals (McPherson et al., 2001). McPherson et al. noted that it was originally assumed that peer groups directly influenced an individual's behavior. However, the surge of longitudinal data led to a shift towards recognition of the importance of homophily: individuals may actually select membership into groups that share one's initial behavioral characteristics. Thus, in the context of homogeneity of smoking between individuals and peer groups, arise two distinct, yet non-mutually exclusive, possibilities: (1) peer group may directly influence smoking behavior (peer influence), and (2) those with a propensity for smoking behavior may self-select into groups with similar characteristics (homophily).

The relative contribution of each of these possibilities, however, remains a matter of debate. Arnett (2007) rejected the assumption that the association between peer and individual smoking is a result of direct peer influence, and suggested that selection of friends based on a number of factors leads to peer group selection that creates a pathway to peer context variables such as group expectations, identity, and opportunities that may influence smoking behavior. Simons-Morton and Farhat (2010), on the other hand, conclude that both homophily and peer influence are important. Most reports emphasize that the magnitude of each influence remains unclear (Dishion & Owen, 2002; Go et al., 2010; Hall & Valente, 2007; Mercken et al., 2010; White et al., 2003).

Peer Relationships in the Context of Genetic Influences

Genetic factors have also been associated with smoking behavior (Munafò & Johnstone, 2008) and should be examined in terms of how they may connect individuals to particular peer groups. The interface between genetics and peer relationships has been demonstrated in results illustrating that the relationship between peer smoking and substance use depends on genetic liability (Agrawal et al., 2010; Harden et al., 2008; Johnson et al., 2010). Genetics may also play a direct role in friend selection, as it has been demonstrated that more genetically similar siblings have more similar peer groups (Harakeh et al., 2008) and that genetic factors may regulate exposure to peer substance use (Cleveland et al., 2005).

There is also evidence that the genetic contribution to smoking behavior may depend on the stage of smoking progression, from initiation to regular use and dependence. For example, Sullivan and Kendler (1999) demonstrated that shared environment may be more important for smoking initiation, while genetics may be more important in the transition from initiation to regular smoking and dependence. Numerous other studies have attempted to understand the degree to which the genetic and environmental factors influencing each of these phenotypes are related, but the general consensus is that there are factors that are both common and unique to each stage of the smoking process (Koopmans et al.,1999; Madden et al., 1999; Maes et al., 2004). However, little has been done to explore the degree to which

peers and the potential genetic implications of these peers may influence different stages of smoking.

Loehlin (2010) further illuminated the complex relationship among peers, genes, and substance use by examining whether more shared friends were associated with similarity in drinking behavior using the National Merit Twin Study (Loehlin & Nichols, 1976). Less shared friends were related to greater differences in alcohol drinking behaviors and, in females, the DZ correlation was stronger than the MZ correlation. Thus, genetic differences in DZ twins may result in different choices of friends that are related to differences in behavior for DZ, but not MZ, twins.

One mechanism that could contribute to such observed patterns of MZ and DZ correlations is homophily based on liability to smoking—including the overt phenotype of smoking—along with the risk and protective factors associated with smoking. When genes influence peer selection, the friends of MZ pairs will be more similar than those of DZ pairs along all genetically influenced domains of homophily, including smoking.

A popular model among behavioral geneticists combines genetically influenced homophily with peer influence. Here, genetic and environmental influences on smoking-related homophily variables lead individuals to associate with similarly inclined peer groups. Individuals within a group then mutually influence one another's behavior. Afterwards, genetic values for smoking become statistically correlated with environmental values for smoking, or active rGE (Eaves et al., 1977; Plomin et al., 1977; Scarr & McCartney, 1983).

Given expanded data sets on twins, adoptees, and biological relatives raised apart, it would be possible to assess and quantify the model described above. However, in this paper, we have the more modest goal of exploring the relationship between similarity in smoking status and similarity in peer groups for MZ and DZ twins raised together. The paper is effectively a "clone" of Loehlin's (2010) analysis of peer group similarity and twin similarity for alcohol use. The only difference is the phenotype.

Thus, using the same National Merit Twin Study dataset as Loehlin (2010), we predicted that (1) more shared friends should be associated with less differences in smoking behavior and (2) that this negative correlation between shared friends and smoking behavior should be stronger for DZ twins. Given Loehlin's differential findings for each gender on the latter, we examined gender differences in the degree to which genetics and shared environment influence smoking behavior. We also examined the degree to which these effects exist when our smoking variable is separated to assess either smoking initiation or persistence.

Method

Participants

The sample consisted of 850 twin pairs (514 MZ and 336 DZ same-sex twins) that participated in the 1962 National Merit Scholarship Qualifying Test as high school juniors (Loehlin & Nichols, 1976). Exclusions and missing values reduced this sample to 509 MZ

twin pairs (216 male and 293 female) and 330 same-sex DZ twin pairs (135 male and 195 female).

Items of Interest and Scoring

Zygosity assignment was made on the basis of a questionnaire on reported similarities of a twin pair (Loehlin & Nichols, 1976). The original survey contained 3 questions on smoking:

1. How much do you smoke?

    With responses: (1) Never smoked, (2) Used to or occasionally smoke, (3) 1 to 19 cigarettes a day, and (4) greater than 20 cigarettes a day

2. If you smoke do you inhale the smoke into your lungs?

    With responses: (1) Don't smoke, (2) Rarely or never inhale, (3) sometimes inhale, and (4) usually inhale

3. (How often have you) smoked a cigarette or cigar before breakfast?

    With responses: (1) Frequently, (2) Occasionally, and (3) Not at all

Participants reported on the frequency of the said action, and a composite score was assigned for each individual in the following manner. Individuals who had never smoked were given a score of 1 (63% of individuals); individuals who occasionally smoked, but had never inhaled were given a score of 2 (10% of individuals); current or former smokers who had inhaled were given a score of 3 (14% of sample); current or former smokers who had both inhaled and had smoked before breakfast were given a score of 4 (3% of sample).

For the initiation part of our analysis, the composite score was used to dichotomize individuals into categories of initiation status: a binary variable of having never smoked (composite score of 1) or having initiated smoking behavior (composite score > 1). To assess smoking persistence, we only included twin pairs where each twin had initiated (27% of sample), and persistent smoking behavior was analyzed using the composite score (scores 2 though 4) described above.

Individuals missing scores on any of the above items were assigned scores based on responses to the other smoking items. For example, a respondent who omitted an answer to "how much do you smoke" but reported smoking before breakfast was assigned a 4. Such assignments involved only a small proportion of the sample (1.50%) and were made without knowledge of zygosity and twin's smoking status.

For the measure of shared friends, participants were asked: "Do you and your twin have the same or different friends? " Responses were on an ordinal scale ranging from a score of 1(all shared friends) to 4 (few to no shared friends). As in Loehlin (2010), we reverse scored this item for ease of interpretation. Thus, a score of 1 indicated few shared friends between twins and a score of 4 indicated complete sharing of friends between twins. To get a shared friends score for each twin pair, we averaged the two twins' shared friend scores. In the case of a missing shared friend score for one twin, we assigned that twin the score given by the other twin in the pair. If both twins had no score, this pair was excluded from the shared friends portion of the analysis.

Evaluation of Heritability and Gender Differences

All four gender by zygosity groups were fitted to a multifactorial threshold model, which assumed a threshold imposed on an underlying continuous distribution of factors related to liability of the smoking composite. The first question of interest was whether there was a sex difference in the additive genetic and environmental influences of the composite smoking variable that may account for differences in results for male and female twin pairs. Here, we tested whether the parameters of an ACE model could be equated across genders.

To examine the relationship between smoking and number of friends in common, we followed Loehlin (2010) and correlated the absolute value of the difference in pair smoking scores with the measure of common friends. This was done for the composite, initiation, and persistent smoking measures.

All statistical and model fitting analyses were conducted in R version 2.12.0 and OpenMx version 1.0.3-1505 (Boker et al., 2011).

Results

For simplicity, we report descriptive and univariate heritability measures for the composite smoking score that includes both components of the initiation and persistence measures.

Gender Differences in Shared Friends and Composite Smoking

Tables I and II present descriptive statistics for the gender-by-zygosity groups. Gender and zygosity effects were tested using a two-by-two ANOVA with an interaction term. No interactions were significant, so here we report the marginal differences.

Males ($M = 1.68$, $SD = .80$) had higher smoking composite scores than females ($M = 1.57$, $SD = .66$), but the difference was only marginally significant ($t(837) = 1.95$, $p = .05$). Females ($M = 3.14$, $SD = .65$) reported more shared friends than males ($M = 3.02$, $SD = .59$), ($t(836) = -2.67$, $p = .008$).

Zygosity Differences in Shared Friends and Smoking

Across sex, MZ and DZ twin pairs only marginally differed in their smoking composite scores, with DZ twin pairs ($M = 1.68$, $SD = .81$) having slightly higher composite scores than MZ twin pairs ($M = 1.57$, $SD = .79$), ($t(837) = -2.01$, $p = .05$). However, DZ twin pairs ($M = .49$, $SD = .76$) were significantly more divergent in their smoking behaviors than MZ twin pairs ($M = .31$, $SD = .60$), ($t(837) = -3.62$, $p = .0003$). MZ twin pairs ($M = 3.22$, $SD = .57$) also shared significantly more friends than DZ twin pairs ($M = 2.87$, $SD = .64$), ($t(836) = 8.03$, $p < .001$).

Heritability of Smoking Composite

Table III presents the biometrical genetic model for the smoking composite measure. Thresholds could be equated across twin pair ($\chi^2 (8) = 3.19$, $p = .92$) and zygosity ($\chi^2 (4) = 6.23$, $p = .18$), but not gender ($\chi^2 (2) = 12.95$, $p = .002$). The ACE model with equal parameters across gender did not significantly reduce fit compared to the model that allowed

these parameters to vary separately for each sex (χ^2 (3) = 4.61, p = .20). Therefore, analysis was continued jointly for male and female twin pairs. Both the Akaike Information Criterion (AIC) and the likelihood ratio tests suggested that the model containing all three variance components is to be preferred. Compared to a full model, the CE model ($\chi^2(1)$ = 14.46, p < 0.001) and AE model ($\chi^2(1)$ = 8.98, p = .003) could convincingly be rejected.

Differences Between MZ and DZ Correlations

To evaluate the possibility of active rGE, we examined the polychoric correlation between absolute differences in smoking between twins and the mean amount of shared friends each twin reported (Table IV).

For the smoking composite, both the correlations for MZ males and MZ females did not significantly differ from 0. However, the DZ correlations were significant for both males and females. Hence, for DZ but not MZ twins, more shared friends predicted greater twin similarity in smoking. However, the difference between the MZ and DZ correlations was not significant for female (χ^2 (1) = 2.44, p = .12), but marginally significant for male ($\chi^2(1)$ = 3.49, p = .06) twin pairs.

For smoking initiation, polychoric correlations between shared friends and differences in smoking for MZ males and females did not significantly differ from 0. Yet, for DZ males and females, the correlation between shared friends and absolute difference in smoking initiation was significant. Further, for both males ($\chi^2(1)$ = 6.15, p = .01) and females ($\chi^2(1)$ = 4.48, p = .03) the DZ correlation between smoking and shared friends was significantly stronger than the MZ correlation. Therefore, the initiation phenotype gave the same results as the composite measure.

For smoking persistence, however, the previous pattern of correlations was not observed. Correlations for MZ males (n =61), MZ females (n = 76), DZ males (n =44), and DZ females (n =48) did not significantly differ from 0. Thus, shared friends did not predict similarity in smoking status beyond initiation.

Discussion

From the results, two major findings are highlighted. First, in DZ, but not MZ, twin pairs there was a relationship between number of shared friends and similarity of the smoking composite and initiation score. Thus, these results were consistent with the possibility that genetic differences within DZ twin pairs may influence the choice of friends with characteristics that correlate with each twin's unique genetic predispositions, which in turn may explain corresponding differences in the pair's smoking.

Second, when the smoking variable was reduced to include only individuals that had initiated for the smoking persistence part of the analysis, we did not find this same pattern. This was unsurprising especially given the small sample size of roughly between 40 to 80 twin pairs per group. The two items used to define smoking persistence, whether one inhales or smokes before breakfast, may also not have been the most optimal or relevant measure of smoking persistence in an adolescent sample (Heatherton et al., 1991). Therefore, our

composite smoking measure may more accurately assess initiation rather than smoking persistence, and while our results for initiation may be valid, limitations of the current dataset may be unable to conclude on the effect of shared peers on smoking persistence past the stage of initiation.

However, before substantive interpretation of these findings, it is important to first rule out other mechanisms that could contribute to our observed pattern of correlations.

One potential issue is the possibility that peer influence may violate a cardinal assumption of the twin method, namely, that the correlations in latent, trait-relevant environmental values are equal for MZ and DZ pairs. Similar peer groups for MZ twins may partly arise from the extra attention these twins receive by being together in a group. Kendler and Gardner (1998) evaluated whether this mechanism could play a role for both smoking initiation and nicotine dependence. They reported that twins with higher 'co-socialization' scores, a factor based upon items related to how often twins socialized together, resembled each other more with respect to smoking initiation but not nicotine dependence. However, this mechanism would also predict high correlations between peer group differences and within-pair smoking differences for *both* MZ and DZ twins. Thus, our results are inconsistent with this being a *strong* mechanism behind differences in correlations between MZ and DZ twins.

A second mechanism that could influence the relationship between peers' smoking behavior is passive assortment, or peer associations based on background variables that are correlated with smoking. Family socioeconomic status is a clear example. In nationally representative twin samples, simple geography, ethnicity, culture and religious affiliation may influence both peer similarity and smoking behavior (Degenhardt et al., 2007). However, neither Loehlin's nor our results were consistent with pure passive assortment as the pre-eminent mechanism for the correlation between self- and peer-drug use behaviors. Friendship groups based on background factors correlated with smoking should be the same for MZ and DZ twins, leading to identical correlations for MZ and DZ twins in peer group differences and within-pair smoking differences.

Hence, the most likely factors contributing to the observed homogeneity in peer group smoking are a combination of homophily and, possibly, peer-influence. Unfortunately, we could not quantify the precise contribution of each mechanism with the current data, because there were no data on smoking in the twin's friends. The data, however, are not consistent with a strong role for peer influence. If peer influence were very important, then we should have observed at least a trend toward significance in the correlations between smoking status and peer-group similarity in MZ twins. Yet, with the exception of smoking persistence, all MZ correlations in Table IV are very close to 0.

Hence, the pattern of results definitely supported homophily as an important mechanism. In contrast to peer influence alone, homophily predicts that peer groups should be more similar for MZ than DZ twins. Hence, our results *could* be consistent with active rGE as an explanation why individuals and their peer groups tend to share smoking habits, if this homophily is accompanied by peer influence. However, we cannot definitively conclude that both of these requirements for rGE are taking place given the limitations of a cross-

sectional, twin dataset. Future analysis on more expanded datasets that include twins, adoptees, and siblings raised apart may illuminate the specific contribution of rGE, especially with longitudinal data from adolescence until early adulthood.

A further caveat was the age of the participants. Participants were only evaluated at the single time point of juniors in high school (~17 years of age). However, longitudinal analyses have suggested that genetic and environmental components related to peer influences might vary across different ages within the span of early adolescence to young adulthood (Vink et al., 2003; White et al., 2003).

Additionally, given only a single time point of evaluation, it is important to take into consideration cohort differences in the etiology of smoking behavior. Boardman et al. (2010) demonstrated the dynamic nature of heritability for regular smoking behavior across a series of cohorts born in the United States, finding rather negligible genetic influences for those born in the 1940s, the cohort of the National Merit Twins (Loehlin & Nichols, 1976). They reasoned that social pressures might have pushed the popularity of smoking to a level in which genetically vulnerable individuals were no more likely to smoke than those without genetic predispositions toward smoking. Kendler et al. (2000) examined the heritability of regular smoking by birth cohort for males and females separately in Sweden and found that heritability for women was actually greatest for those born after 1940. The reasoning in this case was not so different from Boardman et al.'s, but appears to have quite a different effect on the manifestation of genetic influences for women: decline in the social stigma of women smoking may have allowed women to partake in behaviors aligned with their genetic propensities. Given evidence of these changes in the etiology of smoking behavior over time, it becomes difficult to generalize both the genetic and peer influence components of our model to the present era. Therefore, given that we only had participants at a single age from a single point in time, it is possible that our results illustrated only a snapshot of the true mechanisms by which smoking behavior may be regulated through the influence of genes on peer choice.

Further issues discussed by Loehlin (2010) regarding this sample included the ability of the questionnaire to accurately assess the number of shared friends and the key behavior (smoking in this study). Also addressed was the fact that variance in the key behavior may have been restricted by a select sample of high-achieving participants.

Thus, our findings are consistent with Loehlin's (2010) for alcohol-related behavior in that there appeared to be a pathway to smoking through both genes and peer groups. However, the contribution of active rGE that requires both genetically-based homophily and additional peer influence remains unclear. Yet, given evidence in the literature for the dual contribution of selection and peer influence to smoking behavior (Simons-Morton & Farhat, 2010), rGE might be a likely scenario. Thus, future investigations should specifically test for the contribution of rGE using datasets more amenable to quantifying the alternative contributions of unequal twin environments, passive assortment, or peer influence/ homophily that may produce similar results.

In conclusion, our results further evidenced the contribution of genetics on exposure to environments that may influence our behaviors. However, given the unclear effect of sex and age on such findings as well as the inconclusive evidence as to the full range mechanisms at work, the next step is to further illuminate both the ways by which rGE might specifically be at work and what other factors may contribute to homogeneity between individuals and their peer groups in smoking behavior. Particularly, gaining a clearer understanding of this association will allow future research to examine ways toward effective prevention of the problems associated with cigarette smoking.

Acknowledgments

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Table 1

Means and standard deviations on smoking composite and absolute difference in smoking composite between twins for the four samples.

		Composite Smoke Score		Absolute Difference in Smoke Score	
	N	Mean	SD	Mean	SD
MZ Males	216	1.62	.78	.40	.65
MZ Females	293	1.53	.80	.25	.55
DZ Males	135	1.77	.82	.53	.73
DZ Females	195	1.62	.79	.47	.78

Table 2

Mean and standard deviations for mean shared friends for the four samples.

	N	Mean	*SD*
MZ Males	215[a]	3.17	.53
MZ Females	293	3.27	.60
DZ Males	135	2.79	.60
DZ Females	195	2.94	.66

[a] 1 missing value

Table 3

Smoking composite model fit statistics and variance component estimates with 95% confidence intervals for ACE and nested AE and CE models.

Model	EP	df	−2LL	AIC	χ^2	df	P	VA	VC	VE
ACE[a]	7	1623	2594.27	−651.73	---	---	---	.43 (.20–.69)	.39 (.15–.60)	.18 (.13–.24)
AE	6	1624	2603.25	−644.75	8.98	1	<.01	.83 (.78–.87)	---	.17 (.13–.22)
CE	6	1624	2608.73	−639.27	14.45	1	<.01	---	.74 (.68–.79)	.26 (.21–.32)

[a]Threshold values: Males thresholds: t1: .25, t2: .54; Female thresholds: t1: .48, t2: .43

Table 4

Polychoric correlations between absolute difference in twin pair smoking behavior and average shared friends for composite, initiation, and persistence smoking measures.

Group	Composite r^a (s.e)	Initiation r^a (s.e.)	Persistence r^a (s.e.)
MZ males	.01 (.09)	−.05 (.10)	−.07 (.16)
MZ females	−.10 (.09)	−.02 (.10)	−.25 (.15)
DZ males	−.23[*] (.10)	−.39[*] (.11)	.16 (.20)
DZ females	−.29[*] (.09)	−.31[*] (.09)	−.10 (.19)

[*] p < .05

[a] All correlations met assumption of bivariate normality.