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Original Investigation

Genetic and Environmental Influences on the Association Between Depressive Symptom Dimensions and Smoking Initiation Among Chinese Adolescent Twins

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Abstract

Introduction: Extant twin research on the depression–smoking association in adolescents has been conducted in U.S. and European samples and considered depression as a unitary phenotype. This study explored genetic and environmental influences on covariation between smoking initiation and 4 depressive symptom dimensions (positive affect [PA], negative affect [NA], somatic features [SF], and interpersonal problems [IP]) in adolescent Chinese twins.

Methods: Questionnaires measuring current depressive symptoms and lifetime smoking initiation were administered to 602 twin pairs (*M* [*SD*] age = 12.2 (1.93) years, range 9–16 years). Cholesky bivariate decomposition models examined influences on each depressive symptom dimension, smoking initiation, and their covariation using age- and sex-adjusted threshold variables.

Results: Within-twin correlations between smoking initiation and each depressive symptom dimension were significant (|*r*|s = .29–.61). Bivariate twin modeling showed significant genetic effects on overall depressive symptoms (55% variance), shared environment effects on NA (36%) and PA (53%), and shared environment effects on smoking initiation (46%) unique from PA. No other familial influences on the individual phenotypes (apart from those accounting for smoking–depression covariance) were significant. Relations of smoking initiation to overall depressive symptoms and IP were influenced by familial (shared environment and/or genetic) factors and nonshared environmental factors. The SF–smoking initiation relation was influenced mostly by familial factors. Only shared environment significantly influenced the association of lower PA and higher NA to smoking initiation.

Conclusions: Relations between each symptom dimension and smoking initiation are of sizeable magnitude in Chinese adolescents. Genetic and environmental factors underlying depression–smoking comorbidity may vary across different depressive symptom dimensions.

Introduction

Depressive symptoms and cigarette smoking tend to co-occur and are bidirectionally associated in longitudinal studies of adolescents (see Chaiton, Cohen, O'Loughlin, & Rehm, 2009 for a meta-analytic review). Twin studies represent a promising approach to elucidating the extent to which depression–smoking relations are due to genetic and environmental influences. However, prior investigation of depression–smoking association in adolescent twins is limited. This is an important gap in the literature as studying smoking initiation and depressive symptoms in adolescent twins (as opposed to examining retrospective reports of early smoking and depression in adult twins) also offers the methodological advantage of reducing retrospective recall biases.

To our knowledge, only a few adolescent twin studies of depression and smoking have been carried out, all of which were conducted in U.S. and European settings. A study of Finnish adolescent twin pairs who were discordant for depressive disorders found that history of depressive disorder occurring prior to age 14 predicted use of smokeless tobacco by age 17.5 increased

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risk for daily smoking, but did not predict smoking initiation (Sihvola et al., 2008); this study did not utilize genetic modeling approaches. In a genetic modeling study of U.S. adolescent twins, Silberg, Rutter, D'Onofrio, and Eaves (2003) found that early experimental smoking and depression were genetically correlated in girls but environmentally correlated among boys. Finally, in U.S. adolescent twin pairs, McCaffery, Papandonatos, Stanton, Lloyd-Richardson, and Niaura (2008) found that nonshared environmental factors explained the relation between level of depressive symptoms and smoking involvement and that there was a common genetic source of depression–smoking covariation in girls.

Given the substantial public health burden of smoking in Asian countries, such as China (Qian et al., 2010), it is important to understand influences on smoking initiation in Chinese adolescents. A sizeable number of Chinese begin smoking in adolescence, with evidence that for boys, the hazard of smoking initiation is very low (<2%) before 7 years of age, increases rapidly after age 10 years, and peaks at 14–15 years, whereas the hazard for girls is low (<1%) until 12 years of age before it increases by age 15 years, but the overall prevalence of smoking is higher in boys than in girls (Chen et al., 2001). The cultural characteristics of Asia are different from western societies, which may modulate the expression of genetic vulnerabilities on depressive symptoms, smoking initiation, and their association (Unger et al., 2011). For example, although most Asian countries have laws restricting youth access to tobacco, enforcement of these policies is inconsistent, and it is common for adults to offer cigarettes to adolescents on special occasions or as a rite of passage into adulthood (Okamoto et al., 2010). Adolescents in China often report high levels of academic stress because admission to prestigious high schools is very competitive, and they tend to report smoking to cope with stress while studying for stressful exams (Booker et al., 2007; Okamoto et al., 2010). Also, Asian populations also have different allele frequencies for genetic variants implicated in depression and smoking (e.g., Kang, Palmatier, & Kidd, 1999). Altogether, these factors could impact the relation between depressive symptoms and smoking in Chinese adolescents as well as the relative roles of environmental and genetic influences on this association.

It is also important to identify which aspects of the depression phenotype are most strongly associated with smoking. Prior twin studies of depression–smoking associations have primarily modeled depressive symptoms as a unitary phenotype. However, depressive symptoms may be more aptly characterized as a collection of multiple intermediate phenotypes that can each be isolated by parsing depression into its constituent symptom dimensions (Hasler, Drevets, Manji, & Charney, 2004). For example, investigators have used the four-factor model based on the Center for Epidemiological Studies Depression Scale (CESD; Radloff, 1977; Shafer, 2006), which separates depression into discrete dimensions of negative affect (NA; sadness, distress, and worthlessness), somatic features (SF; poor appetite, low energy, and sleep disturbance), low positive affect (PA; diminished positive emotions and pleasure), and interpersonal problems (IP; poor social adjustment). Separate dimensions of depressive symptomatology exhibit disparate patterns of association with some smoking characteristics in adults (Korhonen et al., 2011; Leventhal, Ramsey, Brown, LaChance, & Kahler, 2008; Mickens et al., 2011; Pomerleau, Zucker, & Stewart, 2003; but also see Prochaska et al., 2004). Thus, it is of interest to examine the relation between smoking initiation and depressive symptom dimensions in adolescents.

The present study explored genetic and environmental sources of covariation between depressive symptom dimensions and smoking initiation in Chinese twins aged 9–16 years old. Examining smoking initiation in this age group is important, given evidence that those who have their first cigarette before (vs. after) age 17 are more likely to be persistent smokers in adulthood (Breslau & Peterson, 1996). Of particular interest was whether the magnitude of correlation and influences on covariation between depressive symptoms and smoking initiation differed across separate symptom dimensions.

Methods

Participants and Procedure

Participants in this cross-sectional study were members of the Qingdao Twin Registry (QTR; Pang et al., 2006), which is a subset of the national Chinese Twin Registry. Twins were recruited to join the QTR via the general newborn registry for the city of Qingdao, connections with school nurses, and outreach using media campaigns (Pang et al., 2003). As of 2005, the QTR had 10,655 twin pairs and was estimated to include 74% of all twins living in Qingdao (Pang et al., 2006).

The current sample comprises a youth cohort of 321 monozygotic (MZ) pairs and 281 dizygotic (DZ) twin pairs who were contacted from the QTR in 2005 and 2006 and asked to take part in a multiwave study of health and behavior (Unger et al., 2011); the current study focuses on only the first wave to maximize data and offset attrition at later waves. Of the 281 DZ pairs, 109 were opposite sex pairs, 81 were same-sex male pairs, and 81 were same-sex female pairs. Of the 321 MZ pairs, 170 were female pairs and 151 were male pairs.

Participants attended a study session at the Qingdao Centers for Disease Control (QCDC), conducted by QCDC staff members trained in the study protocol. After a description of the study procedures and confidentiality, parental consent, and twin assent to participate, twins completed surveys that assessed depressive symptoms, smoking behavior, and other characteristics not reported here. The study was approved by Institutional Review Boards at the University of Southern California and the QCDC.

Measures

Zygosity

Zygosity was determined by simultaneous detection of multiple short tandem repeat loci in blood samples (Lv, Zhan, & Qin, 2003). In the Chinese National Twin Registry, the probability of correctly identifying monozygosity based on these markers was ≥ .996.

Surveys

As part of a multistage translation process, bilingual researchers, public health experts, study staff, and QCDC staff members translated, verified, and evaluated each question on the survey for local idioms and reading level (Unger et al., 2011).

The Center for Epidemiological Studies Depression Scale

The CESD is a 20-item self-report measure of past-week depressive symptoms with suitable psychometric properties in prior studies of Chinese adolescents (Chou, 1999, 2000; Radloff, 1977; Yang, Soong, Kuo, Chang, & Chen, 2004). Each item lists a particular symptom for which respondents indicated how often they felt that way in the past week: rarely or none of the time (0–1 days, 0 point), some or a little of the time (1–2 days, 1 point), occasionally or a moderate amount of the time (3–4 days, 2 points), or most or all of the time (5–7 days, 3 points).

Although the factor structure is not entirely consistent across all studies, a meta-analysis of CESD factor analyses in 28 diverse samples ($N = 22{,}340$) found a clear four-factor solution that distinguishes PA, NA, SF, and IP (Shafer, 2006). An exploratory factor analysis using principal axis factoring with a promax rotation in our data found two primary factors accounting for 34% and 13% of the variance, respectively, with one factor exhibiting strong loadings from PA items and another with strong loadings from items representing NA, SF, and IP dimensions. Nevertheless, as in previous work (Leventhal et al., 2008; Pettit et al., 2008), we utilized the four-dimension approach and computed subscale scores for each of the four dimensions by computing its respective items' average score (these continuous scores were then later categorized for twin modeling, see "Data Analysis" section below). This approach was adopted for several reasons. First, several prior studies examining relations between depressive symptom dimensions on the CESD and smoking variables have utilized the four-subscale approach and have found that all four of the different subscales exhibit different patterns of associations with smoking characteristics (Leventhal et al., 2008; Mickens et al., 2011; Pomerleau et al., 2003). Thus, by utilizing this method, we allow for greater comparability with past research and prevent potentially obscuring certain depression–smoking associations by collapsing across subscales. Second, all four subscales are phenomenologically different from one another. Thus, examining the dimensions separately from one another may yield insights into different theoretical mechanisms that may underlie depression–smoking comorbidity across these subscales.

The items for each dimension are PA (hopeful about future, enjoyed life, felt as good as others, and was happy; Cronbach's $\alpha = .79$), NA (felt sad, crying spells, could not shake blues, felt depressed, felt lonely, felt fearful, and life is a failure; $\alpha = .85$), SF (appetite poor, restless sleep, could not get going, can't keep mind on tasks, everything an effort, bothered by things, and talked less than usual; $\alpha = .73$), and IP (people dislike me and people were unfriendly; $\alpha = .63$). These subscales have evidenced good reliability and validity in prior research (Leventhal et al., 2008; Pettit et al., 2008). A total score was computed using the sum of all 20 items.

Smoking Initiation

One item from the Youth Risk Behavior Surveillance Survey (Brener et al., 2004) was utilized to assess smoking experimentation: "Have you ever tried cigarette smoking, even a few puffs?" (Yes/No). If participants endorsed that item or reported any smoking in the past thirty days, they were coded as having initiated smoking.

Data Analysis

Initial analyses of sample characteristics were performed using SAS (SAS Institute Inc., 2003). Genetic analyses were conducted with participant-level data using Mx Software (Neale, Boker, Xie, & Maes, 2004). Smoking initiation was a dichotomous variable. Because the distributions of CESD subscale scores were skewed, we categorized participants following the standard scoring procedure for the CESD such that participants scoring 16 or higher on the total score were classified as having depressive symptoms (Radloff, 1977). We translated this approach for the subscales by assuming that a mean score of 0.8 per item (16/20) or higher represents presence of depressive symptoms for that particular subscale (this was reversed for PA). Genetic modeling employed a threshold model, assuming that a continuous liability distribution underlies ordinal variables (i.e., 0 = *Never smoked/Below CESD cutoff*, 1 = *Have initiated smoking /Above CESD cutoff*). This addresses the skewness of raw data, retains the statistical advantages conferred by normality assumptions, and recovers underlying correlations and parameter estimates (Derks, Dolan, & Boomsma, 2004; Stallings et al., 2001). Thresholds accounting for age and sex trends for each variable for every participant were used as definition variables in all analyses to account for the fact that not all respondents have passed through the age of risk and that prevalence for depression and smoking initiation varies by sex.

Biometric genetic modeling assumes that the variance in a given phenotype is due to additive genetic effects (denoted A), shared (family) environmental (denoted C), and nonshared environmental (denoted E) influences. It takes advantage of the differing genetic relationship between MZ and DZ pairs, with MZ pairs sharing 100% of their genes and DZ pairs, on average, sharing 50% of their genes identical by descent (Neale & Cardon, 1992; Plomin, DeFries, McClearn, & McGuffin, 2008).

We conducted bivariate analyses examining the association between each CESD scale and smoking initiation using Cholesky bivariate decomposition models. We decomposed the variance of smoking initiation into genetic and environmental influences common with depressive symptom dimensions and genetic and environmental influences that are unique to smoking initiation (see Figure 1). To clarify the terms, "shared" and "nonshared" refer to putative environmental factors that lead to similarity or dissimilarity between twin pairs, respectively, whereas the terms "common" and "unique" refer to variance components that are mutual or distinct across the two phenotypes (i.e., depression and smoking), respectively. Model comparisons were conducted by dropping each genetic and environmental path in the Cholesky decomposition model presented in Figure 1 (with the exception of the nonshared environmental influences on depression and the nonshared environmental influences unique to smoking initiation, as dropping these would assume no measurement error). Separate models were tested for each depressive symptom dimension and the CESD total score.

For descriptive purposes, the fit of each model was illustrated by reporting the −2 times log likelihood fit function (−2LL) and the Akaike's information criterion (AIC), with lower −2LL relative to its *df* and lower AIC indicating better fit. For primary determinations of model fit regarding whether a parameter is statistically significant, comparisons of the fit of each reduced

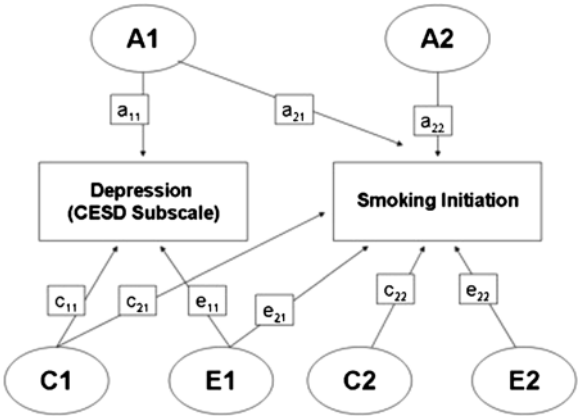

Figure 1. Bivariate Cholesky decomposition model. A_1, additive genetic effects common to depressive symptom dimension and smoking initiation; A_2, additive genetic effects unique to depressive symptom dimension; C_1, shared environmental effects common to depressive symptom dimension and smoking initiation; C_2, shared environmental effects unique to smoking initiation; E_1, nonshared environmental effects common to depressive symptom dimension and smoking initiation; and E_2, nonshared environmental effects unique to smoking initiation.

model with the corresponding full model were carried out using the χ^2 comparison test. A significant difference between a reduced model and the full model ($p < .05$) indicates that the parameter dropped from the full model significantly departs from zero (Neale & Cardon, 1999).

Analyses included all twin pairs, and there were 15 participants (both members of 5 twin pairs and 5 singletons from 5 twin pairs) with some missing phenotype data. Mx implements the full-information maximum likelihood method to address missing data (Neale, Boker, Xie, & Maes, 2004).

Results

Sample Characteristics

The sample was 51.6% female and had an average age of 12.2 (*SD* = 1.93, range 9–16) years. The descriptive statistics of CESD continuous raw scores (before they were categorized) were NA (*M* [*SD*] = 0.29 [0.48], median = 0, range 0–3.0, 53.5% scored 0 indicating no NA), SF (*M* [*SD*] = 0.45 [0.50], median = 0.29, range 0–2.86, 29.4% scored 0), PA (*M* [*SD*] =1.72 [1.00], median = 1.75, range 1.00–3.00, 18% scored 3.0 indicating highest possible level of PA), and IP (*M* [*SD*] = 0.31 [0.60], median = 0, range 0–3.00, 70.0% scored 0) and total (*M* [*SD*] = 10.9 [8.39], median = 10, range 0–56, 7.2% scored 0). The prevalence of participants who initiated smoking and scored above the cutoff on each depressive symptom dimension by gender and age is reported in Table 1. Of those who initiated smoking (*n* = 170, 14%), 43 (26%) participants reported being a daily smoker at some point in their life and 95 (56%) had at least one cigarette in the 30 days prior to the assessment.

Phenotypic and Genetic Correlations

Within-twin phenotypic correlations between CESD subscales and smoking initiation were statistically significant for each symptom dimension (see Table 2). The absolute magnitude of correlations with smoking initiation was significantly larger for CESD total, NA, SF, and IP than for PA as evidenced by nonoverlapping confidence intervals (*CI*s).

Cross-twin within-trait correlations were higher among MZ than among DZ twins for CESD total, NA, SF, and IP (see Table 3). However, the DZ correlation was higher than the MZ correlation for smoking initiation and PA (Table 3). The cross-trait cross-twin correlations, also presented in Table 3, suggest higher MZ than DZ correlations.

Bivariate Model Fitting

The standardized path coefficients from each Cholesky model are presented in Figure 2. Table 4 presents the magnitude of additive genetic (A), shared environmental (C), and nonshared environmental (E) influences on each CESD subscale and smoking initiation as well as their influence on depression–smoking covariance. These estimates were derived by squaring the path coefficients presented in Figure 2. Smoking initiation variance was decomposed into genetic, shared environmental, and nonshared environmental influences: (a) in common with depression and (b) unique to smoking initiation apart from depression. Results from the full model were interpreted.

Regarding influences on the individual phenotypes (apart from influences on cross-trait covariation), bivariate models illustrated significant genetic (a_{11}) effects on CESD total score, shared environment (c_{11}) effects on NA and PA, and unique shared environment (c_{22}) effects on smoking initiation apart from PA (see Table 4 for variance estimates). No other familial influences on the individual phenotypes (apart from those accounting for smoking–depression covariance) were significant.

Common genetic (a_{21}) and shared environmental (c_{21}) influences between smoking initiation and total, NA, SF, PA, and IP could not be dropped simultaneously without a significant deterioration in fit (see Table 5), suggesting that there were significant common familial influences (that result from either genetic sources or shared environment) on the relation between smoking initiation and depression. For total, SF, and IP, there was not sufficient power to distinguish whether these common familial influences were genetic (a_{21}) or shared environmental (c_{21}) effects as dropping the individual paths one at a time did not significantly alter model fit. For the relations of NA and PA to smoking initiation, there were statistically significant common shared environmental influences (c_{21}), but common genetic (a_{21}) and common nonshared environmental (e_{21}) influences were not significant. Also, common nonshared environmental influences (e_{21}) significantly explained part of the covariation of smoking initiation with IP and total.

Across the models, there were several instances in which paths were equal to zero. Additional models dropping these paths yielded identical parameters for the path estimates (with expected changes in *df*) as those found in the full model. Therefore, we report path estimates for only the full models in Table 4 and Figure 2 for ease of interpretation across subscales.

Discussion

This study found that four different symptom dimensions of depression were significantly associated with smoking initiation

Table 1. Prevalence of Participants Who Initiated Smoking and Scored Above the Cutoff on Depressive Symptom Dimensions by Gender and Age

		Female		Age		Smoking initiation,
	N (%)	%	χ^{2a}	*M* (*SD*)	*F*[b]	yes (%)
Smoking initiation	–	–	36.9**	–	ns	
No	1,034 (85.9)	55.1		12.1 (1.7)		0
Yes	170 (14.1)	30.0		12.3 (2.0)		100
CESD dimensions						
Negative affect	–	–	ns	–	ns	
Below	1,040 (87.1)	52.6		12.2 (1.9)		9.6
Above	154 (12.9)	46.1		12.0 (2.0)		45.6
Somatic features	–	–	ns	–	ns	
Below	961 (80.4)	52.8		12.2 (1.9)		9.3
Above	234 (19.6)	47.4		12.1 (2.0)		34.6
Positive affect[c]	–	–	ns	–	10.5**	
Below	508 (42.4)	54.5		12.4 (1.9)		8.3
Above	689 (57.6)	49.6		12.0 (1.9)		18.9
Interpersonal problems	–	–	ns	–	ns	
Below	995 (83.5)	52.3		12.2 (1.9)		9.9
Above	196 (16.5)	49.4		12.0 (2.0)		35.7
Total score			4.7*		ns	
Below	938 (78.9)	53.5		12.2 (1.9)		8.0
Above	251 (21.1)	45.8		12.0 (2.0)		36.7

Note. $N = 1{,}204$ (602 twin pairs), though samples vary across phenotypes due to missing data, Ns = 1,191–1,204. CESD cutoff ≥ .8 per item. CESD = Center for Epidemiological Studies Depression Scale; ns = nonsignificant.

[a]Test for differences in gender across groups.

[b]Test for differences in age across groups.

[c]Scores reflected such that scoring above the cutoff reflects lower PA (and greater PA-related depressive symptoms).

*$p < .05$. **$p < .01$.

among Chinese adolescents. Furthermore, the magnitude of these correlations differed across symptom dimensions (i.e., more robust correlations for NA, SF, and IP than for PA). These findings are consistent with prior results in U.S. and European adult samples documenting that different symptom dimensions of depression exhibit disparate patterns of associations with smoking status and other smoking variables (Korhonen et al., 2011; Leventhal et al., 2008; Mickens et al., 2011; Pomerleau et al., 2003) and extends these results to a marker of smoking with a very low threshold and a sample of Chinese adolescents.

Concerning sources of variation within the individual phenotypes, bivariate twin modeling illustrated significant genetic effects on overall depressive symptoms (55% variance), shared environment effects on NA (36%) and PA (53%), and unique shared environment effects on smoking initiation (46%) apart

Table 2. Within-Twin Phenotypic Tetrachoric Correlations of Depressive Symptom Dimensions and Smoking Initiation (95% *CI*)

Variable	1	2	3	4	5
1. Smoking initiation					
2. CESD-NA	.609 (.510–.694)				
3. CESD-SF	.514 (.412–.605)	.882 (.836–.917)			
4. CESD-PA[a]	.291 (.179–.396)	.407 (.297–.508)	.275 (.173–.371)		
5. CESD-IP	.513 (.408–.607)	.807 (.744–.858)	.735 (.665–.794)	.384 (.281–.480)	
6. CESD-Total	.579 (.485–.662)	.941 (.910–.941)	.894 (.856–.924)	.578 (.493–.654)	.845 (.795–.886)

Note. 321 MZ pairs and 281 DZ pairs. CESD = Center for Epidemiological Studies Depression Scale (dichotomized ≥ .8 per item); DZ = dizygotic twins; IP = interpersonal problems subscale; MZ = monozygotic twins; NA = negative affect subscale; PA = positive affect subscale; SF = somatic features subscale.

[a]Scores reflected such that scoring above the cutoff reflects lower PA (and greater PA-related depressive symptoms).

Table 3. Cross-Twin Monozygotic (MZ) and Dizygotic (DZ) Tetrachoric Correlations of Smoking Initiation and Depressive Symptom Dimensions (95% *CI*)

	MZ twins	DZ twins
Within-trait		
SI	.562 (.354–.725)	.582 (.355–.754)
CESD-NA	.568 (.374–.723)	.434 (.147–.661)
CESD-SF	.507 (.325–.660)	.365 (.132–.566)
CESD-PA	.474 (.319–.608)	.592 (.440–.716)
CESD-IP	.414 (.199–.598)	.298 (.052–.516)
CESD-total	.610 (.445–.742)	.271 (.044–.475)
Cross-trait		
SI × CESD-NA	.567 (.428–.685)	.302 (.098–.484)
SI × CESD-SF	.454 (.306–.584)	.253 (.070–.422)
SI × CESD-PA[a]	.378 (.231–.510)	.067 (-.102–.234)
SI × CESD-IP	.338 (.171–.490)	.198 (.011–.374)
SI × CESD-tot	.454 (.306–.585)	.200 (.021–.369)

Note. CESD = Center for Epidemiological Studies Depression Scale (dichotomized ≥ .8 per item); DZ = dizygotic twins; IP = interpersonal problems subscale; MZ = monozygotic twins; NA = negative affect subscale; PA = positive affect subscale; SF = somatic features subscale; SI = smoking initiation; tot = total score.

[a]Scores reflected such that scoring above the cutoff reflects lower PA (and greater PA-related depressive symptoms).

from PA. No other familial influences on the individual phenotypes (apart from those accounting for smoking–depression covariance) were statistically significant.

Influences on the covariation between depressive symptoms and smoking initiation differed across the symptom dimensions. For both PA and NA, there was a significant effect of common shared environmental influences on depression–smoking covariation and a nonsignificant effect of genetic influences. Thus, it is possible this relation could be accounted for by environmental risk factors shared between twins (e.g., parenting factors, family-wide stressors, being a member of the same peer group) that may collectively drive affect disturbance and increase liability for smoking initiation. For instance, parental influences, such as behavioral modeling of smoking and depressogenic behavior as well as poor parenting practices, may increase their offspring's propensity to experiment with smoking and develop affective dysregulation (Alloy et al., 2001; White, Johnson, & Buyske, 2000). In addition, family-wide stressors, such as coping with poverty, may impact offspring risk of smoking initiation and emotional disturbance (Repetti, Taylor, & Seeman, 2002).

Part of the relation of IP and overall depressive symptom level to smoking initiation was significantly accounted for by common nonshared environmental factors. This result is consistent with, although not necessarily evidence for, a direct causal model whereby depressive symptoms increase risk of smoking initiation (or vice versa). These results concord with past studies of U.S. and Finnish adolescent twins, which found that nonshared environmental factors affected smoking–depression relations in some groups (McCaffery et al., 2008; Sihvola et al., 2008), and suggests that IP-related depressive symptoms may be particularly important to this pattern.

For overall depressive symptoms, SF, and IP, there was evidence that common familial (either genetic or shared environmental) factors accounted for part of the covariation with smoking initiation. Attempts to disentangle these two sources of covariation were unsuccessful as dropping the individual paths did not significantly diminish model fit, which suggests insufficient statistical power. Indeed, post-hoc power analyses illustrated that, given the current effect sizes, we would need a larger sample to have 0.8 power detect significant genetic influences on covariation between these two symptom dimensions and smoking initiation (SF: required N = 1,319 twin pairs and IP: N = 6,199 twin pairs). Similar analyses examining the sample required to detect significant shared environment influences on

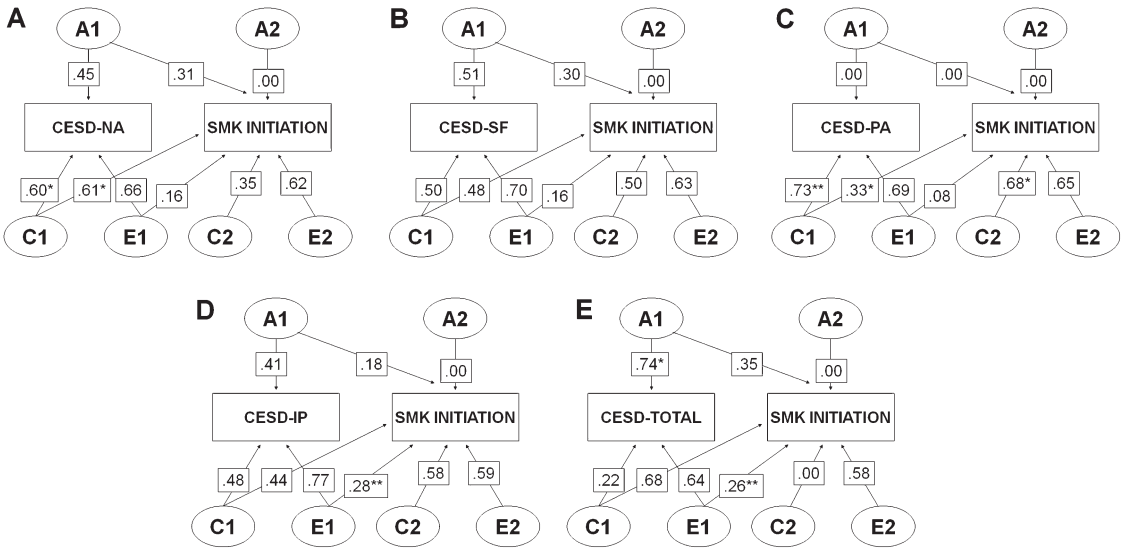

Figure 2. Bivariate Cholesky decomposition model results for the covariance between smoking initiation and negative affect (NA; A), somatic features (SF; B), positive affect (PA; C), interpersonal problems (IP; D), and total score (E). *Note.* *$p < .05$. **$p < .01$.

Table 4. Magnitude of Additive Genetic (A), Shared Environmental (C), and Nonshared Environmental (E) Variances Contributing to Depression, Smoking Initiation, and the Covariance Between Depressive Symptom Dimensions and Smoking Initiation

		Smoking initiation		
	Depressive symptom dimension total	Shared in common with depressive symptom dimension	Unique to smoking initiation	Covariance between depressive symptom dimension and smoking initiation
CESD-NA				
Additive genetic (A)	.20	.10	.00	.14
Shared environment (C)	.36	.37	.12	.37
Unique environment (E)	.44	.03	.38	.11
CESD-SF				
Additive genetic (A)	.26	.09	.00	.15
Shared environment (C)	.25	.23	.25	.24
Unique environment (E)	.49	.03	.40	.12
CESD-PA[a]				
Additive genetic (A)	.00	.00	.00	.00
Shared environment (C)	.53	.11	.46	.24
Unique environment (E)	.47	.01	.42	.06
CESD-IP				
Additive genetic (A)	.17	.03	.00	.08
Shared environment (C)	.23	.20	.34	.21
Unique environment (E)	.60	.08	.35	.22
CESD-Total				
Additive genetic (A)	.55	.12	.00	.26
Shared environment (C)	.05	.47	.00	.15
Unique environment (E)	.40	.07	.34	.17

Note. 321 MZ pairs and 281 DZ pairs. Separate analyses were performed for each depressive symptom dimension. CESD = Center for Epidemiological Studies Depression Scale (dichotomized ≥ .8 per item); DZ = dizygotic twins; IP = interpersonal problems subscale; MZ = monozygotic twins; NA = negative affect subscale; PA = positive affect subscale; SF = somatic features subscale.

[a]Scores reflected such that scoring above the cutoff reflects lower PA (and greater PA-related depressive symptoms).

the covariance between these two symptom dimensions and smoking initiation with 0.8 power and the current effect sizes suggested that our sample size was adequate (SF: required N = 449 twin pairs and IP: N = 639 twin pairs). Though we cannot isolate the source of familial influence, these findings suggest that there are indeed significant common familial influences on the cross-trait associations between smoking initiation and these three symptom dimensions.

This study should be considered in light of its strengths and limitations. Because there was limited prevalence of smoking in the sample, we could not analyze markers of smoking heaviness, dependence, and persistence, which is important given that these phenotypes may have strong genetic loadings (Lessov-Schlaggar, Pergadia, Khroyan, & Swan, 2008). In addition, depression assessment focused on current mood state only. It is possible that some individuals may have had a past depressive episode that remitted prior to the study, and these individuals would be categorized as not having the phenotype. Similarly, due to the age of the sample, some of the participants may not have fully progressed through their period of risk for smoking initiation and depressive symptoms.

An important limitation was that this sample was too small to explore moderation by gender and age. Gender differences should be studied in future research, especially given the finding of McCaffery et al. (2008) of common genetic influences between smoking and depression only in female U.S. adolescents and because of social sanctions against smoking among girls in China that could impact expression of genetic liability to smoking initiation. Age differences should also be studied across adolescence as depression–smoking covariation may differ later in adolescence given neurodevelopmental and socioenviornmental factors that change across the teenage years. Indeed, the level of depressive symptoms was low in comparison with older previous samples of adolescents from China and Hong Kong (Chou, 1999; Okamoto et al., 2010), with sizeable proportion of participants in the current sample reporting no symptoms. Thus, it is not clear whether these findings will generalize to variability at the higher end of the depressive severity continuum and to samples of older adolescents. Finally, the concomitant role of age and gender should also be addressed in future work in Chinese adolescents. Indeed, there were relatively high rates of smoking in girls in our sample, which may reflect a trend of rising prevalence of smoking among young Chinese females, particularly due to tobacco company advertising targeting young Chinese females in the previous decade (Okamoto et al., 2010; Samet & Yoon, 2001). However, longitudinal modeling or larger cross-sectional designs are required to explore

Table 5. Model Fitting Results From Bivariate Cholesky Decompositions Models of Depressive Symptom Dimensions and Smoking Initiation

	Model fit			Comparison with full model		
Model	−2ll	*df*	AIC	$\Delta\chi^2$	*df*	*p* value
CESD-NA						
Full	1,666.5	2,391	−3,115.5	N/A	N/A	N/A
Drop a_{11}	1,667.2	2,392	−3,116.8	0.70	1	.40
Drop a_{21}	1,667.2	2,392	−3,116.8	0.70	1	.40
Drop a_{22}	1,666.5	2,392	−3,117.5	0.00	1	1.0
Drop c_{11}	1,670.8	2,392	−3,113.2	4.30	1	.04
Drop c_{21}	1,670.8	2,392	−3,113.2	4.30	1	.04
Drop c_{22}	1,666.9	2,392	−3,117.1	0.40	1	.53
Drop e_{21}	1,669.8	2,392	−3,114.2	3.30	1	.07
Drop a_{21}, c_{21}	1,723.6	2,393	−3,062.4	57.1	2	<.0001
Drop a_{21}, c_{21}, e_{21}	1,773.5	2,394	−3,014.5	107.0	3	<.0001
CESD-SF						
Full	1,964.5	2,392	−2,819.5	N/A	N/A	N/A
Drop a_{11}	1,965.6	2,393	−2,820.4	1.10	1	.29
Drop a_{21}	1,965.4	2,393	−2,820.6	0.90	1	.34
Drop a_{22}	1,964.5	2,393	−2,821.6	0.00	1	1.0
Drop c_{11}	1,966.7	2,393	−2,819.3	2.20	1	.14
Drop c_{21}	1,966.7	2,393	−2,819.3	2.20	1	.14
Drop c_{22}	1,965.0	2,393	−2,821.0	0.50	1	.48
Drop e_{21}	1,967.9	2,393	−2,818.1	3.40	1	.07
Drop a_{21}, c_{21}	1,998.2	2,394	−2,789.8	33.7	2	<.0001
Drop a_{21}, c_{21}, e_{21}	2,040.5	2,395	−2,749.5	76.0	3	<.0001
CESD-PA[a]						
Full	2,413.6	2,394	−2,374.4	N/A	N/A	N/A
Drop a_{11}	2,413.6	2,395	−2,376.4	0.00	1	1.0
Drop a_{21}	2,413.6	2,395	−2,376.4	0.00	1	1.0
Drop a_{22}	2,413.6	2,395	−2,376.4	0.00	1	1.0
Drop c_{11}	2,427.5	2,395	−2,362.5	13.9	1	<.001
Drop c_{21}	2,417.3	2,395	−2,372.7	3.70	1	.05
Drop c_{22}	2,419.6	2,395	−2,370.4	6.00	1	.01
Drop e_{21}	2,414.5	2,395	−2,375.5	0.90	1	.34
Drop a_{21}, c_{21}	2,428.9	2,396	−2,363.1	15.3	2	<.001
Drop a_{21}, c_{21}, e_{21}	2,439.4	2,397	−2,354.6	25.8	3	<.0001
CESD-IP						
Full	1,870.1	2,388	−2,905.9	N/A	N/A	N/A
Drop a_{11}	1,870.4	2,389	−2,907.6	0.30	1	.58
Drop a_{21}	1,870.3	2,389	−2,907.7	0.20	1	.65
Drop a_{22}	1,870.1	2,389	−2,907.9	0.00	1	1.0
Drop c_{11}	1,871.7	2,389	−2,906.3	1.60	1	.21
Drop c_{21}	1,871.7	2,389	−2,906.3	1.60	1	.21
Drop c_{22}	1,870.6	2,389	−2,907.4	0.50	1	.48
Drop e_{21}	1,880.4	2,389	−2,897.6	10.3	1	<.01
Drop a_{21}, c_{21}	1,887.2	2,390	−2,892.8	17.1	2	<.001
Drop a_{21}, c_{21}, e_{21}	1,939.3	2,391	−2,842.7	69.2	3	<.0001
CESD-Total						
Full	1,965.8	2,386	−2,806.2	N/A	N/A	N/A
Drop a_{11}	1,971.3	2,387	−2,802.7	5.50	1	.02
Drop a_{21}	1,968.3	2,387	−2,805.8	2.50	1	.11
Drop a_{22}	1,965.8	2,387	−2,808.2	0.00	1	1.0
Drop c_{11}	1,966.9	2,387	−2,807.1	1.10	1	.29
Drop c_{21}	1,966.9	2,387	−2,807.1	1.10	1	.29
Drop c_{22}	1,965.8	2,387	−2,808.2	0.00	1	1.0

Table 5. Continued

Table 5. Continued

Model	Model fit −2ll	*df*	AIC	Comparison with full model $\Delta\chi^2$	*df*	*p* value
Drop e_{21}	1,975.1	2,387	−2,798.9	9.30	1	<.01
Drop a_{21}, c_{21}	2,002.2	2,388	−2,773.8	36.4	2	<.0001
Drop a_{21}, c_{21}, e_{21}	2,068.1	2,389	−2,709.9	102.3	3	<.0001

Note. 321 MZ pairs and 281 DZ pairs. See Figure 1 for a depiction of which paths correspond to a_{11} through e_{21}. Separate sets of models were performed for each depressive symptom dimension. AIC = Akaike's information criterion; CESD = Center for Epidemiological Studies Depression Scale (dichotomized ≥ .8 per item); DZ = dizygotic twins; IP = interpersonal problems subscale; MZ = monozygotic twins; NA = negative affect subscale; N/A = Not Applicable; PA = positive affect subscale; SF = somatic features subscale.

[a]Scores reflected such that scoring above the cutoff reflects lower PA (and greater PA-related depressive symptoms).

genetic and environmental influences on depression–smoking covariation in adolescent girls across specific age groups.

Limitations notwithstanding, this study yielded novel information, suggesting that the associations between individual depressive symptom dimensions and smoking initiation extend to Chinese adolescents, and both familial and nonfamilial factors may play a role in their covariation. Furthermore, the results suggest that influences on this association may vary across different depression symptom dimensions. Thus, it may be fruitful for future research to parse depressive symptoms into component subdimensions to avoid failing to model important phenotypic heterogeneity in depression that may differentially impact smoking behavior.

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Declaration of Interests

None declared.

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