Gene-Environment Transactions between Peer Tobacco Use, Parental Supervision, and Chinese Adolescent Cigarette Smoking Initiation

By

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Abstract

Following 602 Chinese twin pairs (52% female) from early to middle adolescence at two time points (*M*age = 12 and 15), this study examined gene-environment interplay between perceived parental supervision, peer tobacco use, and adolescent cigarette smoking initiation. From early to middle adolescence, genetic influences on cigarette smoking initiation became more pronounced, whereas shared environmental influences that promote similarity between family members diminished. Genetic factors primarily explained the links between parental supervision and cigarette smoking initiation in mid-adolescence. Peer tobacco use displayed stronger associations with and moderating potential in adolescent cigarette smoking initiation than parental supervision. High levels of peer tobacco use amplified genetic risk for cigarette smoking initiation in mid-adolescence. The patterns of gene-environment interplay between peer processes and Chinese adolescent cigarette smoking initiation are dynamic throughout adolescence.

Preface

This thesis is an original work by Zachary Meyer. The research projects, of which this thesis is a part, received research ethics approval from the University of Alberta Research Ethics Board (Protocol 00115717)

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Introduction

Tobacco use is a major source of preventable disease and death, putting considerable financial strain on healthcare systems globally (Goodchild et al., 2018). Tobacco use during adolescence can exacerbate the risk for continued tobacco use and abuse of other substances (e.g., alcohol, cocaine; Silveira et al., 2018). In addition, adolescents are at heightened risk for tobacco experimentation, given the confluence of biological, social, and cognitive changes that occur during this developmental period (Jamner et al., 2003). Therefore, a comprehensive understanding of the aetiologies and mechanisms contributing to adolescent initiation and use of tobacco products is essential to inform prevention efforts. Cumulative evidence has demonstrated genetic and environmental aetiologies of adolescent tobacco use (Dick et al., 2007a; Maes et al., 2004). However, scant research has simultaneously investigated how peer substance use and parenting practices—two major sources of socialization and antecedents of adolescent substance use—are implicated in the dynamic processes of gene-environment transactions involved in the development of adolescent cigarette smoking initiation, especially in ethnically/racially diverse populations (Dick, 2011; Zheng et al., 2023). The present study sought to elucidate the gene-environment transaction mechanisms linking parental supervision and peer tobacco use with Chinese adolescent cigarette smoking initiation in a large sample of Chinese adolescent twins followed prospectively from early to middle adolescence.

Peer and Parental Influences on the Development of Adolescent Tobacco Use

Developmental approaches play a crucial role in identifying risk and protective factors that impact the development of adolescent initiation and use of tobacco products (Bricker et al., 2009; Hemphill et al., 2010). Longitudinal research can illustrate how the influences of specific factors that contribute to the onset and development of tobacco use manifest over time. Behavioral

genetic research has robustly shown that genetic liabilities and environmental exposures constitute substantial risk factors for tobacco use throughout adolescence (Dick et al., 2009; Maes et al., 2004). Notably, developmental behavioral genetic studies have demonstrated that genes play an increasingly important role in tobacco use from adolescence into young adulthood, while the contributions of shared environment (common experiences shared across family members that promote similarity) generally decrease (Do et al., 2015; Kendler et al., 2008; Maes et al., 2017). Identifying specific and modifiable environmental exposures that could alter genetic and environmental influences on adolescent cigarette use is vital for further understanding its development and informing prevention programs.

Affiliation with substance-using peers is robustly linked with adolescent tobacco use (Cambron et al., 2018; Liu et al., 2017). Peer influence/socialization and selection constitute two primary ways peer affiliation could influence adolescent tobacco use. Adolescents may adopt favorable attitudes towards substance use and use norms to achieve social acceptance (i.e., socialization) or seek out like-minded peers to reinforce their behaviors (i.e., selection; Hoffman et al., 2006; Simons-Morton & Farhat, 2010). Having peers who engage in deviant behaviors may also facilitate increased tobacco access while creating more experimentation opportunities (Sun et al., 2021). The influence of these peer processes may differ across cultural norms. For instance, studies using Western samples typically reveal peer selection in tobacco use without peer influence/socialization (Kiuru et al., 2010), whereas a recent study reveals peer influence/socialization in Chinese adolescent tobacco use without peer selection (DeLay et al., 2023). Furthermore, research has consistently linked low parental supervision (e.g., knowledge, active regulation, and control of adolescent activities) with adolescent tobacco use (Blustein et al., 2015; Van Ryzin et al., 2012). As the importance of peer influences increases throughout

adolescence, high parental supervision can potentially buffer against pro-use attitudes promoted through association with substance-using peers (Hemovich et al., 2011). Parental efforts to regulate friend groups, prevent unsupervised activities, and establish stricter expectations and rules around substance use may reduce the likelihood of adolescents accessing tobacco products in the first place (Tornay et al., 2013). Thus, peer affiliation and parental supervision are two pertinent candidate correlates with adolescent tobacco use and warrant investigations into the gene-environment transaction mechanisms involved in their influences on adolescent cigarette smoking initiation.

Gene-Environment Interplay between Parental Supervision, Peer and Adolescent Tobacco Use

Gene-environment correlations (rGE) can partly explain the links between adolescent tobacco use, peer tobacco use, and parental supervision. For instance, tobacco-using parents may also show low parental supervision and pass their smoking-inclined genes to their offspring (i.e., passive rGE). Adolescents genetically at risk for tobacco use could actively seek out peers who also smoke (i.e., active rGE; Knafo & Jaffee, 2013). Genetically influenced smoking behaviors may elicit varying parenting responses, prompting more lenient or stricter parental monitoring (i.e., evocative rGE; Elam et al., 2017). Twin studies have revealed common genetic factors between adolescent tobacco use and affiliation with tobacco-using peers, suggesting the presence of active or evocative rGE (Cleveland et al., 2005; Harden et al., 2008; Wills & Carey, 2013). Molecular genetic research has shown that polygenic risk scores (PGS) for behavioral problems in childhood predicted lower parental monitoring and subsequently increased affiliation with tobacco-using peers, supporting evocative rGE (Elam et al., 2017). Scarce research has explored rGE between parenting practices and adolescent cigarette use, although relatively more studies

have focused on adolescent alcohol use (e.g., Zheng et al., 2021, 2023). Pertinent research has similarly revealed common genetic as well as common non-shared environmental (unique experiences promoting dissimilarity) factors between parenting practices (e.g., parental knowledge, warmth, and autonomy permissiveness) and adolescent alcohol use (Latendresse et al., 2010). Additional efforts are needed to further elucidate the rGEs between adolescent cigarette use, parental supervision, and peer tobacco use.

Besides rGE, environmental experiences and exposures could also potentially modify genetic and environmental contributions to adolescent substance use, termed gene-environment interaction (G×E). The diathesis-stress model posits that individuals with genetic vulnerabilities are more likely to develop problem behaviors in adverse environments; a benign environment can buffer or suppress genetic risk for psychopathology (Monroe & Simons, 1991). Accordingly, twin research has shown that higher levels of parental knowledge can suppress the genetic risk for and amplify shared environmental influences on adolescent smoking in Finnish twins at ages 14 and 17 (Dick et al., 2007a, 2007b), and that parent–child relationship problems (high conflict and low involvement) amplify genetic influences on child externalizing problems including nicotine dependence in 17-year-old American twins (Hicks et al., 2009). Moreover, peer tobacco use amplifies the genetic risk for U.S. adolescent substance use (Harden et al., 2008). Examinations of PGS for tobacco use trajectories in 12–21-year-old Black adolescents suggest G×E between elevated polygenic risk and low environmental risk characterized by moderate parental monitoring and low prevalence of substance-using peers, which can increase the likelihood of following lower tobacco use trajectories (Musci et al., 2015). Additionally, the influences of PGS on adolescent externalizing disorders (encompassing substance use but not tobacco) were more prominent at low levels of parental monitoring and high levels of peer

substance use (Salvatore et al., 2015). Collectively, extant findings consistently implicate parental and peer factors involved in gene-environment influences on adolescent substance use, including cigarettes.

Many of the aforementioned studies are cross-sectional. Genetically informed longitudinal studies have demonstrated that genetic and environmental influences, as well as G×E patterns involved in problem behaviors (e.g., alcohol use, conduct problems), can vary throughout development (Burt, 2015; Dick, 2011; Zheng et al., 2023) and across distinct developmental trajectories (e.g., low vs. increasing alcohol use; Zheng et al., 2019, 2021). For example, peer alcohol use amplifies genetic influences on Finnish adolescent alcohol use at age 17 but not at age 14 (Dick et al., 2007a). Parental supervision suppresses genetic risks for Chinese adolescent alcohol initiation only in middle adolescence but not in early adolescence (Zheng et al., 2023). Such findings highlight the importance of considering how gene-environment contributions to adolescent tobacco use could change during development. Moreover, previous studies have separately explored the contributions of parenting practices (Dick et al., 2007b) and peer tobacco use (Harden et al., 2008). No study has concurrently explored their relative contributions to adolescent tobacco use while implementing a longitudinal design. However, relevant studies on adolescent alcohol use have shown a stronger role of peer alcohol use than parenting practices in modulating genetic influences on adolescent alcohol use (Cooke et al., 2015; Dick et al., 2007a; Zheng et al., 2019, 2021, 2023).

Genetic Research on Chinese Tobacco Use

Extant behavioral genetics studies have primarily focused on populations of European descent (Polderman et al., 2015), as have studies on adolescent alcohol and drug use (D'amico et al., 2014). As such, human genetics research has increasingly called for greater diversity initiatives

to address these issues (Sirugo et al., 2019). Including and investigating diverse ethnic/racial populations could further elucidate the mechanisms contributing to tobacco initiation, persistence, and desistence during adolescence under various environmental experiences and broader sociocultural contexts. Differences in the genetic compositions and sociocultural structures between non-European populations and European populations can result in differences in how genetic and environmental factors contribute to the development of substance use in adolescence (Zheng et al., 2023). For example, culture can heavily influence adolescent socialization processes (i.e., parent–child and peer relationships; Chen & French, 2008). Differences in these processes may result in distinct rGE and G×E patterns in other ethnic/racial populations compared with European populations. For instance, recent research found smaller genetic and greater shared environmental influences on attention-deficit/hyperactivity disorder, depression, and anxiety symptoms in Chinese adolescent twins, suggesting different patterns of gene-environment transactions than what is typically found in Western populations (Zheng et al., 2016, 2020).

Tobacco use continues to pose a significant threat to China's public health. With the world's second-largest population of youth (United Nations Children's Fund [UNICEF], 2018), the prevalence of regular tobacco use in mainland China youth is estimated at 8.17% (Xiong et al., 2020), indicating that a sizeable portion of Chinese youth is at risk for developing tobacco-related adverse outcomes. However, scant genetically informed studies have explored genetic and environmental influences on cigarette use in Chinese populations, and the few extant studies primarily focused on adults. For example, Lessov-Schlaggar and colleagues (2006) found substantial genetic (70%) and modest shared environmental (4%) on current smoking in Chinese adult twins (≥ 24 years). Similar heritability estimates (69%) for tobacco initiation have been

found in Chinese adult twins (≥ 18 years; Gao et al., 2010). In contrast, Bao et al. (2016) found moderate genetic influences on smoking (26%) in Chinese adult male twins (≥ 25 years). The only Chinese twin study on adolescent tobacco use found moderate genetic (28%) and shared environmental (34%) influences on lifetime smoking and negligible genetic but substantial shared environmental influences (64%) on past month smoking (Unger et al., 2011).

The Current Study

Low parental supervision and high peer tobacco use are potent risk factors for adolescent cigarette initiation and use. Few genetic studies have explored the relative contributions of genetic and environmental factors and their interplay to the links between peer tobacco use, parenting practices, and adolescent cigarette use, lending to much inconclusive and weaker evidence than those on adolescent alcohol use. Moreover, previous relevant research primarily employed cross-sectional designs that fail to illustrate how gene-environment transactions involved in adolescent cigarette smoking initiation could change across different ages. Most previous research only examined parenting practices or peer tobacco use independently, without jointly considering their influences in the same study. Given the scarcity of studies on Chinese adolescent cigarette smoking initiation, the lack of ethnic/racial diversity in genetic tobacco research, and the ongoing risk tobacco use presents to Chinese youth, additional research addressing these issues is paramount. The current study aimed to address these literature gaps by investigating the gene-environment interplay linking perceived parental supervision and peer tobacco use with Chinese adolescent cigarette smoking initiation. Specifically, rGEs were first examined between adolescent cigarette smoking initiation, parental supervision, and peer tobacco use in a large sample of Chinese twins followed prospectively from early to middle adolescence. Next, it investigated whether parental supervision and peer tobacco use could

modify genetic and environmental contributions (i.e., G×E) to Chinese adolescent cigarette smoking initiation. Based on the pertinent but scant literature, three exploratory hypotheses were formulated: 1) Chinese adolescent cigarette smoking initiation will be heritable, while shared environmental influences may be larger than in Western populations; 2) common genetic factors will partly explain the links between adolescent cigarette smoking initiation, peer tobacco use, and parental supervision (i.e., rGE); and 3) parental supervision and peer tobacco use will modulate genetic influences on cigarette smoking initiation (i.e., G×E), where higher levels of parental supervision will suppress and higher levels of peer tobacco use will amplify genetic influences on adolescent cigarette smoking initiation. This G×E pattern, nonetheless, may change from early to middle adolescence.

Methods

Participants and Procedure

Participants were drawn from the Qingdao Twin Registry (QTR) and recruited via medical records and outreach efforts through schools and the media. The QTR enrolled an estimated 74% of all twins in Qingdao in 2005 (see Duan et al., 2013 for additional information on survey and recruitment methods). DNA testing of blood samples determined the zygosity of twin pairs by examining 16 short tandem repeat markers—a method that produced an over 99.6% likelihood of correct zygosity identification. The current sample consisted of an adolescent cohort (Han ethnicity) that was first contacted and assessed through paper-and-pencil questionnaires in 2006 (n = 1200), followed up between 2007–2008 (n = 1106), and again in 2009 (n = 917). This study used data from 2006 and 2009, given the short interval between the first two assessment periods. At time 1, the sample comprised 602 twin pairs ages 9–16 (M = 12.16 years, SD = 1.93, 51.6% female). In 2006, 59.1% of twins were between 10–12 years and enrolled in elementary school

(grades 4–6). The sex-zygosity breakdown of the twin pairs is as follows: 321 monozygotic (MZ) and 281 dizygotic (DZ) twins (170 female MZ, 151 male MZ, 80 female DZ, 80 male DZ, 119 opposite-sex, 2 unknown sex DZ) pairs (4 unpaired twins at time 1). In 2009 average participant age was 14.99 (*SD* = 1.88, 50.7% female), with 63.1% of participants between 13–15 years and enrolled in middle school (grades 7–9). Adolescent-reported parental monitoring at time 1 did not differ significantly between participants who did or did not participate at time 3, $t(466.64) = -1.70$, $p = .09$, nor did their sex differ significantly, $\chi^2(1) = 2.31$, $p = 13$. However, participants lost over time due to attrition reported significantly higher levels of peer tobacco use at time 1 than those who were retained, $t(462.35) = 3.29$, $p < .01$, were older, $t(473.15) = 5.06$, $p < .05$, and were more likely to report lifetime cigarette smoking initiation at time 1, $\chi^2(1) = 5.50$, $p = .02$.

Parent-reports (55.7% mother completion) indicated that parents in most households were married (96.1%). Regarding educational attainment, 62% had a middle school or lower education, 19.2% had a high school degree, 6.5% graduated from training/vocational school, and 11.3% graduated from college/university. The reported average annual household income was between 5,001–10,000 RMB and 10,001–15,000 RMB, revealing a relatively more rural and below-average socioeconomic sample compared to the provincial 2006 individual income of approximately 12,000 RMB. Informed assents and consent were obtained from participants and their parents respectively. All procedures were approved by the institutional review board at the University of Alberta (Protocol 00115717).

Measures

Cigarette smoking initiation. At each time, participants answered one question "Have you ever tried cigarette smoking, even a few puffs?" (1 = *yes*, 0 = *no*). A binary response option for "Have

you ever tried cigarette smoking" aims to assess participants' current lifetime history of smoking initiation behavior by capturing whether individuals have ever experimented with cigarette smoking up to that point. This measure is appropriate to use across waves as changes in lifetime history from times 1 to 2 (i.e., no use to any use) would indicate a change in cigarette initiation behavior. Capturing a participant's current initiation status allows for investigation into how various factors (e.g., peer use and parental supervision) as well as gene-environment transactions potentially influence initiation status at each time. The term "initiation" was chosen instead of "lifetime use" to avoid confusion with cumulative tobacco usage and to indicate changes in experimentation behavior.

Parental supervision. At each time, participants answered two questions: "Are you allowed to go out with friends that your parents don't know?" (1 = *yes*, 2 = *maybe yes*, 3 = *maybe no*, 4 = *no*) and "How important is it to your parents to know where you are at all times?" (1 = *not important at all,* 2 = *a little bit important,* 3 = *important*, 4 = *very important*). The two items moderately correlated at both times (*rs* = .33 and .27, *p*s < .001). An average score was created each time, with higher values indicating higher levels of parental supervision.

Peer tobacco use. At each time, participants answered two items: "How often are you around people your age who are smoking?" on a 4-point scale (1 = *often,* 2 = *sometimes,* 3 = *hardly ever,* 4 = *never*), and "How many of your good friends smoke cigarettes at least once a month?" on a 4-point scale (1 = *none,* 2 = *a few,* 3 = *most,* 4 = *all*). The first item was reverse coded, and both items were rescaled with 0 = "never/none" and 3 = "often/all." The two items moderately correlated at both times (*rs* = .35 and .45, *p*s< .001). An average score was created, with higher values indicating higher levels of peer tobacco use.

Analytic Strategy

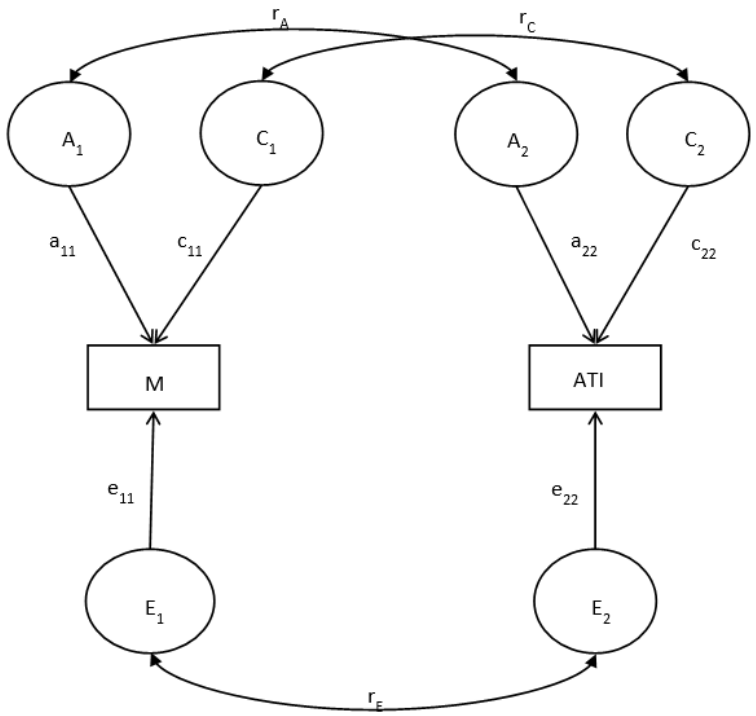
Differences in genetic relatedness between MZ and DZ twins, who share all and half of their segregating genes on average, respectively, are foundational to biometric twin modeling. Univariate biometric modeling separates the variance of a given trait (phenotype) into three distinct sources: additive genetic factors (A) denoting the sum of allelic effects that influence a given phenotype, shared environmental effects (C) constituting common experiences that foster sibling similarities, and non-shared environmental effects (E) corresponding to unique environmental exposures that promote twin dissimilarities and measurement error (Neale & Maes, 2004). The correlation of A between siblings is 1 for MZ twins and .50 for DZ twins, indicating their expected genetic similarity. The correlations between siblings for C and E are 1 and 0, respectively, representing the anticipated associations of shared and unique experiences between siblings regardless of zygosity (Neale & Maes, 2004). For the binary outcome of adolescent cigarette smoking initiation, a biometric liability threshold model was specified, which assumes that the underlying liability for cigarette smoking initiation is normally distributed and decomposes the latent continuous liability for cigarette smoking initiation into A, C, and E components. Preliminary analyses revealed no sex difference in genetic or environmental influences on all variables (i.e., males and females were influenced by the same genetic and environmental factors to the same degree; Neale et al., 2006).

Bivariate biometric modeling compares cross-twin cross-trait correlations across MZ and DZ twin pairs to disentangle the covariance between two phenotypes (e.g., parental supervision and adolescent cigarette smoking initiation) into A, C, and E factors. First, a bivariate model using a correlated-factors approach (Figure 1) where distinct genetic factors influence each variable was used to examine rGE (Maes et al., 2004). Here, r_A denotes the similarity between the two genetic factors, with similar specifications for the shared (r_C) and non-shared (r_E) environmental factors.

To determine the presence of rGE, a significant r_A accompanied by non-zero 95% confidence intervals (CIs) is required.

Figure 1

Bivariate Biometric Model with the Correlated-Factors Approach



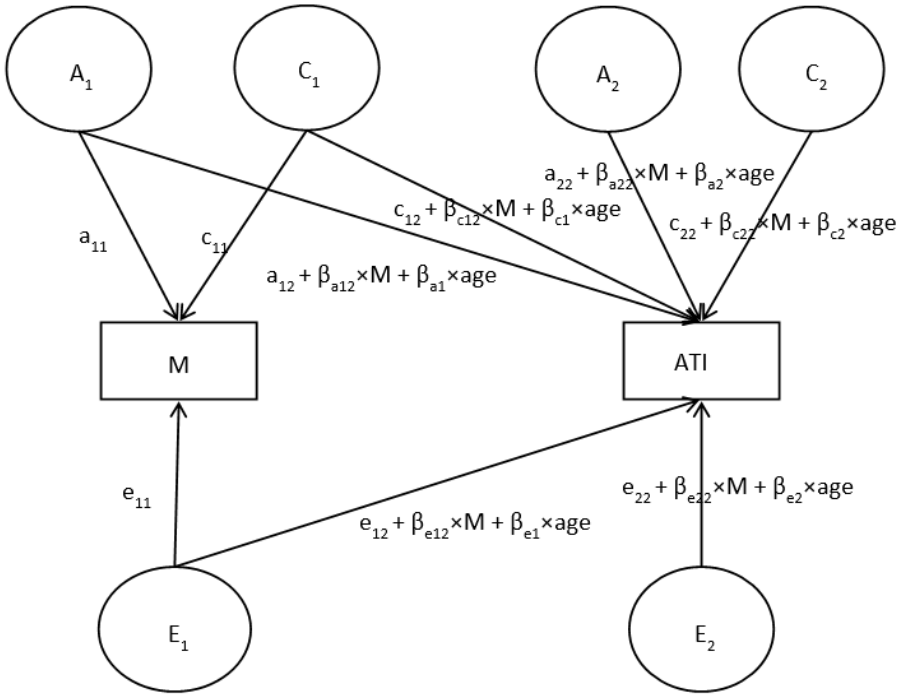
Note. A = additive genetic influences. C = shared environmental influences. E = non-shared environmental influences. M = moderator. ATI = adolescent cigarette smoking initiation.

Bivariate biometric moderation models were used to assess G×E while also controlling for rGE (van der Sluis et al., 2012). A Cholesky decomposition containing common paths between variables (e.g., a_{12}, c_{12}, e_{12}) and unique paths (e.g., a_{11}, c_{11}, e_{11}, a_{22}, c_{22}, e_{22}) was specified to examine the covariance of these variables (Figure 2). Parental supervision and peer tobacco use were independently included as the moderator to evaluate their influences on each of the specified paths to adolescent cigarette smoking initiation (βa_{21}, βc_{21}, βe_{21}, βa_{22}, βc_{22}, βe_{22}). Age was incorporated into each moderation model to control for potential age differences in genetic

and environmental influences on cigarette smoking initiation. All moderators were first z-standardized (with a 0-value indicating group average levels) to facilitate the interpretation of moderation models.

Figure 2

Bivariate Biometric Moderation Model



Note. A = additive genetic influences. C = shared environmental influences. E = non-shared environmental influences. M = moderator. ATI = adolescent cigarette smoking initiation.

The structural equation modeling package OpenMx 2.0 in R 3.4.1 was used to calculate the parameter estimates, 95% CIs, as well as fit indicators (AIC) for each model (Neale et al., 2016). For nested models, the goodness of fit was determined using -2 times the log-likelihood (-2LL). The goodness of fit was compared for competing models via χ^2 tests, wherein the degree of freedom equaled the difference in the number of parameters estimated between a full and nested model. Nested models (more parsimonious ones) were selected when the χ^2 tests were non-significant. AIC additionally assisted model selection, wherein smaller values suggested a better-

fitting model, and missing data were addressed using the full information maximum likelihood (FIML) estimation.

Results

Univariate model fit suggested the full ACE model as the best fit for adolescent cigarette smoking initiation at time 1, while the AE model was the best fit at time 2 (see Table 1). For parental supervision and peer tobacco use, a full ACE model was selected as the best fit at both time points. Adolescent cigarette smoking initiation at time 1 was explained by genetic (29%), shared environmental (35%), and non-shared environmental (36%) factors (see Table 2). In contrast, adolescent cigarette smoking initiation at time 2 was primarily explained by genetic factors (78%), with non-shared environmental influences (22%) explaining the remaining variance as shared environmental influences were negligible. At time 1, parental supervision was primarily explained by non-shared environmental factors (55%), with genetic (29%) and shared environment (15%) factors accounting for the remaining variance. At time 2, parental supervision was again explained mainly by non-shared environmental factors (51%), while the relative contributions of genetic and shared environmental factors switched from time 1 to 10% and 39%, respectively. Peer tobacco use at time 1 was primarily explained by non-shared environmental factors (61%), with the remaining variance explained by genetic (26%) and shared environmental (13%) factors. In contrast, at time 2, it was explained primarily by non-shared and shared environmental (49% and 39%) factors, while genetic factors only explained a modest portion of the variance (12%).

The prevalence of adolescent cigarette smoking initiation (9% vs. 10%) and the levels of parental supervision (*M*s = 3.31 and 3.29) were generally stable from early to middle adolescence (see Table 2). In contrast, peer tobacco use (0 = “never/none” and 3 = “often/all”)

slightly increased from time 1 (.62) to time 2 (.75). Given the wide age range across the adolescent twin cohort at each wave, supplementary univariate analyses were conducted splitting the sample into young and old subgroups to assess whether gene-environment influences varied by age group across each variable at each wave of assessment. Results revealed no substantial differences in gene-environment influences across age subgroups for any variable at either timepoint (Table 3).

Bivariate biometric modeling (Table 4) indicated that adolescent cigarette smoking initiation was positively correlated with peer tobacco use (*r*s = .42 and .45) and negatively with parental supervision (*r*s = -.14 and -.16) at both time points. The links between adolescent cigarette smoking initiation and peer tobacco use appear stronger than with parental supervision at both time points. Furthermore, bivariate modeling suggested that at time 1, common genetic factors predominantly explained the correlation between adolescent cigarette smoking initiation and parental supervision. However, given the wide range of 95% CIs for r_A, r_C, and r_E, these results are inconclusive. The correlation between adolescent cigarette smoking initiation and peer tobacco use was similarly inconclusive at time 1. At time 2, common genetic factors (r_A = -.66) primarily explained the correlation between adolescent cigarette smoking initiation and parental supervision. In contrast, the modest non-shared environmental correlation was not significant (r_E = .14). Like time 1, genetic and environmental contributions to the correlation between adolescent cigarette smoking initiation and peer tobacco use were inconclusive, given the wide ranges of 95% CIs across ACE correlation estimates.

Table 1

Univariate Biometric Model Fit Indices and Model Estimates (95% Confidence Intervals)

	Model	-2LnL (df)	AIC	Comparison Model	$\Delta\chi^2$ (Δdf)	*p*	A	C	E
Time 1 (M_{age} = 12)									
Adolescent cigarette smoking initiation (sex different threshold)									
Saturated model	1	645.91 (1155)	-1664.09	–	–	–	–	–	–
Constrained model	2	659.58 (1166)	-1672.42	1	13.68 (11)	.251	–	–	–
ACE	**3**	**659.58 (1166)**	**-1672.42**	**–**	**–**	**–**	**.29 (.00, .80)**	**.35 (.00, .72)**	**.36 (.19, 1.00)**
AE	4	660.70 (1167)	-1673.30	3	1.12 (1)	.290	.70 (.46, .82)	–	.33 (.18, .54)
CE	5	660.28 (1167)	-1673.72	3	6.35 (1)	.012	–	.58 (.39, .73)	.42 (.27, .61)
Parental supervision									
Saturated model	1	2620.39 (1155)	310.39	–	–	–	–	–	–
Constrained model	2	2636.83 (1176)	284.83	1	16.44 (21)	.744	–	–	–
ACE	**3**	**2636.83 (1176)**	**284.83**	**–**	**–**	**–**	**.29 (.03, .52)**	**.15 (.00, .36)**	**.55 (.47, .65)**
AE	4	2638.67 (1177)	284.67	3	1.83 (1)	.176	.46 (.38, .54)	–	.54 (.46, .62)
CE	5	2641.66 (1177)	287.66	3	4.83 (1)	.028	–	.37 (.30, .44)	.63 (.56, .70)
Peer tobacco use (different ***M*** and ***SD*** across sex)									
Saturated model	1	2424.30 (1152)	120.30	–	–	–	–	–	–
Constrained model	2	2446.73 (1171)	104.73	1	23.44 (19)	.263	–	–	–
ACE	**3**	**2446.73 (1171)**	**104.73**	**–**	**–**	**–**	**.26 (.00, .47)**	**.13 (.00, .36)**	**.61 (.53, .71)**
AE	4	2447.87 (1172)	103.87	3	1.14 (1)	.286	.40 (.32, .48)	–	.60 (.52, .68)
CE	5	2449.97 (1172)	105.97	3	3.24 (1)	.072	–	.33 (.26, .40)	.67 (.60, .74)
Time 2 (M_{age} = 15)									
Adolescent cigarette smoking initiation									
Saturated model	1	515.80 (882)	-1248.20	–	–	–	–	–	–
Constrained model	2	526.37 (893)	-1259.63	1	10.57 (11)	.480	–	–	–
ACE	3	526.37 (893)	-1259.63	–	–	–	.78 (.00, .90)	.00 (.00, 1.00)	.22 (.10, 1.00)
AE	**4**	**526.37 (894)**	**-1261.63**	**3**	**0.00 (1)**	**1.00**	**.78 (.58, .90)**	**–**	**.22 (.10, .42)**
CE	5	531.54 (894)	-1256.46	3	5.17 (1)	.023	–	.62 (.44, .77)	.38 (.23, .56)
Parental supervision									
Saturated model	1	1806.87 (882)	58.87	–	–	–	–	–	–
Constrained model	2	1839.07 (895)	49.07	1	32.20 (21)	.056	–	–	–
ACE	**3**	**1839.07 (895)**	**49.07**	**–**	**–**	**–**	**.10 (.00, .37)**	**.39 (.15, .53)**	**.51 (.43, .60)**
AE	4	1848.50 (896)	56.50	3	9.43 (1)	.000	.52 (.44, .60)	–	.48 (.40, .56)
CE	5	1839.60 (896)	47.60	3	0.53 (1)	.470	–	.47 (.39, .54)	.53 (.46, .61)
Peer tobacco use (different ***M*** and ***SD*** across sex)									
Saturated model	1	1816.30 (875)	66.30	–	–	–	–	–	–
Constrained model	2	1839.51 (894)	51.51	1	23.21 (19)	.228	–	–	–
ACE	**3**	**1839.51 (894)**	**51.51**	**–**	**–**	**–**	**.12 (.00, .38)**	**.39 (.18, .54)**	**.49 (.41, .59)**
AE	4	1849.92 (895)	59.92	3	10.40 (1)	.001	.55 (.46, .62)	–	.45 (.38, .54)
CE	5	1840.33 (895)	50.33	3	0.82 (1)	.366	–	.48 (.40, .55)	.52 (.45, .60)

Note. Constrained model constrained equal the variable mean and variance across twins in the same pair, across sex, and across zygosity, as well as intra-class correlations across sex within zygosity, except for adolescent tobacco initiation in time 1 where thresholds were allowed to be different across sex, and for peer tobacco use at both times where different means and variances were allowed across sex. -2LnL = -2 log-likelihood; df = degrees of freedom, AIC = Akaike information criterion. A = additive genetic influences. C = shared environmental influences. E = non-shared environmental influences. The *p*-values indicate tests in model fit between the full and the reduced models. Final selected models bolded.

Table 2

Descriptive Statistics and Univariate Model Estimates (95% CIs)

	n	M or (Yes)%	SD	Skewness	Kurtosis	Univariate estimates		
						A	C	E
Time 1								
Adolescent cigarette smoking initiation	1172	.09	.29	2.83	6.04	.29 (.00, .80)	.35 (.00, .72)	.36 (.19, 1.00)
Parental supervision	1182	3.31	.77	-1.10	.58	.29 (.03, .52)	.15 (.00, .36)	.55 (.47, .65)
Peer tobacco use	1179	.62	.72	.97	.12	.26 (.00, .47)	.13 (.00, .36)	.61 (.53, .71)
Time 2								
Adolescent cigarette smoking initiation	903	.10	.30	2.64	4.98	.78 (.58, .90)	—	.22 (.10, .42)
Parental supervision	902	3.29	.72	-.71	-.48	.10 (.00, .37)	.39 (.15, .53)	.51 (.43, .60)
Peer tobacco use	903	.75	.72	.60	-.64	.12 (.00, .38)	.39 (.18, .54)	.49 (.41, .59)

Note. MZ = monozygotic. DZ = dizygotic. A = additive genetic influences. C = shared environmental influences. E = non-shared environmental influences. C in time 2 adolescent cigarette smoking initiation was estimated to be 0 in the full ACE model and consequently fixed in subsequent analyses.

Table 3

Descriptive Statistics and Univariate Model Estimates by Young & Old Subgroups (95% CIs)

	n	M or (Yes)%	SD	Skewness	Kurtosis	Univariate estimates		
						A	C	E
Time 1 young subgroup (≥ 9 & ≤12)								
Adolescent cigarette smoking initiation	716	.07	.25	3.41	9.65	.14 (.00, .80)	.38 (.00, .73)	.48 (.20, .81)
Parental supervision	729	3.48	.67	-1.46	1.93	.12 (.00, .42)	.19 (.00, .36)	.70 (.58, .81)
Peer tobacco use	720	.45	.65	1.43	1.49	.27 (.00, .38)	.00 (.00,.21)	.73 (.62,.86)
Time 1 old subgroup (≥ 13 & ≤ 16)								
Adolescent cigarette smoking initiation	456	.13	.33	2.23	2.98	.51 (.00, .89)	.20 (.00, .77)	.29 (.11, .60)
Parental supervision	453	3.06	.84	-.64	-.39	.52 (.20, .63)	.00 (.00, .25)	.48 (.37, .62)
Peer tobacco use	459	.88	.75	.46	-0.58	.29 (.00, .52)	.10 (.00, .42)	.61 (.48, .76)
Time 2 young subgroup (≥ 12 & ≤ 15)								
Adolescent cigarette smoking initiation	592	.08	.27	3.10	7.65	.60 (.23, .85)	—	.40 (.15,.77)
Parental supervision	593	3.39	.68	-.90	-.07	.00 (.00, .37)	.40 (.30, .49)	.60 (.51, .70)
Peer tobacco use	595	.61	.68	.91	-.01	.00 (.00, .28)	.45 (.20, .53)	.55 (.47, .65)
Time 2 old subgroup (≥ 16)								
Adolescent cigarette smoking initiation	308	.14	.35	2.03	2.13	.89 (.67,1.00)	—	.11 (.02, .33)
Parental supervision	309	3.11	.75	-.40	-.90	.35 (.00, .70)	.26 (.00, .59)	.40 (.29, .55)
Peer tobacco use	308	1.02	.73	.11	-.95	.49 (.01, .67)	.06 (.00, .44)	.45 (.33, .63)

Note. MZ = monozygotic. DZ = dizygotic. A = additive genetic influences. C = shared environmental influences. E = non-shared environmental influences.

Table 4

Correlations with Adolescent Cigarette Smoking Initiation based on Bivariate Correlated Models (95% CIs)

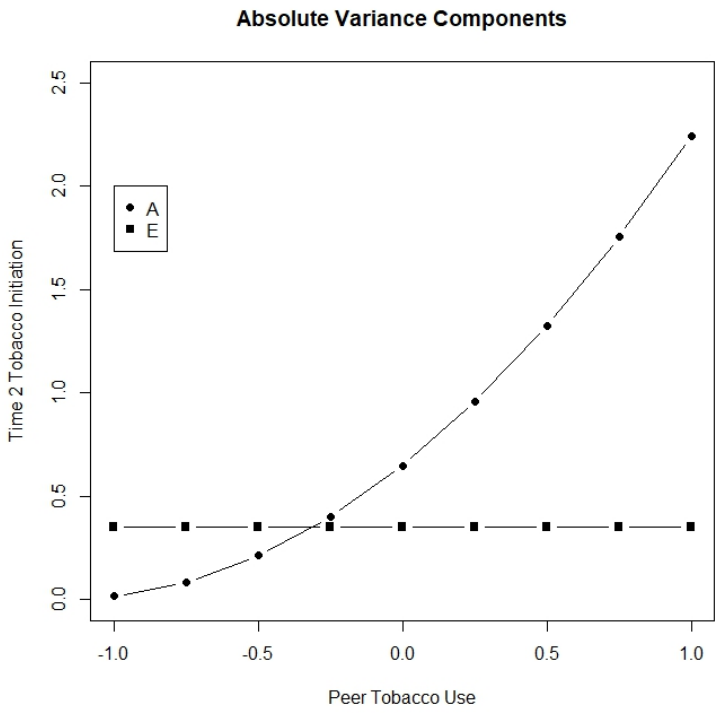
	Phenotypic	Cross-twin cross-trait		ACE factors		
	r	r_{MZ}	r_{DZ}	r_A	r_C	r_E
Time 1						
Parental supervision	-.14 (-.24, -.04)	-.21 (-.33, -.09)	-.09 (-.23, .06)	-.83 (-1.00, 1.00)	-.16 (-1.00, 1.00)	.16 (-.08, .39)
Peer tobacco use	.42 (.33, .50)	.36 (.24, .47)	.20 (.05, .34)	.88 (-1.00, 1.00)	.69 (-1.00, 1.00)	-.14 (-.36, .09)
Time 2						
Parental supervision	-.16 (-.27, -.04)	-.18 (-.31, -.04)	-.21 (-.35, -.05)	-.66 (-1.00, -.16)	–	.14 (-.14, .40)
Peer tobacco use	.45 (.34, .54)	.34 (.20, .46)	.25 (.09, .39)	1.00 (-1.00, 1.00)	–	.26 (-.01, .51)

Note. MZ = monozygotic. DZ = dizygotic. A = additive genetic influences. C = shared environmental influences. E = non-shared environmental influences.

The bivariate biometric moderation model (Table 5) indicated the presence of G×E involving peer tobacco use at time 2. Specifically, peer tobacco use at time 2 only moderated the unique genetic path to adolescent cigarette smoking initiation (βa = .69, 95% CI [0.49, 0.92]), but not the unique non-shared environmental path or the common genetic and non-shared environmental paths to adolescent cigarette smoking initiation. Parental supervision demonstrated no evidence of G×E or E×E at either time point. As shown in Figure 3, the absolute genetic variance of adolescent cigarette smoking initiation was amplified at higher levels of peer tobacco use in middle adolescence.

Figure 3

Estimated Absolute Variance Components of Adolescent Cigarette Smoking Initiation in Mid-Adolescence as a Function of Peer Tobacco Use



Note. A = additive genetic influences. E = non-shared environmental influences.

Table 5

Bivariate Biometric Moderation Model Fit Indices Controlling Age

	Model	-2LnL (df)	AIC	Comparison Model	$\Delta\chi^2$ (Δdf)	*p*
Peer Tobacco Use						
Time 1						
Common β_a, β_c, β_e & unique β_a, β_c, β_e	1	3004.17 (2304)	3054.17	–	–	–
drop common β_a, β_c, β_e	2	3006.11 (2307)	3050.11	1	1.94 (3)	.585
drop unique β_a, β_c, β_e	3	3005.65 (2307)	3049.65	1	1.48 (3)	.686
drop all	**4**	**3007.46 (2310)**	**3045.46**	**1**	**3.29 (6)**	**.771**
Time 2						
Common β_a, β_e & unique β_a, β_e	1	2283.99 (1779)	2319.99	–	–	–
drop common β_a, β_e	2	2285.91 (1781)	2317.91	1	1.92 (2)	.384
drop unique β_a, β_e	3	2289.93 (1781)	2321.93	1	5.94 (2)	.051
drop common β_a, β_e and unique β_a	4	2305.35 (1782)	2335.35	1	21.36 (3)	.000
				2	19.44 (1)	.000
drop common β_a, β_e and unique β_e	**5**	**2288.59 (1782)**	**2318.59**	**1**	**4.59 (3)**	**.204**
				2	**2.68 (1)**	**.102**
Parental Supervision						
Time 1						
Common β_a, β_c, β_e & unique β_a, β_c, β_e	1	3213.02 (2291)	3261.02	–	–	–
drop common β_a, β_c, β_e	2	3217.88 (2294)	3259.88	1	4.86 (3)	.183
drop unique β_a, β_c, β_e	3	3217.59 (2294)	3259.59	1	4.57 (3)	.206
drop all	**4**	**3220.25 (2297)**	**3256.25**	**1**	**7.23 (6)**	**.300**
Time 2						
Common β_a, β_e & unique β_a, β_e	1	2333.32 (1764)	2367.32	–	–	–
drop common β_a, β_e	2	2336.17 (1766)	2366.17	1	2.85 (2)	.240
drop unique β_a, β_e	3	2335.84 (1766)	2365.84	1	2.53 (2)	.283
drop all	**4**	**2337.60 (1768)**	**2363.60**	**1**	**4.28 (4)**	**.370**

Note. -2LnL = -2 log-likelihood; df = degrees of freedom, AIC = Akaike information criterion. The *p*-values indicate tests in model fit between the full and the reduced models. Final selected models bolded.

Discussion

Scant research has explored the genetic and environmental contributions to adolescent tobacco use in non-Western samples. There is also scarce knowledge on how parental supervision and peer tobacco use could potentially moderate genetic and environmental influences on adolescent tobacco use. The current study investigated gene-environment interplay between perceived parental supervision and peer tobacco use on cigarette smoking initiation among Chinese adolescent twins from early to middle adolescence based on sample average age at each assessment time. Cigarette smoking initiation was under genetic influence during early and middle adolescence. While moderate in early adolescence, shared environmental influences on cigarette smoking initiation became negligible in middle adolescence. Non-shared environmental influences on cigarette smoking initiation were notable at both times, although they became comparatively less prominent than genetic influences in middle adolescence. Adolescent cigarette smoking initiation was negatively linked with parental supervision in early and mid-adolescence, and positively related to peer tobacco use at both times. The links between peer tobacco use and adolescent cigarette smoking initiation appear stronger than parental supervision. Common genetic factors largely explained the relation between parental supervision and adolescent cigarette smoking initiation in mid-adolescence. High levels of peer tobacco use amplified genetic risk for adolescent cigarette smoking initiation only in mid-adolescence.

Gene-Environment Contributions to Adolescent Cigarette Smoking Initiation, Parental Supervision, and Peer Tobacco Use

Genetic, shared, and non-shared environmental factors all contributed to Chinese adolescents' cigarette smoking initiation in early adolescence. In mid-adolescence, genetic influences became larger while shared environmental influences became negligible. These findings are consistent

with the patterns of change in gene-environment contributions to tobacco use revealed in previous research using Western and Eastern samples (Kendler et al., 2008; Lessov-Schlaggar et al., 2006), providing further evidence for the increased heritability of tobacco use over time.

Notably, shared environmental influences became more pronounced, while genetic influences became less prominent for parental supervision. Enhanced shared environmental influence could reflect proactive efforts taken by Chinese parents to more closely monitor their children's behavior to ensure their successful adaptation to increasingly complex social and academically challenging environments during the transition from primary to secondary education (Zheng et al., 2023). The reduced salience of genetic influences on parental supervision in mid-adolescence could suggest that a greater implementation of parental control strategies counteracts or buffers against the evocative effects of genetically influenced adolescent behaviors (Elam et al., 2017). However, more future research is needed to further elaborate on these potential relations.

Active rGE has been implicated in adolescent affiliation with substance-using peers, suggesting genetic influences on peer selection processes (Wills & Carey, 2013). In the current sample, peer cigarette use was influenced by non-shared environmental and genetic factors in early adolescence, but primarily by environmental (shared and non-shared) factors in mid-adolescence. Recent work conducting social network analyses in Chinese adolescents has found that peer selection does not influence their tobacco use, suggesting that shared interests in smoking does not meaningfully contribute to peer group selection (Delay et al., 2023). A reduced importance of peer selection processes in Chinese adolescent smoking behaviors could potentially explain the modest genetic influences that were found on peer cigarette use in mid-adolescence for the current sample.

Links between Peer Tobacco Use, Parental Supervision, and Adolescent Cigarette Smoking Initiation

Chinese adolescent cigarette smoking initiation and peer tobacco use were positively linked in both early and middle adolescence, whose magnitudes were noticeably larger than with parental supervision at both times. This finding suggests the increased importance of peer influences during adolescence as evidenced in previous studies (Cambron et al., 2018; Liu et al., 2017). Adolescents increasingly rely more on their peers when determining which activities and values they endorse (Simons-Morton & Farhat, 2010). Adolescents' social circle and surroundings could impact their behaviors and decision-making in using tobacco. For instance, adolescents could feel pressured to conform to certain behaviors to be accepted by their peers (DeLay et al., 2023). Current findings nonetheless cannot provide conclusive answers regarding potential environmental or genetic mechanisms due to the wide 95% CIs. Further research with larger sample sizes is required to further determine how common genetic and environmental factors could explain the links between cigarette initiation and peer tobacco use to better understand the mechanisms that potentiate genetic risk in adolescent tobacco use.

Consistent with previous studies (Blustein et al., 2015; Van Ryzin et al., 2012), parental supervision was negatively linked with Chinese adolescent cigarette smoking initiation, which could correspond to parental efforts to actively regulate their children’s peer groups and activities and attempts at reducing exposure to tobacco products (Tornay et al., 2013). However, the results showed that common genetic factors mainly explained the relation between parental supervision and cigarette smoking initiation in mid-adolescence. This finding may suggest the presence of a passive rGE in parent–child processes related to adolescent cigarette smoking

initiation (Knafo & Jaffee, 2013), wherein parents who provide low supervision may also be genetically susceptible to tobacco use and may pass on relevant risk genes to their children.

Gene-Environment Interactions between Parental Supervision, Peer and Adolescent Cigarette Smoking Initiation

Consistent with previous twin studies on tobacco use (Harden et al., 2008) and broadly with those on alcohol use (Dick et al., 2007a; Dick, 2011; Zheng et al., 2019, 2023), peer tobacco use amplified unique genetic influences on adolescent cigarette smoking initiation in mid-adolescence, showing evidence for G×E in accordance with the diathesis stress model (Monroe & Simons, 1991). As teenagers strive for independence and spend more time with their peers, peer pressure may substantially impact their experimentation and use of substances more than parental guidance (Walden et al., 2004; Zheng et al., 2023). Notably, the presence of significant moderation in mid-adolescence but not in early adolescence suggests the changing role of genetic influences, or dynamic G×E throughout development consistent with previous findings on conduct problems (Burt, 2015) and alcohol use (Dick, 2011; Zheng et al., 2023). This finding indicates mid-adolescence as a vulnerable developmental period relative to early adolescence wherein genetic vulnerabilities to cigarette smoking initiation are particularly susceptible to peer influences. Future studies should further adopt more rigorous longitudinal approaches to explore the shifting dynamics of genetic influences on tobacco use across development, especially into late adolescence and young adulthood.

Contrary to our expectation and inconsistent with prior twin studies that have investigated parental knowledge on tobacco use (Dick et al., 2007a, 2007b) or parent–child relationship problems on externalizing problems including nicotine dependence (Hicks et al., 2009), the current findings revealed no evidence of parental supervision modifying genetic or

environmental influences on Chinese adolescent cigarette smoking initiation. The moderating role of parenting practices on genetic and environmental influences are relatively explored more in adolescent alcohol use, which also demonstrate inconsistent and inconclusive findings with positive (Hicks et al., 2008; Zheng et al., 2021, 2023) and null (Cooke et al., 2015; Dick et al., 2007 findings. Different measures of outcomes (e.g., externalizing problems vs. tobacco initiation), ranges of ages (e.g., early to mid-adolescence vs. late adolescence), operationalization of parenting practices (e.g., parental knowledge vs. supervision), as well as the (first) use of a non-Western adolescent sample could all potentially contribute to these discrepant findings. Additionally, different sociocultural norm leniency and parental attitudes could possibly explain the discrepant findings regarding G×E between adolescent tobacco and alcohol use. Nonetheless, by considering both peer tobacco use and parental supervision in the same study, the current findings are congruent with the general pattern revealed in the small pertinent G×E literature that has exclusively focused on adolescent alcohol use (Cooke et al., 2015; Dick et al., 2007a; Zheng et al., 2019, 2021, 2023) by providing the first empirical evidence on the stronger role of peer substance use than parenting practices in modulating genetic influences on adolescent cigarette smoking initiation. Future studies are sorely needed to replicate and extend the current findings.

Strengths, Limitations, and Future Directions

The current study constitutes the first effort to jointly investigate the relative contributions of parental supervision and peer tobacco use to gene-environment influences on adolescent cigarette smoking initiation in a sample of Chinese twins prospectively followed from early to middle adolescence. Besides contributing to ethnic and racial diversity in genetic research on tobacco use, the current study provides novel insights into the dynamic processes in which peer processes differentially modify genetic risk for cigarette smoking initiation over development.

Despite the strengths, there are some limitations to consider when interpreting the findings. First, the current sample includes participants from a broad age range. However, age was statistically controlled for in all moderation analyses. Controlling for age allows us to clarify the unique contributions of genetic and environmental factors of peer use and parental supervision on cigarette smoking initiation while accounting for potential confounds associated with age-related differences. Future studies should follow twins of similar ages in other non-Western populations longitudinally to better understand how environmental and genetic factors impact the initiation of cigarette use throughout development. Additionally, the current sample only includes adolescents from one Chinese province; therefore, the findings may not apply to populations from other regions of China. Second, genetically informed studies on adolescent tobacco use have historically used a mixture of measures capturing both initiation and frequency (e.g., Dick et al., 2007a, 2007b). Future studies should investigate the extent to which genetic factors differ between tobacco use frequency and initiation in adolescent populations. Third, while the measure of parental supervisory behaviors in this study covers aspects pertaining to parental knowledge and control, it does not include active supervision components outlined in previous research (Zheng et al., 2023). Fourth, assessments in the current study were all based on adolescent self-reports, which could suffer the issue of shared method variance. Future studies should use multiple sources to obtain more precise measures of adolescent tobacco use, parental supervision, and peer tobacco use. Finally, the data utilized in the current study were obtained before the widespread prevalence of e-cigarette use and vaping in China (Dai et al., 2022). Future investigations should determine whether the current findings would replicate across these alternative methods of tobacco consumption.

Conclusion

The initiation and continued use of tobacco products constitutes an ongoing source of preventable disease that continues to pose significant risk to adolescent health. Scarce research has sought to explore the influences of two well-known environmental risk factors, parental supervision and peer tobacco use, on genetic and environmental influences on adolescent cigarette smoking initiation. The current study shows that Chinese adolescent cigarette smoking initiation involves dynamic gene-environment transactions primarily with peer processes over development. Peer tobacco use exerts larger impact than parental supervision on Chinese adolescent cigarette smoking initiation during both early and mid-adolescence. Moreover, peer tobacco use amplifies genetic risks for cigarette smoking initiation in middle adolescence but not in early adolescence. This finding suggests that mid-adolescence constitutes a developmental period wherein underlying genetic risk for cigarette smoking initiation is particularly sensitive to peer influences. Targeted interventions aimed at reducing Chinese adolescent cigarette smoking initiation should focus on peer processes during this developmental period.

References

Bao, Z. Q., Yu, C. Q., Wang, B. Q., Cao, W. H., Gao, W. J., Lyu, J., Wang S. F., Pang, Z. C., Cong, L. M., Dong, Z., Wu, F., Wang, H., Wu, X. P., Wang, D. Z., Wang, X. J., Wang, B. Y., & Li, L. M. (2016). Influences of genetic and environmental factors on smoking related behaviors among male twin adults in China. *Chinese Journal of Epidemiology*, *37*(5), 618–623. [In Chinese]. https://doi.org/10.3760/cma.j.issn.0254-6450.2016.05.006

Bricker, J. B., Rajan, K. B., Zalewski, M., Andersen, M. R., Ramey, M., & Peterson, A. V. (2009). Psychological and social risk factors in adolescent smoking transitions: A population-based longitudinal study. *Health Psychology*, *28*(4), 439–447. https://doi.org/10.1037/a0014568

Blustein, E. C., Munn-Chernoff, M. A., Grant, J. D., Sartor, C. E., Waldron, M., Bucholz, K. K., Madden, P. A., & Heath, A. C. (2015). The association of low parental monitoring with early substance use in European American and African American adolescent girls. *Journal of Studies on Alcohol and Drugs*, *76*(6), 852–861. https://doi.org/10.15288/jsad.2015.76.852

Burt, S. A. (2015). Evidence that the gene–environment interactions underlying youth conduct problems vary across development. *Child Development Perspectives*, *9*(4), 217–221. https://doi.org/10.1111/cdep.12136

Cambron, C., Kosterman, R., Catalano, R. F., Guttmannova, K., & Hawkins, J. D. (2018). Neighborhood, family, and peer factors associated with early adolescent smoking and alcohol use. *Journal of Youth and Adolescence*, *47*(2), 369–382. https://doi.org/10.1007/s10964-017-0728-y

Chen, X., & French, D. C. (2008). Children's social competence in cultural context. *Annual Review of Psychology*, *59*, 591–616. https://doi.org/10.1146/annurev.psych.59.103006.093606

Cleveland, H. H., Wiebe, R., & Rowe, D. C. (2005). Sources of exposure to smoking and drinking friends among adolescents: A behavioral-genetic evaluation. *The Journal of Genetic Psychology*, *166*(2), 153–169.

Cooke, M. E., Meyers, J. L., Latvala, A., Korhonen, T., Rose, R. J., Kaprio, J., Salvatore, J. E., & Dick, D. M. (2015). Gene-environment interaction effects of peer deviance, parental knowledge and stressful life events on adolescent alcohol use. *Twin Research and Human Genetics*, *18*(5), 507–517. https://doi.org/10.1017/thg.2015.56

Cruz, J. E., Emery, R. E., & Turkheimer, E. (2012). Peer network drinking predicts increased alcohol use from adolescence to early adulthood after controlling for genetic and shared environmental selection. *Developmental Psychology*, *48*(5), 1390–1402. https://doi.org/10.1037/a0027515

Dai, L., Lu, W., Wang, J., Zhang, L., & Zhu, J. (2022). Social environment exposure to electronic cigarettes and its association with e-cigarette use among adolescents in Shanghai, China. *Frontiers in Public Health*, *10*, 1 –11. https://doi.org/10.3389/fpubh.2022.1005323

D'amico, E. J., Tucker, J. S., Shih, R. A., & Miles, J. N. (2014). Does diversity matter? The need for longitudinal research on adolescent alcohol and drug use trajectories. *Substance Use & Misuse*, *49*(8), 1069–1073. https://doi.org/10.3109/10826084.2014.862027

DeLay, D., Shen, M., Cook, R. E., Zhao, S., Logis, H., & French, D. C. (2023). Peers influence the tobacco and alcohol use of Chinese adolescents. *Journal of Research on Adolescence*, *33*(2), 591–602. https://doi.org/10.1111/jora.12828

Dick, D. M. (2011). Developmental changes in genetic influences on alcohol use and dependence. *Child Development Perspectives, 5*(4)*,* 223–230. https://doi.org/10.1111/j.1750-8606.2011.00207.x

Dick, D. M., Prescott, C., & McGue, M. (2009). The genetics of substance use and substance use disorders. In Kim Y-K (ed.), *Handbook of behavior genetics* (pp. 433–453). Springer.

Dick, D. M., Pagan, J. L., Viken, R., Purcell, S., Kaprio, J., Pulkkinen, L., & Rose, R. J. (2007a). Changing environmental influences on substance use across development. *Twin Research and Human Genetics*, *10*(2), 315–326. https://doi.org/10.1375/twin.10.2.315

Dick, D. M., Viken, R., Purcell, S., Kaprio, J., Pulkkinen, L., & Rose, R. J. (2007b). Parental monitoring moderates the importance of genetic and environmental influences on adolescent smoking. *Journal of Abnormal Psychology*, *116*(1), 213–218. https://doi.org/10.1037/0021-843X.116.1.213

Do, E. K., Prom-Wormley, E. C., Eaves, L. J., Silberg, J. L., Miles, D. R., & Maes, H. H. (2015). Genetic and environmental influences on smoking behavior across adolescence and young adulthood in the Virginia Twin Study of adolescent behavioral development and the transitions to substance abuse follow-up. *Twin Research and Human Genetics*, *18*(1), 43–51. https://doi.org/10.1017/thg.2014.78

Duan, H., Ning, F., Zhang, D., Wang, S., Zhang, D., Tan, Q., Tian, X., & Pang, Z. (2013). The Qingdao Twin Registry: A status update. *Twin Research and Human Genetics*, *16*(1), 79–85. https://doi.org/10.1017/thg.2012.113

Elam, K. K., Chassin, L., Lemery-Chalfant, K., Pandika, D., Wang, F. L., Bountress, K., Dick, D., & Agrawal, A. (2017). Affiliation with substance-using peers: Examining gene-environment correlations among parent monitoring, polygenic risk, and children's impulsivity. *Developmental Psychobiology*, *59*(5), 561–573. https://doi.org/10.1002/dev.21529

Gao, W. J., Li, L. M., Cao, W. H., Zhan, S. Y., Lv, J., Pang, Z. C., Chen, W. J., Wang, S. J.,

Chen, R. F., & Hu, Y. H. (2010). Study of twin-birth on genetic or environmental determinants of male Chinese smoking initiation. *Journal of Peking University (Health Sciences)*, *42*(3), 284–287. [In Chinese].

Goodchild, M., Nargis, N., & d'Espaignet, E. T. (2018). Global economic cost of smoking-attributable diseases. *Tobacco Control*, *27*(1), 58–64. http://dx.doi.org/10.1136/tobaccocontrol-2016-053305

Harden, K. P., Hill, J. E., Turkheimer, E., & Emery, R. E. (2008). Gene-environment correlation and interaction in peer effects on adolescent alcohol and tobacco use. *Behavior Genetics*, *38*(4), 339–347. https://doi.org/10.1007/s10519-008-9202-7

Hicks, B. M., South, S. C., DiRago, A. C., Iacono, W. G., & McGue, M. (2009). Environmental adversity and increasing genetic risk for externalizing disorders. *Archives of General Psychiatry*, *66*(6), 640–648. doi:10.1001/archgenpsychiatry.2008.554

Hemovich, V., Lac, A., & Crano, W. D. (2011). Understanding early-onset drug and alcohol outcomes among youth: The role of family structure, social factors, and interpersonal perceptions of use. *Psychology, Health & Medicine*, *16*(3), 249–267. https://doi.org/10.1080/13548506.2010.532560

Hemphill, S. A., Heerde, J. A., Herrenkohl, T. I., Patton, G. C., Toumbourou, J. W., & Catalano, R. F. (2011). Risk and protective factors for adolescent substance use in Washington State, the United States and Victoria, Australia: A longitudinal study. *Journal of Adolescent Health*, *49*(3), 312–320. https://doi.org/10.1016/j.jadohealth.2010.12.017

Hoffman, B. R., Sussman, S., Unger, J. B., Valente, T. W. (2006). Peer influences on adolescent cigarette smoking: A theoretical review of the literature. *Substance Use & Misuse*, *41*(1), 103–155. doi: 10.1080/10826080500368892

Jamner, L. D., Whalen, C. K., Loughlin, S. E., Mermelstein, R., Audrain-McGovern, J., Krishnan-Sarin, S., Worden, J. K., & Leslie, F. M. (2003). Tobacco use across the formative years: A road map to developmental vulnerabilities. *Nicotine & Tobacco Research*, *5*(S1), S71–S87. https://doi.org/10.1080/14622200310001625573

Kendler, K. S., Schmitt, E., Aggen, S. H., & Prescott, C. A. (2008). Genetic and environmental influences on alcohol, caffeine, cannabis, and nicotine use from early adolescence to middle adulthood. *Archives of General Psychiatry*, *65*(6), 674–682. doi:10.1001/archpsyc.65.6.674

Kiuru, N., Burk, W. J., Laursen, B., Salmela-Aro, K., & Nurmi, J. E. (2010). Pressure to drink but not to smoke: Disentangling selection and socialization in adolescent peer networks and peer groups. *Journal of Adolescence*, *33*(6), 801–812. https://doi.org/10.1016/j.adolescence.2010.07.006

Knafo, A., & Jaffee, S. R. (2013). Gene-environment correlation in developmental psychopathology. *Development and Psychopathology*, *25*(1), 1–6. https://doi.org/10.1017/S0954579412000855

Latendresse, S. J., Rose, R. J., Viken, R. J., Pulkkinen, L., Kaprio, J., & Dick, D. M. (2010). Examining the etiology of associations between perceived parenting and adolescents' alcohol use: Common genetic and/or environmental liabilities? *Journal of Studies on Alcohol and Drugs*, *71*(3), 313–325. https://doi.org/10.15288/jsad.2010.71.313

Lessov-Schlaggar, C. N., Pang, Z., Swan, G. E., Guo, Q., Wang, S., Cao, W., Unger, J. B., Johnson, C. A., & Lee, L. (2006). Heritability of cigarette smoking and alcohol use in Chinese male twins: The Qingdao twin registry. *International Journal of Epidemiology*, *35*(5), 1278–1285. https://doi.org/10.1093/ije/dyl148

Liu, J., Zhao, S., Chen, X., Falk, E., & Albarracín, D. (2017). The influence of peer behavior as a

function of social and cultural closeness: A meta-analysis of normative influence on adolescent smoking initiation and continuation. *Psychological Bulletin*, *143*(10), 1082–1115. https://doi.org/10.1037/bul0000113

Maes, H. H., Prom-Wormley, E., Eaves, L. J., Rhee, S. H., Hewitt, J. K., Young, S., Corley, R., McGue, M., Iacono, W.G., Legrand, L., Samek, D. R., Murrelle, E. L., Silberg, J. L., Miles, D. R., Schieken, R. M., Beunen, G. P., Thomis, M., Rose, R. J., Dick, D. M., … Neale, M. C. (2017). A genetic epidemiological mega analysis of smoking initiation in adolescents. *Nicotine & Tobacco Research*, *19*(4), 401–409. https://doi.org/10.1093/ntr/ntw294

Maes, H. H., Sullivan, P. F., Bulik, C. M., Neale, M. C., Prescott, C. A., Eaves, L. J., & Kendler, K. S. (2004). A twin study of genetic and environmental influences on tobacco initiation, regular tobacco use and nicotine dependence. *Psychological Medicine*, *34*(7), 1251–1261. https://doi.org/10.1017/S0033291704002405

Metzger, A., Wakschlag, L. S., Anderson, R., Darfler, A., Price, J., Flores, Z., & Mermelstein, R. (2013). Information management strategies within conversations about cigarette smoking: Parenting correlates and longitudinal associations with teen smoking. *Developmental Psychology*, *49*(8), 1565–1578. https://doi.org/10.1037/a0030720

Monroe, S. M., & Simons, A. D. (1991). Diathesis-stress theories in the context of life stress research: Implications for the depressive disorders. *Psychological Bulletin*, *110*(3), 406–425. https://doi.org/10.1037/0033-2909.110.3.406

Musci, R. J., Uhl, G., Maher, B., & Ialongo, N. S. (2015). Testing gene×environment moderation of tobacco and marijuana use trajectories in adolescence and young adulthood. *Journal of Consulting and Clinical Psychology*, *83*(5), 866–874. https://doi.org/10.1037/a0039537

Neale, M. C., Hunter, M. D., Pritikin, J. N., Zahery, M., Brick, T. R., Kirkpatrick, R. M., Estabrook, R., Bates, T. C., Maes, H. H., & Boker, S. M. (2016). OpenMx 2.0: Extended structural equation and statistical modeling. *Psychometrika*, *81*(2), 535–549. https://doi.org/10.1007/s11336-014-9435-8

Neale, M. C., & Maes, H. H. (2004). Methodology for genetic studies of twins and families. Kluwer Academic Publishers.

Neale, M. C., Røysamb, E., & Jacobson, K. (2006). Multivariate genetic analysis of sex limitation and G×E interaction. *Twin Research and Human Genetics, 9*(4)*,* 481–489. https://doi.org/10.1375/twin.9.4.481

Polderman, T. J., Benyamin, B., De Leeuw, C. A., Sullivan, P. F., Van Bochoven, A., Visscher, P. M., & Posthuma, D. (2015). Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nature Genetics*, *47*(7), 702–709. https://doi.org/10.1038/ng.3285

Salvatore, J. E., Aliev, F., Bucholz, K., Agrawal, A., Hesselbrock, V., Hesselbrock, M., Bauer, L., Kuperman, S., Schuckit, M. A., Kramer, J. R., Edenberg, H. J., Foroud, T. M., & Dick, D. M. (2015). Polygenic risk for externalizing disorders: Gene-by-development and gene-by-environment effects in adolescents and young adults. *Clinical Psychological Science*, *3*(2), 189–201. https://doi.org/10.1177/2167702614534211

Silveira, M. L., Conway, K. P., Green, V. R., Kasza, K. A., Sargent, J. D., Borek, N., Stanton, C. A., Cohn, A., Hilmi, N., Cummings, K. M., Niaura, R. S., Lambert, E. Y., Brunette, M. F., Zandberg, I., Tanski, S. E., Reissig, C. J., Callahan-Lyon, P., Slavit, W. I., Hyland, A. J., & Compton, W. M. (2018). Longitudinal associations between youth tobacco and substance use in waves 1 and 2 of the Population Assessment of Tobacco and Health (PATH) Study. *Drug and Alcohol Dependence*, *191*, 25–36. https://doi.org/10.1016/j.drugalcdep.2018.06.018

Simons-Morton, B. G., & Farhat, T. (2010). Recent findings on peer group influences on adolescent smoking. *The Journal of Primary Prevention*, *31*(4), 191–208. https://doi.org/10.1007/s10935-010-0220-x

Sirugo, G., Williams, S. M., & Tishkoff, S. A. (2019). The missing diversity in human genetic studies. *Cell*, *177*(1), 26–31. https://doi.org/10.1016/j.cell.2019.02.048

Sun, X., Yao, L., Niu, G., & Lin, S. (2021). The moderating role of deviant peer affiliation in the relation between cyber-victimization, tobacco and alcohol use, and age differences. *International Journal of Environmental Research and Public Health*, *18*(16), 8294–8303. https://doi.org/10.3390/ijerph18168294

Tornay, L., Michaud, P. A., Gmel, G., Wilson, M. L., Berchtold, A., & Surís, J. C. (2013). Parental monitoring: A way to decrease substance use among Swiss adolescents? *European Journal of Pediatrics*, *172*(9), 1229–1234. https://doi.org/10.1007/s00431-013-2029-0

Unger, J. B., Lessov-Schlaggar, C. N., Pang, Z., Guo, Q., Ning, F., Gallaher, P., Lee, L., Cao, W., Conti, D., & Johnson, C. A. (2011). Heritability of smoking, alcohol use, and psychological characteristics among adolescent twins in Qingdao, China. *Asia Pacific Journal of Public Health*, *23*(4), 568–580. https://doi.org/10.1177/1010539509351052

United Nations Children's Fund. (2018). *Children in China: An atlas of social indicators of children in China 2018*. New York: UNICEF. Retrieved from https://www.unicef.cn/sites/unicef.org.china/files/2019-03/Atlas%202018%20ENG.pdf.

van der Sluis, S., Posthuma, D., & Dolan, C. V. (2012). A note on false positives and power in G×E modelling of twin data. *Behavior Genetics*, *42*(1), 170–186. https://doi.org/10.1007/s10519-011-9480-3

Van Ryzin, M. J., Fosco, G. M., & Dishion, T. J. (2012). Family and peer predictors of substance

use from early adolescence to early adulthood: An 11-year prospective analysis. *Addictive Behaviors*, *37*(12), 1314–1324. https://doi.org/10.1016/j.addbeh.2012.06.020

Walden, B., McGue, M., Burt, S. A., & Elkins, I. (2004). Identifying shared environmental contributions to early substance use: The respective roles of peers and parents. *Journal of Abnormal Psychology*, *113*, 440–450. https://doi.org/10.1037/0021-843X.113.3.440

Wills, A. G., & Carey, G. (2013). Adolescent peer choice and cigarette smoking: Evidence of active gene–environment correlation? *Twin Research and Human Genetics*, *16*(5), 970–976. https://doi.org/10.1017/thg.2013.51

Xiong, P. S., Xiong, M. J., Liu, Z. X., & Liu, Y. (2020). Prevalence of smoking among adolescents in China: An updated systematic review and meta-analysis. *Public Health*, *182*, 26–31. https://doi.org/10.1016/j.puhe.2020.01.011

Zheng, Y., Brendgen, M., Girard, A., Dionne, G., Boivin, M., & Vitaro, F. (2019). Peer alcohol use differentially amplifies genetic and environmental effects on different developmental trajectories of adolescent alcohol use. *Journal of Adolescent Health*, *65*(6), 752–759. https://doi.org/10.1016/j.jadohealth.2019.07.005

Zheng, Y., Brendgen, M., Meyer, Z., Vitaro, F., Dionne, G., & Boivin, M. (2021). Maternal parenting behaviors amplify environmental influences on developmental trajectories of alcohol use during adolescence. *Behavior Genetics*, *51*(5), 528–542. https://doi.org/10.1007/s10519-021-10063-x

Zheng, Y., Meyer, Z., Unger, J. B., & Rijsdijk, F. (2023). Gene-environment interplay linking perceived parental supervision and peer drunkenness with Chinese adolescent alcohol initiation. *Child Development*, *94*(4), 853–864. https://doi.org/10.1111/cdev.13902

Zheng, Y., Pingault, J. B., Unger, J. B., & Rijsdijk, F. (2020). Genetic and environmental

influences on attention-deficit/hyperactivity disorder symptoms in Chinese adolescents: A longitudinal twin study. *European Child & Adolescent Psychiatry*, *29*(2), 205–216. https://doi.org/10.1007/s00787-019-01346-0

Zheng, Y., Rijsdijk, F., Pingault, J. B., McMahon, R. J., & Unger, J. B. (2016). Developmental changes in genetic and environmental influences on Chinese child and adolescent anxiety and depression. *Psychological Medicine*, *46*(9), 1829–1838. https://doi.org/10.1017/S003329171600031