Health Psychology
2008, Vol. 27, No. 6, 819–828

0278-6133/08/$12.00 DOI: 10.1037/0278-6133.27.6.819

Multiple Trajectories of Cigarette Smoking and the Intergenerational Transmission of Smoking: A Multigenerational, Longitudinal Study of a Midwestern Community Sample

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Objective: To investigate the relation between developmental phenotypes of parental smoking (trajectories of smoking from adolescence to adulthood) and the intergenerational transmission of smoking to their adolescent children. ***Design:*** A longitudinal, multigenerational study of a midwestern community sample followed individuals from adolescence into adulthood and was combined with Web-based assessment of participants' spouses and adolescent children. Mixture modeling identified multiple trajectories of smoking, and path analyses related these trajectories to adolescents' smoking (beyond both parents' current smoking). Potential mediations were parental education and adolescents' personality characteristics. ***Main Outcome Measure:*** The outcome measure was adolescent smoking. ***Results:*** A parent's smoking trajectory had a unique effect on their adolescent's smoking, beyond both parents' current smoking and the parent's educational attainment. However, although adolescents' personality characteristics were related both to adolescent smoking and to their parents' smoking, these characteristics could not explain the effects of the parent's smoking trajectory. ***Conclusion:*** Parents whose smoking had an early onset, steep acceleration, high levels of smoking, and persistence over time had the highest risk for intergenerational transmission of smoking to their adolescent children.

Keywords: smoking trajectories, adolescent smoking, intergenerational transmission, personality characteristics

Cigarette smoking shows significant heritability, with estimates ranging from 46% to 84% (Batra, Patkar, Berrettini, Weinstein, & Leone, 2003; Li, 2003). Heritability estimates are somewhat smaller for adolescent samples, but still substantial. For example, a review of twin and adoption studies of adolescent samples shows tobacco use heritability estimates ranging from 36% to 60% (Hopfer, Crowley, & Hewitt, 2003). These data suggest that cigarette smoking is both genetically and environmentally influenced. Given these findings, cigarette smoking would be expected to show intergenerational transmission, and parental cigarette smoking should be a powerful influence on adolescent smoking. However, there are somewhat conflicting findings, with some studies (e.g., Bricker et al., 2006; Chassin et al., 2005) showing parental smoking to be predictive of adolescent smoking and others showing weak or nonsignificant relations (e.g., Conrad, Flay, & Hill, 1992). These inconsistent findings may be due to methodological variations across studies (Avenevoli & Merikangas, 2003), as well as whether effects of parental smoking are lost in the context of other predictors that might mediate their effects. However, there is another important possibility—namely, that parents' current smoking status is too crude a phenotype to provide optimal insight into the etiology and intergenerational transmission of smoking behavior.

The idea that phenotypes must be refined beyond "current smoking status" has been proposed in genetic research (Lessov, Sawn, Ring, Khroyan, & Lerman, 2004), because different dimensions of smoking behavior have shown different heritabilities. For example, amount of smoking shows higher heritability than does smoking initiation (Koopmans, Slutske, Heath, Neale, & Boomsma, 1999). Moreover, although age of smoking onset is heritable (Broms, Silventoinen, Madden, Heath, & Kaprio, 2006; Heath, Jardine, Meyer, & Martin, 1999), the same genetic influences that account for age of onset do not account for amount smoked or for smoking cessation (Broms et al., 2006). These behavioral genetic findings suggest that a more careful specification of smoking phenotypes (beyond simple current smoking status) might produce a clearer understand-

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This research was supported by Grant DA13555 from the National Institute on Drug Abuse to Steven J. Sherman, Principal Investigator.

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ing of smoking etiology. Consistent with this idea, data for parents' current smoking, compared with former smoking, show different relations to adolescent smoking (Chassin, Presson, Rose, & Sherman, 2002; Otten, Engels, van de Ven, & Bricker, 2007). Thus, a more detailed specification of the parents' smoking phenotypes may produce a clearer understanding of the intergenerational transmission of smoking behavior.

Given the goal of identifying more refined and informative parental smoking phenotypes, developmental phenotypes that capture age of onset and time course of smoking (in terms of speed of escalation, peak of use, and persistence over time) may be particularly useful for examining etiological mechanisms and intergenerational transmission (Chassin, Presson, Sherman, Wirth, & Curran, in press; Jackson & Sher, in press). A similar proposal has been made in the study of antisocial behavior, in which two different developmental phenotypes have been distinguished: a childhood-onset, life course-persistent phenotype and an adolescent-limited phenotype (Moffitt & Caspi, 1991). These two phenotypes differ in etiological predictors, such that childhood-onset delinquents have backgrounds of inadequate parenting, neurocognitive problems, and early behavior problems, whereas adolescent-onset delinquents do not (Moffitt & Caspi, 2001). Moreover, a family history of externalizing disorders distinguishes life course-persistent males from other children with conduct problems (Odgers et al., 2007), suggesting that intergenerational transmission may be particularly strong for early-onset, life course-persistent trajectories.

In the smoking literature, longitudinal studies have empirically identified multiple trajectories of smoking that vary in their age of onset, peak of use, and persistence over time (e.g., Brook, Balka, Ning, & Brook, 2007; Chassin, Presson, Sherman, & Pitts, 2000; Orlando, Tucker, Ellickson, & Klein, 2004; White, Pandina, & Chen, 2004) and that are differentially related both to antecedent risk factors and to outcomes. These trajectories represent developmental phenotypes of smoking, and, similar to the antisocial behavior literature, they may also vary in the risk that they carry for the intergenerational transmission of smoking. Chassin et al. (2000) found that a trajectory group that was of particular interest for intergenerational transmission was the group of early-onset persistent smokers. Compared with other smoking trajectories, these individuals began smoking at a young age, escalated steeply to a high rate of smoking, and persisted in their smoking into adulthood, and they had the highest levels of smoking among their biological parents. Moreover, a more recent follow-up of this sample at ages 32–42 (Chassin et al., in press) found that, of these individuals, 90% were still classifiable in this trajectory group and that this group showed the highest prevalence of tobacco dependence and the lowest educational attainment. The goal of the present study was to test whether this early-onset persistent group also showed the greatest risk of intergenerational transmission of smoking to their own adolescent children; that is, the present study related developmental phenotypes of smoking behavior based on longitudinal data from adolescence to adulthood to adolescent smoking behavior in the next generation.

Moreover, if these trajectory groups differ in their risk for intergenerational transmission, it is important to identify the mechanisms underlying this risk. Although a single study cannot address all possible mechanisms, the present study focused on several theory-based possibilities. First, a parent's life course history of smoking might predict their adolescent's smoking only because this history affected the parent's own likelihood of being a current smoker at the time that their adolescent was assessed. That is, smoking among those with an early onset and steep acceleration is also more likely to persist into adulthood, and parents who are current smokers may influence their adolescent's smoking through modeling mechanisms as well as by providing more opportunities to smoke and easier access to cigarettes. Thus, we tested whether parent's trajectory group membership was a unique predictor of their adolescent's smoking beyond the parent's current smoking status. Similarly, because of assortative mating, individuals with more severe and persistent life courses of smoking might be more likely to marry other similar individuals, and thus we tested whether the other parent's current smoking could explain the effects of trajectory group membership. Third, as noted earlier, those with early-onset persistent smoking had low educational attainment, and adolescents from less educated families have more smokers in their social networks and are themselves more likely to smoke (Chassin, Presson, Sherman, & Edwards, 1992; Hanson & Chen, 2007). Accordingly, we tested whether the parent's educational attainment mediated the effects of their smoking trajectory group membership.

Finally, we tested whether the adolescent's personality characteristics mediated the effect of the parent's smoking trajectory on adolescent smoking. This choice was based on theories of "behavioral undercontrol" as one pathway underlying the intergenerational transmission of early-onset substance use (Yoon, Iacono, Malone, & McGue, 2006). Behavioral undercontrol is a broad construct that includes impulsivity, sensation seeking, and conduct problems (Sher, 1991). Theorists suggest that adolescents whose parents show externalizing behaviors (including substance abuse) transmit these heritable personality characteristics to their offspring (as well as exacerbate these dispositions through poor parenting) and that adolescents who are behaviorally undercontrolled experience academic failure, ejection from mainstream peer groups, and affiliation with substance-using peers and are themselves more likely to use substances (Sher, 1991). Given the link between behavioral undercontrol and early-onset substance use, we hypothesized that adolescents whose parents showed early-onset persistent smoking would demonstrate the most behavioral undercontrol and thus be most likely to smoke.

We examined behavioral undercontrol using personality characteristics from the five-factor model (i.e., NEO personality characteristics). Specifically, high levels of neuroticism, low conscientiousness, and low agreeableness have been linked to behavioral undercontrol as defined by impulsivity and social deviance (Lynam et al., 2005), as well as inattention and hyperactivity–impulsivity (Nigg et al., 2002). In support of their utility as potential mediators of parental smoking trajectory group membership effects on adolescent smoking, these characteristics are also related to smoking in both adolescence (Harakeh, Scholte, deVries, & Engels, 2006) and adulthood (Malouff, Thortseinsson, & Schutte, 2006), and they are moderately heritable (Jang et al., 1996). Accordingly we tested whether adolescents' five-factor model personality characteristics (neuroticism, conscientiousness, and agreeableness, as well as openness and extraversion)

mediated the effects of the parent's smoking trajectory group membership on adolescent smoking. Finally, we tested adolescent's temperamental resistance to control as a mediator because it has also been linked to adolescents' externalizing behaviors (Bates, 1994).

Method

Participants

Adult participants were from the Indiana University (IU) Smoking Survey, an ongoing cohort-sequential study of the natural history of cigarette smoking (see, e.g., Chassin et al., 2000). Between 1980 and 1983, all consenting 6th–12th graders in a Midwestern county school system completed annual surveys (total *N* who were assessed at least once = 8,487). Follow-ups were conducted in 1987, 1993, 1999, and 2005 (ages 32–42). In each case, 70% or more of the original sample were successfully retained. Because the sample is 96% non-Hispanic Caucasian, ethnic differences were not considered.

Sample representativeness has been described in detail elsewhere (Chassin, Presson, Rose, & Sherman, 1996; Chassin et al., 2000). Demographically, the sample is similar to the community from which it was drawn (e.g., 64% marriage rates in this sample, compared with 66% among similarly aged adults in the Midwest [Lugaila, 1998] and 97% high school graduation rates in this sample, compared with 92% among similarly aged adults in the Midwest [Day & Curry, 1998]). At the most recent follow-up (2005), the smoking rate in the sample was 23%, compared with a 2006 statewide rate of 24% (Centers for Disease Control and Prevention [CDC], 2006) and regional rate of 17% (Indiana Tobacco Prevention and Cessation [ITPC], 2006). Thus, the sample is representative of its community, one that is predominantly White and well educated. Attrition biases have been discussed in detail elsewhere (e.g., Rose, Chassin, Presson, & Sherman, 1996). For each follow-up, those who were lost were compared with those who were retained in terms of their earlier data. Dropouts were more likely to be smokers, have more positive attitudes and beliefs about smoking, and have parents and friends who were more likely to smoke (effect sizes ranged from $r^2 = .01$ to $r^2 = .02$). Although these biases are small in magnitude, caution is warranted when making generalizations.

For the present analyses, we selected participants who had adolescent children between the ages of 10 and 16 and who had participated in a concurrent multigenerational study ($n = 1,023$) or in multigenerational studies conducted in 1995 and 1999 ($n = 315$). This yielded a sample of 1,338 participant–adolescent children pairs and 1,218 spouses of the participants. The mean age of the children was 13.6, and 51% were male.

Procedures

The original data (1980–1983) were collected with group-administered questionnaires in school. In 1987, these procedures were followed for cohorts who were still in high school; for older cohorts (and for all participants in 1993, 1999, and 2005), a survey was mailed and followed up by telephone interviews if surveys were not returned. Participants were paid $15-$30 over the waves, and in 1999 and 2005, they were also entered into a lottery for cash prizes.

Data on adolescent smoking, adolescent personality, and the other parent's smoking were obtained from our multigenerational studies. In 1995 and 1999, IU Smoking Survey participants who currently lived in the county, their spouses/partners, and their adolescent children were recruited for a laboratory study of parenting and adolescent smoking that included a measurement of smoking behavior (verified by carbon monoxide in expired air) and questionnaire measures (see Chassin, Presson, Rose, & Sherman, 2002; Chassin et al., 2005, for more details). In 2005, IU Smoking Survey participants, their adolescent children, and their spouses/partners were recruited into a Web-based study of parenting and adolescent smoking that included Web-based measures of cigarette smoking and child characteristics. Participants were paid $25 for these studies.

Measures

Parents' smoking and smoking trajectory. At each wave, IU Smoking Survey participants self-reported their smoking status (as "never smoked, not even a single puff"; "smoked once or twice 'just to try' but not in the last month"; "do not smoke, but in the past I was a regular smoker"; "smoke regularly, but no more than once a month"; "smoke regularly, but not more than once a week"; "smoke regularly, but not more than once a day"; and "smoke more than once a day"), and they reported the number of cigarettes that they typically smoked each day (from 0 to ≥20). For improved validity of self-reported smoking in adolescence, a bogus pipeline procedure was used from 1981 to 1983. As reported elsewhere (Chassin, Presson, Sherman, & Edwards, 1990), a study using an unannounced bioassay with a subsample supported the validity of the self-reports.

To obtain the parent's smoking trajectory group, we used latent class growth analysis on data collected from all eight waves (ages 10–42). Groups that were defined a priori were stable abstainers (those who had never progressed past "trying a cigarette"; 46.8% of parents), stable quitters (who were never measured as smokers, but only as ex-smokers; 8.6% of parents), and relapsing/remitting smokers (who reported periods of smoking, quitting, and then smoking again over the different waves; 12.7% of parents). All other participants were clustered empirically using Proc Traj (Jones, Nagin, & Roeder, 2001; see also Chassin et al., in press, for details and Chassin et al., 2000, for a similar clustering based on the first six waves of data). In addition to the three a priori groups, the latent class growth analysis produced a six-class solution, as follows: experimenters (5.6% of parents, who never smoked more than occasionally and generally quit by age 22), developmentally limited smokers (4.3% of parents, who smoked less than half a pack at their peak and gave up smoking by age 30), successful quitters (2.2% of parents, who started to smoke around age 12 and smoked fairly heavily but quit in adulthood), early-onset persistent smokers (9.7% of parents, who started smoking around age 11, escalated quickly, and smoked more than half a pack per day), high school-onset persistent smokers (7.8% of parents, who started to smoke around age 16 and smoked almost as heavily as the

early-onset group), and late-onset smokers (2.3% of parents, who started to smoke around age 21 and smoked at low levels).[1]

In addition to the trajectory group data, each IU Smoking Survey participant (as well as the child's other parent) was categorized as a current smoker or not, on the basis of whether they had smoked at least monthly at the time of the child measurement. When a parent did not provide data, their current smoking status was determined with an informant report provided by the spouse/partner. Rates of current smoking were 26% for parents who were IU Smoking Survey participants and 27% for the adolescent's other parent. Most of these current smokers were daily smokers (98.4% of IU Smoking Survey participants and 99.1% of other-parent smokers).[2]

Parental education. Education was dichotomized as whether the parent from the larger survey had (27%) or had not attained a bachelor of arts degree or higher by Wave 8 (ages 32–42).[3]

Adolescent personality. Adolescent personality measures included parents' reports of temperamental resistance to control (Bates, 1994), which has successfully predicted externalizing behavior (Goodnight, Bates, Staples, Petit, & Dodge, 2007). There were five items with a 5-point response scale (sample item: "When your child is doing something and someone tells your child not to, how often does your child ignore him/her and keep doing it?"; coefficient $\alpha = .83$ for both fathers' and mothers' reports). On the basis of the correlation between mother and father reports ($r = .49$), their responses were averaged, unless there was only one reporting parent.

Adolescent five-factor model personality was assessed with parents' reports on a shortened version of the Child NEO Personality Inventory (five items for each dimension; John, Caspi, Robins, Moffitt, & Stouthamer-Loeber, 1994). Internal consistencies (coefficient alphas) across the five dimensions averaged .80 for mothers (range = .76–.85) and .79 for fathers (range = .72–.85). On the basis of the correlation between mother and father reports (*r*s ranged from .51 to .66 across dimensions), their responses were averaged unless there was only one reporting parent.

Child smoking outcome. Adolescent smoking was dichotomized on the basis of those who had ever smoked a cigarette (22%) versus those who had never smoked (78%).

Analytic Strategy

We used logistic regression to test the relation between the parent's smoking trajectory and adolescent smoking, first as a bivariate relation and then beyond the parent's current smoking, parent education, and the other parent's current smoking. Because the adolescent's age was not related to parent's smoking trajectory ($\eta = .075$), age was not included in the model. Then, the adolescent's personality variables were added to the model (scores were centered to reduce multicollinearity). We tested mediation using Baron and Kenny's (1986) methods, with coefficients derived from a series of models. Potential mediators were regressed on the smoking trajectory (Path A). Then, the binary outcome variable was regressed on the potential mediators (Path B). The outcome variable was also regressed on parent smoking trajectory (Path C). If both Paths A and B were significant and Path C decreased substantially when a presumed mediator was controlled, findings were consistent with mediation (Baron & Kenny, 1986; Little, Card, Bovaird, Preacher, & Crandall, 2007). We performed analyses using Mplus 4.21 with Monte Carlo numerical integration. Missing data were handled with full information maximum likelihood estimation (Schafer, 1997), and analyses with listwise deletion of cases with missing data produced no change in the pattern of findings.

Results

Bivariate Associations With Parent Smoking Trajectory

Bivariate relations between the parent's smoking trajectory and adolescent smoking as well as the potential mediators were tested either with logistic regressions (for adolescent smoking, parent's current smoking, parent education, and other parent's smoking) or multiple regressions (for adolescent personality characteristics). Scores on these measures as a function of the parent's smoking trajectory are presented in Table 1 (significant pairwise differences for each group compared with the early-onset persistent smoking parents are noted with asterisks).

As shown in Table 1, there was a significant relation between the parent's smoking trajectory and adolescent smoking. The highest rate of adolescent ever smoking (47%) was found for those whose parents were early-onset persistent smokers, and this rate was strikingly higher than that for all other groups. High smoking rates were also found for adolescents with relapsing/remitting parents (34%) and high school-onset persistent parents (30%). Lowered rates of adolescent smoking were found for those whose parents were stable abstainers (14%) and developmentally limited smokers (9%), with moderate rates for adolescents with experimenter and successful-quitter parents (24% each), late-onset parents (23%), and stable-quitter parents (22%). Also, as seen in

[1] The trajectories reported in Chassin et al. (2000) examined earlier ages and produced similar but not identical solutions (see Chassin et al., in press, for details). Chassin et al. (2000) labeled groups as abstainers, experimenters, early stable (now called early-onset persistent), late stable (now called late onset), erratics (now called relapsers/remitters), and quitters (now called developmentally limited smokers to differentiate them from the successful quitters who quit in adulthood and were not identified in the earlier analysis) The current solution also differentiated stable quitters as an a priori group and identified a high school-onset group.

[2] We also estimated models defining parents' current smoking as at least daily and eliminating nondaily smokers, as well as reclassifying nondaily smokers as nonsmokers. There were no changes in findings except that the path from parents' developmentally limited trajectory group to adolescent smoking in the final mediational model became marginally significant ($p < .06$).

[3] Another possibility would be to consider both parents' educational attainment. Categorizing parental education based on the higher of the two parents' educational attainment produced a prevalence of 33% with a bachelor of arts degree or higher (compared with 27% using only the IU Smoking Survey parent). The two measures of parent education were highly intercorrelated ($r = .84$). Moreover, we reestimated the logistic regression model that predicted adolescent smoking from the parent's smoking trajectory group, each parent's current smoking, and parent education, using this alternate definition of parent education. All findings and all significant effects were maintained, and the relation between the parent trajectory group and parent education was somewhat stronger. To more appropriately examine parent educational attainment as a mediator of the parent's smoking trajectory effect, we chose to use the educational attainment of the parent whose trajectory group was measured.

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Table 1
Prevalence of Children's Ever Smoking, Parent's Current Smoking, Parent's Educational Attainment, and Children's Personality Measures by Parent's Smoking Trajectory

Parent's smoking trajectory group (*n*)	Children's ever smoking: % Yes	Parent current smoking: % Yes	Spouse current smoking: % Yes	Parent's BA[a] % Yes	Mean (*SE*) of children's personality measures					
					Open	Agree	Extra	Neurot	Cons	Resist
Group differences[b]	$\chi^2 = 93.91^{***}$	$\chi^2 = 846.57^{***}$	$\chi^2 = 355.02^{***}$	$\chi^2 = 105.04^{***}$	$R^2 = .01$	$R^2 = .02^{*}$	$R^2 = .01$	$R^2 = .01^{**}$	$R^2 = .02^{**}$	$R^2 = .05^{***}$
Overall ($N = 1{,}338$)[c]	22%	26%	27%	27%	4.06 (0.65)	3.77 (0.75)	3.64 (0.73)	2.83 (0.76)	3.37 (0.89)	2.12 (0.64)
Stable abstainers (626)	14%***	2%***	9%***	38%***	4.06 (0.65)	3.84* (0.69)	3.60 (0.75)	2.75** (0.74)	3.46* (0.89)	2.01*** (0.60)
Stable quitters (115)	22%***	6%***	17%***	32%***	4.10 (0.61)	3.80 (0.67)	3.59 (0.73)	2.80 (0.73)	3.37 (0.87)	2.05*** (0.56)
Developmentally limited smokers (57)	9%***	0%[d]	12%***	27%***	3.94 (0.84)	3.69 (0.85)	3.58 (0.69)	2.86 (0.84)	3.18 (0.89)	2.11** (0.60)
Experimenters (75)	24%***	6%***	5%***	21%***	4.05 (0.71)	3.80 (0.81)	3.52 (0.73)	2.79 (0.81)	3.48 (1.05)	2.00*** (0.56)
Relapsing/remitting (170)	34%*	59%***	46%***	15%**	4.08 (0.60)	3.61 (0.80)	3.73 (0.73)	3.00 (0.77)	3.18 (0.85)	2.30 (0.63)
Successful quitters (29)	24%*	17%***	14%***	21%**	3.97 (0.73)	3.85 (0.53)	3.89 (0.60)	2.88 (0.78)	3.54 (0.73)	2.11* (0.58)
High school-onset persistent (105)	30%**	93%	74%	8%	4.15 (0.66)	3.70 (0.83)	3.71 (0.65)	2.90 (0.78)	3.25 (0.92)	2.26 (0.76)
Late-onset smokers (31)	23%*	40%***	37%***	16%*	3.99 (0.68)	3.59 (0.75)	3.53 (0.70)	3.14 (0.66)	3.35 (0.88)	2.30 (0.66)
Referent group Early-onset persistent (130)	47%	89%	78%	4%	3.98 (0.60)	3.63 (0.87)	3.76 (0.70)	2.99 (0.76)	3.25 (0.85)	2.40 (0.80)

Note. Asterisks indicate significant pairwise differences from early-onset persistent smoking parents (the reference group). Open = openness; Agree = agreeableness; Extra = extraversion; Neurot = neuroticism; Cons = conscientiousness; Resist = resistance to control. Higher values indicate higher levels of the personality characteristics.
[a] Parent's bachelor's degree or higher educational attainment. [b] For dichotomous measures, chi-square values are given from logistic regression with maximum likelihood estimation with robust standard errors. For continuous variables, R^2s are given from multiple regression with maximum likelihood estimation with robust standard errors. [c] *N*s vary across analyses because of missing data. [d] Not reported because of a nonpositive, definite first-order derivative product matrix.
$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

Table 1, among parents from the early-onset persistent trajectory, 89% were current smokers, and 96% had less than a BA degree. Finally, the parent's smoking trajectory was significantly, although modestly, related to all adolescent personality characteristics except openness and extraversion. For agreeableness, neuroticism, and conscientiousness, the only group to significantly differ from adolescents whose parents were early-onset persistent smokers was that of adolescents whose parents were stable abstainers. However, adolescents whose parents were stable abstainers, stable quitters, developmentally limited smokers, experimental smokers, and successful quitters all were less resistant to control than were adolescents whose parents were early-onset persistent smokers.

The bivariate associations of all predictors with the adolescent ever-smoking outcome are shown in Table 2, Model A. As noted earlier, adolescents whose parents were early-onset persistent smokers were significantly more likely to smoke than were any other groups. In addition, adolescents were significantly more likely to have ever smoked if their parents were current smokers, were less educated, and were more resistant to control, higher in neuroticism, lower in openness, lower in agreeableness, and lower in conscientiousness. Only extraversion was unrelated to adolescent's ever smoking.

Multivariate Associations

We next estimated a logistic regression model to test whether the parent's smoking trajectory was related to their children's ever-smoking behavior beyond the parent's current smoking, the

Table 2
Coefficients (and Standard Errors) and Odds Ratios From Logistic Regression Analyses of Children's Ever Smoking as a Function of Parent's Smoking Trajectory

Predictor	Children's ever smoking		
	Raw (*SE*)	Standardized	Odds ratio
A. Bivariate unadjusted models			
Parent's smoking trajectory			
Stable abstainers	−1.74 (0.21)***	−0.45	0.18
Stable quitters	−1.17 (0.30)***	−0.17	0.31
Developmentally limited smokers	−2.17 (0.50)***	−0.23	0.11
Experimenters	−1.06 (0.33)***	−0.13	0.35
Relapsing/remitting	−0.57 (0.24)*	−0.10	0.57
Successful quitters	−1.04 (0.47)*	−0.08	0.36
High school-onset persistent smokers	−0.72 (0.28)**	−0.10	0.49
Late-onset smokers	−1.08 (0.47)*	−0.09	0.34
Early-onset persistent smokers	Referent		
Parent's current smoking[a]	1.26 (0.14)***	0.29	3.51
The other parent's current smoking[a]	1.12 (0.16)***	0.26	3.05
Parent's bachelor's degree or higher education[b]	−1.27 (0.21)***	−0.29	0.28
Children's temperamental resistance to control	1.04 (0.10)***	0.34	2.82
Children's extraversion	0.15 (0.10)	0.06	1.16
Children's neuroticism	0.35 (0.10)***	0.15	1.42
Children's openness	−0.29 (0.10)**	−0.10	0.75
Children's agreeableness	−0.69 (0.10)***	−0.27	0.50
Children's conscientiousness	−0.39 (0.08)***	−0.19	0.68
B. Multivariate adjusted model with eight control variables			
Parent's smoking trajectory			
Stable abstainers	−1.19 (0.41)**	−0.28	0.31
Stable quitters	−0.72 (0.47)	−0.10	0.49
Developmentally limited smokers	−2.52 (0.87)**	−0.24	0.08
Experimenters	−0.40 (0.52)	−0.04	0.67
Relapsing/remitting	−0.55 (0.35)	−0.08	0.58
Successful quitters	−1.21 (0.77)	−0.08	0.30
High school-onset persistent smokers	−1.25 (0.39)**	−0.16	0.29
Late-onset smokers	−1.16 (0.59)*	−0.09	0.31
Early-onset persistent smokers	Referent		
Control variables			
Parent's current smoking[a]	0.23 (0.32)	0.05	1.26
The other parent's current smoking[a]	0.44 (0.24)	0.09	1.56
Parent's bachelor's degree or higher education[b]	−0.70 (0.24)**	−0.15	0.50
Children's temperamental resistance to control	0.66 (0.18)***	0.20	1.93
Children's neuroticism	−0.19 (0.14)	−0.07	0.82
Children's openness	−0.01 (0.16)	−0.00	0.99
Children's agreeableness	−0.41 (0.16)**	−0.14	0.66
Children's conscientiousness	0.01 (0.13)	0.00	1.01

[a] Referent is no current smoking. [b] Referent is lower than bachelor's degree.
*$p < .05$. **$p < .01$. ***$p < .001$.

other parent's current smoking, the parent's education, and the personality variables. As is shown in Model B in Table 2, compared with early-onset persistent parents, parents who were stable abstainers (adjusted odds ratio [OR] = 0.31; 95% confidence interval [CI] = 0.14, 0.69), developmentally limited smokers (adjusted OR = 0.08; 95% CI = 0.02, 0.44), high school-onset persistent smokers (adjusted OR = 0.29; 95% CI = 0.13, 0.61), and late-onset smokers (adjusted OR = 0.31; 95% CI = 0.10, 0.99) were less likely to have children who had ever smoked. In addition, lower levels of parent education were significantly associated with a greater likelihood of adolescent smoking. Moreover, adolescents who were more resistant to control and lower in agreeableness were more likely to have ever smoked. Although the significant bivariate relations between adolescent smoking and parents' current smoking were maintained when parent's smoking trajectory and education were entered into the model, they were eliminated when adolescent personality variables were included.

Final Mediational Model

Finally, we constructed a path model to test the effect of the parent's smoking trajectory on adolescent ever smoking mediated through the effects of parent education, adolescent resistance to control, and adolescent agreeableness. These three potential mediators were chosen on the basis of their significant relations to the parent's smoking trajectory and their unique relations to adolescent smoking, beyond the other variables in our multivariate model (i.e., Model B in Table 2). Both parents' current smoking statuses were included as covariates (see Figure 1).

F1

As is shown by the comparison of standardized coefficients in Table 2 and Figure 1, the magnitudes of path coefficients from the parent's smoking trajectory to adolescent smoking were substantially reduced when the mediators were included in the model, and the paths from the three mediators to adolescent smoking remained significant in the mediated model even when other relevant covariates of the outcome variable were controlled. These findings are consistent with parent's educational attainment, adolescent's agreeableness, and adolescent's temperamental resistance to control as mediators in the longitudinal influence of the parent's smoking trajectory on adolescent smoking. However, the mediation was only partial in that there were still significant effects of the parent's smoking trajectory on adolescent ever smoking, even with the mediators included in the model. Specifically, compared with adolescents whose parents were early-onset persistent smokers, those whose parents were abstainers, developmentally limited smokers, or high school-onset persistent smokers were still significantly less likely to have ever smoked, even when the mediators were considered in the model.

Discussion

The first finding of note was that the parent's smoking trajectory group significantly predicted smoking initiation in their adolescent children, beyond the parent's current smoking and the other parent's current smoking. Thus, an important and novel conclusion of this study is that "history matters" in terms of risk for the intergenerational transmission of smoking. Previous studies have shown that adolescents with ex-smoking parents have some elevated risk of smoking, although not to the level of adolescents with current smoking parents (Otten et al., 2007). Our findings extend this work to demonstrate that adolescents whose parents' smoking was of early onset, steep acceleration, high quantity/frequency, and persistent over time were the most likely to themselves have smoked. It is interesting that our earlier work (Chassin et al., 2000) found that individuals in the early-onset persistent trajectory were themselves more likely to have biological parents who smoked. Thus, smoking in this trajectory group is linked to smoking in both the earlier and the later generation. Taken together, these findings suggest that early onset, steeply escalating, high quantity, and persistent smoking may be one de-

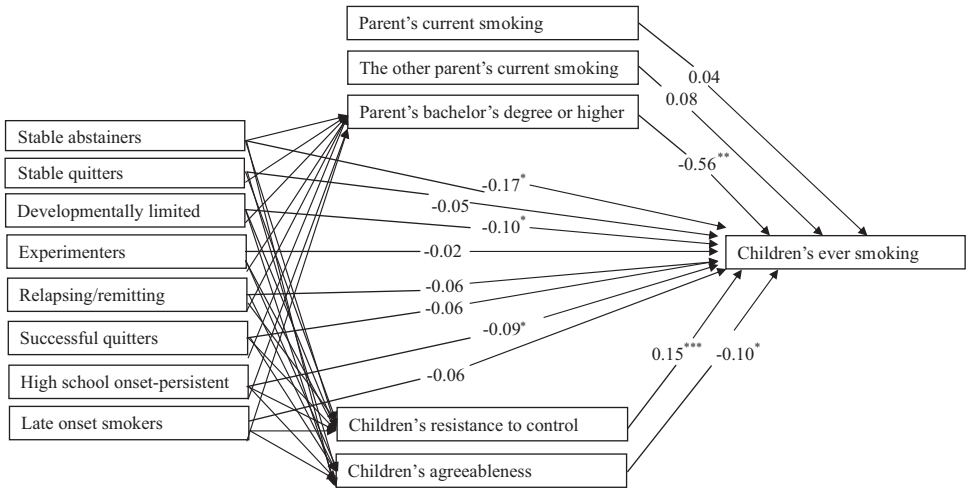

Figure 1. Full path model for children's ever-smoking behavior as a function of parent's smoking trajectory. Values shown are standardized path coefficients. Path coefficients from parent's smoking trajectory to mediators are not shown because of space constraint. All the path coefficients from parent's smoking trajectory to parent's educational attainment and children's agreeableness took a positive sign and those to children's temperamental resistance to control took a negative sign. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

velopmental phenotype of smoking that is at particularly high risk for intergenerational transmission. An important implication of these findings is that this developmental phenotype could be useful for behavioral and molecular genetic studies of smoking etiology.

The present findings are also important for ruling out some mechanisms that could have accounted for this intergenerational transmission. One possibility is that trajectories of parental smoking would matter only because parents in particular trajectory groups would be more likely to be current smokers and (because of assortative mating) that their adolescents' other parent would also be a current smoker. However, we were able to reject this possibility. Thus, the effect of a parent's life course trajectory of smoking on risk for intergenerational transmission cannot be entirely explained through mechanisms such as modeling or cigarette availability in the home, which would be uniquely linked to parents' current smoking rather than to the trajectory of their smoking history. Similarly, our findings showed that parental smoking trajectory effects could not be entirely explained by the parent's educational attainment, although low parental educational attainment has been shown to embed adolescents within social networks characterized by high rates of smoking (Chassin et al., 1992). Thus, although modeling, availability, and lowered educational attainment are all known to influence adolescent smoking, they cannot entirely account for the effects of parents' smoking trajectories.

Finally, we tested whether adolescent personality characteristics could account for the effects of the parent's smoking trajectory on adolescent smoking. Replicating earlier literature, adolescents who were higher in neuroticism, lower in openness, lower in conscientiousness, and lower in agreeableness were more likely to have smoked (Harakeh et al., 2006; Malouff et al., 2006), as were those with high temperamental resistance to control. These findings provide confidence in the validity of the parent-reported personality constructs. However, these personality characteristics could not entirely explain the effects of the parent's trajectory group membership on adolescent smoking. In fact, for the five-factor model characteristics, adolescents whose parents were early-onset persistent smokers significantly differed only from adolescents whose parents were abstainers. Thus, with the exception of resistance to control, personality characteristics were not uniquely linked to parental smoking age of onset, escalation, and persistence. Moreover, although agreeableness and resistance to control were partial mediators, personality characteristics were stronger mediators of parents' current smoking (completely eliminating these effects) rather than parents' smoking trajectory.

Why might these personality characteristics fail to explain parent smoking trajectory effects? The explanation might be methodological in that our measures were relatively brief, and stronger effects might be produced by more comprehensive measures. Moreover, our mediational mechanism hypothesized that early-onset smoking in parents would be produced by high levels of behavioral undercontrol, which would then be transmitted from early onset-smoking parents to their adolescents (through both genetic and parenting pathways). However, five-factor model characteristics are correlates but not direct measures of behavioral undercontrol, and measures that more directly reflect behavioral undercontrol might produce stronger results. This interpretation is consistent with our finding that resistance to control was the only characteristic that differentiated adolescents of early onset-smoking parents from every other group. Finally, our model considered transmission from only one parent. However, the smoking history, personality characteristics, and parenting of the other parent would also be expected to influence the adolescent's personality and smoking. Thus, more complete mediation (rather than partial mediation) might have been produced if the other parent's smoking trajectory could have been considered. Given these possibilities, it would be premature to dismiss adolescent personality characteristics (and, particularly, behavioral undercontrol) as mediators of parent smoking trajectory effects.

Finally, it is possible that other mediators better account for parent smoking trajectory effects. Parent smoking trajectories may be associated with particular general parenting styles (e.g., levels of support and control) and/or parents' attitudes and socialization messages about cigarette smoking (i.e., parents' smoking-specific parenting; Chassin et al., 2005). Parents' smoking histories may influence the attitudes (both explicit and implicit) that they hold about smoking, which, in turn, influence their attempts to deter smoking in their children (Chassin et al., 2002). Moreover, given that early-onset parents show the highest levels of tobacco dependence, individual differences in vulnerability to tobacco effects and to developing dependence could be important mediators. However, differential susceptibility to tobacco dependence would not be expected to influence the current outcome variable, which was "ever smoking" among these young adolescents. Nevertheless, future research should test these possible mediators of parent smoking trajectory effects.

Although the present study makes an important contribution by being the first multigenerational study to link heterogeneity in parental smoking trajectories to the intergenerational transmission of smoking, it also has limitations that might influence our interpretations. As noted earlier, as is characteristic of all longitudinal, multigenerational studies, prospective data on smoking trajectories from adolescence to adulthood were available on only one of the adolescent's two parents. Similarly, at this time, we have limited data on emerging smoking in the next generation and thus could not consider the escalation and persistence of adolescent smoking trajectories. Finally, although our sample is representative of its population, the population itself is predominantly White and well educated, and samples with different demographic characteristics might produce different findings. For example, age of smoking onset has been reported to be later in African Americans than in non-Hispanic Caucasians (e.g., Moon-Howard, 2003), so that trajectories of smoking and their intergenerational transmission may show different patterns in samples of different ethnic compositions.

In short, the present study demonstrated that multiple trajectories of parental smoking varied in their risk for the intergenerational transmission of smoking to their adolescent children. Adolescents whose parents' smoking began at an early age, escalated to high levels, and persisted over time were themselves at highest risk to have smoked. Moreover, this risk was significant beyond both parents' current smoking and the parent's education. Although adolescents' personality characteristics were related to their smoking and to their parents' smoking, these characteristics did not entirely explain the heterogeneity in risk associated with multiple smoking trajectories. These findings suggest that developmental phenotypes of smoking are potentially useful for genetic

studies of smoking etiology and intergenerational transmission. Future research should consider additional mediators of parental smoking trajectory effects, such as parental explicit and implicit attitudes and smoking-specific parenting. Because these mediators are modifiable, they are potentially important targets for family-based smoking prevention campaigns.

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