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Risk Factors for Adolescent Smoking: Parental Smoking and the Mediating Role of Nicotine Dependence

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Abstract

Background—Parental smoking and early-emerging nicotine dependence symptoms are well-documented risk factors for adolescent smoking. However, very little is known about the mediating pathways through which these risk factors may act, or whether parental smoking may cause or signal early-emerging nicotine dependence symptoms.

Methods—Data were drawn from the longitudinal Social and Emotional Contexts of Adolescent Smoking Patterns Study. Adolescents who had smoked under 100 cigarettes in their lifetime ($n=594$; low-exposure group) and adolescents who had smoked over 100 cigarettes, but fewer than 5 cigarettes per day ($n=152$) were included in the analyses. Path analysis was performed on longitudinal data to investigate the association between parental smoking and smoking frequency at the 48 month follow-up, both directly and through mediating variables of smoking frequency, smoking quantity, and nicotine dependence.

Results—Father's smoking was associated with higher adolescent nicotine dependence scores at the baseline assessment wave. Structural equation modeling revealed that mother's smoking at baseline was associated with adolescent's smoking frequency at the 48 month follow-up, and its effect was partially mediated by both smoking frequency and nicotine dependence among low-exposure adolescent smokers.

Conclusions—Parental smoking is a risk factor for future smoking in low-exposure adolescent smokers, above and beyond the risks posed by smoking behavior and nicotine dependence. Moreover, parental smoking is associated with early-onset nicotine dependence in low-exposure adolescent smokers. As an easily measureable risk factor, parent smoking status can be used to identify and intervene with novice adolescent smokers who are at high risk for chronic smoking behavior.

Keywords

Smoking; Adolescent; Nicotine Dependence; Parental Smoking; Structural Equation Modeling

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1. Introduction

As one of the most well-documented risk factors for the development of smoking, parental smoking has been found to predict smoking behavior in both adolescent and adult offspring (Rappaport et al., 1968; Kardia et al., 2003; Hu et al., 2006; Fergusson et al., 2007). Parental smoking has been consistently associated with smoking initiation in offspring (Burchfiel et al., 1989; Bricker et al., 2007a; O'Loughlin et al., 2009), progression to monthly (Bricker et al., 2007a; Bricker et al., 2007b), weekly (Forrester et al., 2007), and daily use (Bricker et al., 2007b; O'Loughlin et al., 2009), and nicotine dependence (Avenevoli and Merikangas, 2003; Kardia et al., 2003; Hu et al., 2006; Fergusson et al., 2007). Hypotheses of the mechanisms of this risk factor posit both environmental and genetic influences.

Environmentally, parental smoking may directly impact smoking in offspring through behavioral modeling (Pedersen and Lavik, 1991; Kalesan et al., 2006; Hoving et al., 2007) and permissiveness towards smoking (Banks et al., 1981; Nolte et al., 1983; McDermott, 1984; Newman and Ward, 1989), or indirectly through positive attitudes toward smoking (Flay et al., 1994) and perceived availability of cigarettes (Woodruff et al., 2003; Doubeni et al., 2008; Doubeni et al., 2009). Ample evidence suggests that parental smoking has hereditary (Boomsma et al., 1994; Koopmans et al., 1999; Maes et al., 1999; Maes et al., 2004) or genetic (Swan et al., 1990; Osler et al., 2001; Vink et al., 2003; White et al., 2003; Maes et al., 2004) influences on current smoking, quantity of cigarettes smoked (Koopmans et al., 1999), and nicotine dependence (Kendler et al., 1999; Broms et al., 2007).

Additionally, prenatal or early postnatal exposure to nicotine can disrupt neural development and consequently alter the brain's response to nicotine later in life (Abreu-Villaca et al., 2004; Slotkin et al., 2006; Gold et al., 2009; Blood-Siegfried and Rende, 2010). This disruption causes higher rates of nicotine self-administration in animals (Levin et al., 2006) and may explain more severe smoking behavior and nicotine dependence in humans (Kandel et al., 1994; Niaura et al., 2001; Kardia et al., 2003).

Most notably, evidence has begun to converge to suggest that parental smoking may partially exert its influence on offspring smoking through nicotine dependence (Kardia et al., 2003; Fergusson et al., 2007). Early-emerging nicotine dependence symptoms, that is, those which manifest at low or less-than-daily levels of smoking (DiFranza et al., 2000; DiFranza et al., 2002), are especially indicative of future adolescent smoking. Parental smoking correlates with nicotine dependence in non-smoking offspring through secondhand smoke exposure (Belanger et al., 2008; Okoli et al., 2009). Furthermore, nicotine dependence may be more strongly hereditary than smoking initiation and experimental levels of use (Heath et al., 1995; Kendler et al., 1999; Sullivan and Kendler, 1999). Taken together, these studies point to the possibility that parental smoking may cause or signal greater sensitivity to early-onset nicotine dependence. That is, youth whose parents smoke may experience nicotine dependence symptoms earlier than those whose parents do not, and thus parental smoking may indirectly increase the likelihood of chronic smoking in offspring.

The present study sought to test this hypothesis using a longitudinal, 6-wave study of developing adolescent smokers by investigating (1) whether parental smoking is associated with early-emerging nicotine dependence symptoms at baseline, (2) whether parental smoking directly or indirectly predicts adolescent smoking frequency 48 months later, and (3) which of several possible mediating pathways, including smoking quantity, smoking frequency, and early-emerging nicotine dependence, contribute to this association. Given the equivocal results of previous research as to the effects of maternal (Burchfiel et al., 1989; Griffin et al., 1999; Avenevoli and Merikangas, 2003; Rohde et al., 2003; Hu et al., 2006) vs. paternal (Burchfiel et al., 1989; Rohde et al., 2004) smoking on risk for adolescent smoking, mother and father smoking were examined separately. Investigating the mediating

pathways of parental smoking among distinct subgroups of adolescent smokers may help improve risk detection as well as suggest effective, personalized intervention strategies.

2. Methods

2.1 Participants

The sample was drawn from the longitudinal Social and Emotional Contexts of Adolescent Smoking Patterns (SECASP) Study. The goal of sampling was to obtain a cohort of adolescents that mirrored the racial and ethnic diversity of the greater Chicago metropolitan area. High schools were selected for recruitment based on geographic location, size, ethnic/racial diversity, willingness to work with research staff in gaining parental consent, and willingness to provide a school liaison to the study, along with space for recruitment and data collection activities. Approximately 35 schools were invited to participate in the study, with a final sample of 16 high schools. All 9th and 10th grade students at these 16 Chicago-area high schools completed a brief screener survey of smoking behavior ($N=12,970$). All students who reported smoking in the past 90 days and had a lifetime exposure of <100 cigarettes were invited to participate, as were all those who said that they smoked in the past 30 days and smoked >100 cigarettes/lifetime. Additionally, random samples of never-smokers and youth smoking <100 cigarettes/lifetime prior to the past 90 days were also invited to participate. Of the 3,654 students invited, 1,344 agreed to participate (36.8%). Of these, 1,263 (94.0%) completed the baseline measurement wave two months after screening. The final baseline sample was 56.5% White, 17.2% Hispanic, 16.9% Black, 4.0% Asian/Pacific Islander, and 0.2% American Indian/Alaska Native, and was on average 15.6 years old.

The SECASP Study contains 6 assessment waves: baseline, 6-month, 15-month, 24-month, 33-months, and 48-months following baseline. Retention at 48 months was 86.5% ($N=1092$). Participants and nonparticipants at 48 months did not differ by gender, race/ethnicity, or age. However, nonparticipation was higher among youth whose parent did not complete the extensive parent questionnaire at baseline ($X^2=29.3$, $d.f.=1$), $p<.0001$. Non-completers reported higher 7-day smoking rates at baseline ($M=0.85$ cigarettes/day, $SD=2.47$ vs. $M=0.42$, $SD=1.33$), $p=.025$; a greater number of days smoked in the past 30 ($M=5.5$ days, $SD=8.75$ vs. $M=3.6$, $SD=7.50$), $p=.007$; and higher numbers of cigarettes smoked on days smoked in the past 30 ($M=1.37$ cigarettes, $SD=3.05$ vs. $M=0.82$, $SD=1.74$), $p=.022$. Non-completers also had lower grade point averages ($M=3.50$, $SD=0.68$ vs. $M=3.77$, $SD=0.76$), $p<.0001$. Two groups, primarily distinguished by the 100 cigarette/lifetime criteria for lifetime smoking status (Center for Disease Control, 2002), were the focus of the present analyses: 594 adolescents who, at baseline, had smoked in the past 90 days, but had smoked <100 cigarettes/lifetime; and 152 who smoked in the past 30 days, smoked >100 cigarettes/lifetime, but <5 cigarettes per day at baseline were the focus of the present analyses. A cutoff of 5 cigarettes/day was used to restrict the study sample to “light smokers” (Shiffman et al., 1994; Owen et al., 1995; Kenford et al., 2005) in line with the aim of studying development of chronic smoking. Demographic and smoking characteristics from the baseline measurement wave are presented in Table 1.

2.2 Measures

2.2.1 Smoking Frequency and Quantity—Smoking was measured with two items at baseline. Participants were asked how many days they smoked cigarettes in the past 30 days (smoking frequency) and how many cigarettes they smoked in the past 7 days (smoking quantity). Both frequency and quantity variables were used for greater model accuracy, since each is independently associated with the latent class of smokers (Sutfin et al., 2009) and degree of nicotine dependence (Caraballo et al., 2009). Smoking frequency was assessed

categorically: 0, 1, 2–3, 4–5, 6–7, 8–10, 11–20, 21–29, and 30 days smoked, and was used in all analyses as a numeric variable using the midpoint of each category. Smoking quantity was assessed numerically and was used as such in all analyses. Intermediate measures of smoking frequency and quantity were obtained by averaging participants' responses from the 6, 15, and 24-month assessment waves. Additionally, smoking frequency at the 48 month follow-up was examined.

2.2.2 Parental Smoking—Parental smoking included adolescent reports of the current smoking status (current smoker, ex-smoker, or never-smoker) of each biological parent at the baseline assessment. Adolescents' reports of parents' smoking have been shown to be very reliable (Harakeh et al., 2006).

2.2.3 Nicotine Dependence—Nicotine dependence was assessed with a shortened version of the Nicotine Dependence Syndrome Scale (NDSS) (Shiffman et al., 2004), modified for use with adolescents. The full NDSS scale was reduced to 10 items based on psychometric analyses conducted on an adolescent sample (Sterling et al., 2009), retaining those items reflecting mainly Drive and Tolerance from the original NDSS due to their high predictive validity for smoking and cessation behavior (Sterling et al., 2009). Research supports the reliability, stability, construct validity, and predictive validity of the NDSS for use with adolescents (Clark et al., 2005; Sledjeski et al., 2007), and the modified version demonstrated strong internal consistency with the current sample (coefficient $\alpha=.93$). Items in the current study were answered on a four-point Likert-type scale, ranging from 1 (not at all true) to 4 (very true), and were averaged to obtain a total NDSS score at baseline. An intermediate NDSS score was obtained from the average of total NDSS scores from the 6, 15, and 24 month follow-up assessments.

2.2.4 Other Tobacco Use—Other tobacco use was measured by asking participants how many days out of the past 30 they a) used chewing tobacco, snuff or dip; b) smoked cigars, cigarillos or little cigars; c) smoked bidis or d) smoked kreteks. Reports from the baseline assessment were dichotomized into any other tobacco use vs. no other tobacco use.

2.3 Analyses

Multiple regression analysis was performed to investigate the cross-sectional association between parental smoking (mother's and father's) and NDSS at baseline, in order to establish whether having a parent who smokes is correlated with experiencing early-emerging nicotine dependence symptoms. This analysis complements the following mediation analysis by informing whether the associations of interest are present at baseline, or instead develop later over the course of smoking development. Path analysis, an application of structural equation modeling, was performed using the software AMOS (version 19.0) to investigate whether mother's and father's smoking status prospectively predicts smoking frequency at the 48 month follow-up. The model investigated both direct effects and mediating pathways of parental smoking on adolescent smoking frequency at 48 months. The model included baseline measures of parental smoking, smoking quantity, smoking frequency, NDSS, and control variables of gender and other tobacco use, as well as intermediate (averaged across 6, 15, and 24 month) measures of smoking quantity, smoking frequency, and NDSS. Paths were added according to the following: every baseline variable had a direct path to the outcome in order to examine main effects. Mediation pathways (i.e., from baseline to intermediate variables, and in turn from these to the outcome) were inserted for 1) mother's and father's smoking in order to test the main question of this study, and 2) baseline measures of each mediating variable in order to account for the autocorrelation within these variables as well as their associations with each other. Mediation of gender and other tobacco use were not tested, with the exception of other tobacco use to intermediate

NDSS which were highly correlated at baseline. A multiple-group model was run with both smoking subgroups (<100 vs. >100 cigarettes/lifetime). In order to test the contribution of each significant path to the overall model fit, nested models were analyzed, in which the original model was compared to one without each of the significant paths

3. Results

Characteristics of the two groups of adolescent smokers, those who smoked <100 cigarettes at baseline and those who smoked >100 cigarettes at baseline, are shown in Table 1. The two groups had an approximately equal composition of gender and Caucasian ethnicity, and had approximately the same mean age. The adolescents who had smoked <100 cigarettes were less likely to have smoked in the past day and past week, less likely to have smoked daily in the last month, and on average smoked fewer cigarettes and smoked less often. At the 48-month follow-up, this group had a lower proportion of adolescents who were currently smoking (past 7 days) and currently smoking daily (past 30 days). The adolescents who had smoked >100 cigarettes were more likely to have parents who smoked at baseline, and less likely to have never-smoking parents.

Cross-sectional multiple regression analysis was performed in order to investigate whether parental smoking status was associated with NDSS at baseline (Table 2). Having a father who smoked was significantly associated with a higher NDSS score ($\beta=1.25$, $p=.043$) after controlling for gender, smoking quantity and frequency, other tobacco use, and subgroup membership. The interaction between group membership and father's smoking was not significant, suggesting that the association between father's smoking and NDSS was similar for both smoking groups. Mother's smoking status was not significantly associated with NDSS after including covariates. A combined effect of having both parents smoke was tested by including an interaction between mother's and father's smoking status; this interaction was not significant (not shown). Smoking subgroup, smoking frequency, other tobacco use, and gender were all significantly associated with NDSS (Table 2). Smoking quantity did not reach significance after smoking frequency was included.

The path analysis model investigated the effects of mother's and father's smoking status, adolescent smoking behavior, and NDSS assessed at baseline on the outcome (48-month smoking frequency), both directly and through possible mediating pathways of smoking behavior and NDSS assessed at the 6, 15, and 24 month waves. Though the χ^2 was high ($\chi^2=107.0$, d.f.=40, $p<.001$), several other fit indices demonstrated a good model fit (a good model fit generally meets the following criteria: $\chi^2/\text{d.f.}$ 5; Wheaton et al., 1977; Marsh and Hocevar, 1985), RMSEA .05 (Browne and Cudeck, 1993), CFI 0.95 (Hu and Bentler, 1998), and TLI 0.9 (Bentler and Bonett, 1980; adjusted χ^2 ($\chi^2/\text{d.f.}$)=2.67; RMSEA (root mean square error of approximation)=.052; CFI (comparative fit index)=.977; TLI (Tucker-Lewis index)=.925).

The effect of mother's smoking on adolescent smoking frequency at 48 months was partially mediated by both intermediate adolescent smoking frequency ($\beta=.099$, $p=.017$) and NDSS ($\beta=.088$, $p=.029$) among those smoking <100 cigarettes at baseline (Figure 1; Table 3). Mother's smoking status was also directly associated with smoking frequency at 48 months ($\beta=.097$, $p=.017$) over and above the mediating pathways and other baseline variables. Father's smoking did not have a significant effect. Importantly, the effect of NDSS at baseline on smoking frequency at 48 months was mediated by intermediate measures of smoking frequency ($\beta=.166$, $p<.001$) and NDSS ($\beta=.47$, $p<.001$).

For adolescents who had smoked <100 cigarettes at baseline, nested models were tested in order to assess the contribution of each significant parental smoking pathway to the overall

model. Each of the three significant paths from mother's smoking was removed from the original model one at a time. Removing the direct path to the outcome significantly decreased the fit of the model ($\chi^2=5.27$, d.f.=1, $p=.022$), as did eliminating either of the mediated pathways to intermediate smoking frequency ($\chi^2=5.63$, d.f.=1, $p=.018$) or intermediate NDSS ($\chi^2=5.75$, d.f.=1, $p=.017$), providing further support that the effect of mother's smoking status on smoking frequency at 48 months is partially mediated by both smoking frequency and NDSS. To determine which of the three levels of mother's smoking was most strongly indicative of future smoking, post-hoc analyses were conducted comparing 1) current smoker vs. ex-smoker or never smoker and 2) current or ex-smoker vs. never smoker. Using the latter dichotomization confirmed the findings that the effect of mother's smoking is partially mediated through both intermediate smoking frequency and NDSS.

For the adolescents who had smoked >100 cigarettes at baseline (Figure 2; Table 3), neither mother's nor father's smoking was significantly associated with smoking frequency at 48 months. Further, baseline NDSS in this group was associated with intermediate NDSS, but neither was associated with future smoking behavior.

4. Discussion

This study investigated the relationship between parental smoking and future smoking frequency among offspring and provided new insight into the ways in which parental smoking may contribute to the development of smoking behavior during adolescence. Among youth who had smoked <100 cigarettes at baseline, mother's smoking, but not father's, was found to partially exert its influence on adolescent smoking frequency at 48 months through early-emerging nicotine dependence symptoms. That is, the effect of mother's smoking was partially mediated by both smoking frequency and nicotine dependence, indicating that the direct association between mother's smoking and later adolescent smoking frequency is partially due to mutual associations with both intermediate smoking frequency and nicotine dependence. These results suggest that, independent of smoking exposure, mother's smoking may signal risk for future smoking in adolescents through greater sensitivity to nicotine dependence symptoms, i.e., experiencing symptoms at very low levels of smoking.

The indirect, mediated pathways between mother's smoking and future adolescent smoking likely capture environmental influences on smoking. For example, the association between mother's smoking and intermediate smoking frequency may reflect adolescents' behavioral modeling of smoking (Pedersen and Lavik, 1991; Kalesan et al., 2006; Hoving et al., 2007) and the perception of parents' permissiveness towards smoking (Banks et al., 1981; Nolte et al., 1983; McDermott, 1984; Newman and Ward, 1989). The association between mother's smoking and intermediate nicotine dependence may partially result from exposure to secondhand smoke (Belanger et al., 2008; Okoli et al., 2009) or genetic factors affecting nicotine dependence (O'Loughlin et al., 2004; Baker et al., 2009; Bergen et al., 2009).

The *direct* association between mother's smoking and future adolescent smoking frequency which is independent of both previous smoking behavior and nicotine dependence symptoms suggests the influence of additional variables. One likely explanation may be a genetic or hereditary propensity such as disinhibition of behavior (Hopfer et al., 2003; Keyes et al., 2008), feelings of satiety (Comings et al., 2001), and the brain's response to nicotine (Arinami et al., 2000; O'Loughlin et al., 2004; Timberlake et al., 2006; Bergen et al., 2009; Franklin et al., 2009; Greenbaum et al., 2010). The present results of mother's smoking at baseline being associated with future, but not concurrent, adolescent smoking is consistent with a genetic contribution, which has a weak influence during adolescence but increases

with the age of the offspring (Vink et al., 2003; White et al., 2003). Genetic variants also may affect different components of nicotine dependence at different ages and may interact with age of regular smoking in influencing phenotypes of nicotine dependence (Weiss et al., 2008; Baker et al., 2009; Ducci et al., 2011). Given the retrospective nature of self-reports in prior genetic studies, this study's detailed, prospective, longitudinal design may provide a better window into examining links between specific gene clusters and vulnerability to and patterning of nicotine dependence. Genetic work (ongoing with the current sample) may help to identify adolescents who are vulnerable to developing nicotine dependence at low levels of smoking exposure.

Father's smoking status was not found to predict adolescents' future smoking behavior, confirming previous research that has shown the association only in relation to mother's smoking (Burchfiel et al., 1989; Griffin et al., 1999; Avenevoli and Merikangas, 2003; Rohde et al., 2003; Hu et al., 2006). Father's smoking status was, however, associated with higher levels of adolescent nicotine dependence at baseline. This apparent inconsistency may be attributable to decreased statistical power due to the higher prevalence of missing data for father's smoking, or alternatively may suggest that father's smoking is related to nicotine dependence early, but not late, in adolescence. Since early-emerging nicotine dependence symptoms have been found to predict future smoking behavior independently of prior smoking patterns (Soteriades et al., 2003; Sledjeski et al., 2007; Dierker and Mermelstein, 2010), father's smoking status may indirectly provide useful information for identifying youth at greatest risk for chronic smoking.

Notably, for the adolescents who had smoked <100 cigarettes at baseline, nicotine dependence was strongly associated with future smoking, independent of previous smoking behavior and parental smoking status. Similar to the effect of mother's smoking, the effect of early-emerging nicotine dependence on future smoking was mediated by intermediate smoking frequency and nicotine dependence symptoms. In contrast, for adolescents who had smoked >100 cigarettes, nicotine dependence was not associated with later smoking behavior. Together, these findings suggest that early-emerging nicotine dependence symptoms among adolescents who have had minimal smoking experience may be an important and underutilized marker for chronic smoking risk that is detectable well before the onset of established smoking behavior. Once more regular smoking patterns are established, however, nicotine dependence symptoms no longer seem to independently predict future smoking.

Although few studies have investigated nicotine dependence symptoms in novice adolescent smokers, our findings are consistent with those of existing studies; Sledjeski et al. (2007) reported that nicotine dependence symptoms predict future smoking quantity only in adolescents with a low baseline level of smoking, and Dierker and Mermelstein (2010) showed that early-emerging nicotine dependence symptoms predict daily smoking 2 years later, over and above smoking behavior. Importantly, the present study extends these previous results to 48 months, and shows that early-emerging nicotine dependence retains its predictive validity after also accounting for the considerable effects of parental smoking. The current findings suggest that individual differences in early-emerging nicotine dependence symptoms may drive smoking patterns at low levels of use, whereas for more experienced smokers, past smoking behavior is a better predictors of future behavior.

Risk factors for chronic smoking are traditionally considered to function by increasing active exposure to cigarettes. In contrast, this study adds to a small but growing body of literature supporting an alternate type of risk factor: one which causes or signals future smoking, is present well before established smoking patterns emerge, and is unlinked to the level of smoking exposure (Dierker and Donny, 2008). In that both mother's smoking and

early-onset nicotine dependence indicated susceptibility to higher levels of smoking frequency over and above how much the adolescent smoked, these seem to explain individual variation in smoking behavior which is not attributable to simple exposure.

The present findings should be interpreted within the context of study limitations. First, the detection of significant results for the adolescents who smoked >100 cigarettes at baseline may have been limited by sample size (n=152). Additionally, the detection of significant results for father's smoking may have been affected by the higher prevalence of missing data for father's compared to mother's smoking. Furthermore, though the overwhelming majority of high-risk smokers were retained in follow-up assessments, the current results may under-report risk associated with future smoking, in that adolescents who dropped out of the study had higher smoking rates at baseline. These results may not generalize to adolescents younger than the current sample, who entered the study around age 16 and initiated smoking around age 12. Finally, the current study cannot inform about environmental versus hereditary contributions to these associations.

Despite these limitations, this study provides one of few longitudinal samples available to date that includes a large group of youth at the earliest stages of smoking exposure. Further, though previous studies have reported an association between parental smoking and nicotine dependence (Kardia et al., 2003; Fergusson et al., 2007), this is the first to investigate this association as one of several mediating pathways for future smoking behavior, and one of few to investigate early-emerging nicotine dependence symptoms in particular.

Although the malleability of smoking behavior at this stage of smoking is unknown, developing intervention strategies for adolescent emerging smokers may be promising. For example, high-risk adolescents could be screened based on parental smoking and early-emerging nicotine dependence symptoms, both highly significant risk factors that are detectable in early adolescence. Since these adolescents have already initiated smoking, targeted interventions would aim to prevent escalation to regular smoking. Additionally, the current results may facilitate family conversations about smoking and benefit family-based smoking education programs, by conveying that mother's smoking causes or signals heightened adolescent nicotine dependence in addition to increased smoking behavior. Identifying co-occurring risk factors and their mechanisms may be promising for both increasing public knowledge about smoking and preemptively intervening with those adolescents at highest risk for lifetime chronic smoking.

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Group 1: Under 100 cigarettes/lifetime

Figure 1. Results of path analysis for adolescents who had smoked under 100 cigarettes at baseline

Significant paths and covariances are denoted by solid, single-headed arrows, and non-significant paths are shown in dotted lines. Covariances are shown with double-headed arrows. Middle column: intermediate variables from the 6, 15, and 24- month assessment waves.

Group 2: Over 100 cigarettes/lifetime

Figure 2. Results of path analysis for adolescents who had smoked over 100 cigarettes at baseline Significant paths and covariances are denoted by solid, single-headed arrows, and non-significant paths are shown in dotted lines. Covariances are shown with double-headed arrows. Middle column: intermediate variables from the 6, 15, and 24- month assessment waves.

Table 1
Demographic and smoking characteristics by subgroup

Baseline characteristic*	Under 100 cigarettes/life [†] N = 594	Over 100 cigarettes/life [‡] N=152
Female sex, n (%)	344 (57.9)	71 (46.7)
Mean age, years, mean (SD)	15.6 (0.62)	15.8 (0.63)
Caucasian ethnicity, n (%)	413 (69.5)	121 (79.6)
Smoked in the past 24 hours, n (%)	70 (11.8)	93 (61.6)
Smoked in the past week, n (%)	230 (39.2)	121 (83.2)
Any other tobacco use in past 30 days, n (%)	189 (32.1)	71 (46.7)
Ever smoked daily in lifetime, n (%)	90 (15.2)	119 (79.3)
Smoked daily in the past month, n (%)	4 (0.7)	45 (29.6)
Age of initiation (puff or more), years, mean (SD)	12.7 (2.03)	11.7 (1.91)
Number of days smoked in past month, mean (SD)	3.2 (5.35)	18.2 (11.06)
Number of cigarettes smoked in past week, mean (SD)	2.01 (5.58)	19.4 (23.6)
NDSS score, mean (SD) of 10 items on a scale of 0–3	0.35 (0.48)	1.27 (0.82)
Mother's smoking status, n (%)		
Current smoker	139 (24.7)	61 (41.5)
Ex-smoker	133 (23.6)	35 (23.8)
Never-smoker	291 (51.7)	51 (34.7)
Father's smoking status, n (%)		
Current smoker	157 (32.5)	58 (43.9)
Ex-smoker	136 (28.2)	27 (20.5)
Never-smoker	190 (39.3)	47 (35.6)
48-month outcome, n (%)		
Positive for past week smoking	248 (49.0)	93 (76.9)
Positive for daily smoking	100 (19.8)	57 (47.1)

* Percentages based on valid responses.

[†] Based on the screening phase of the study, youths who indicated smoking in the past 90 days and who have smoked fewer than 100 cigarettes in their lifetime.

[‡] Based on the screening phase of the study, youths who indicated smoking in the past 30 days, smoked more than 100 cigarettes in their lifetime, but smoke 5 or fewer cigarettes a day.

Table 2
Cross-sectional regression results for the association between parent smoking and NDSS at baseline

For each independent variable, standardized coefficients are shown. Standardized coefficients represent the change in standard deviations of NDSS score with each 1-standard deviation increase in the independent variable.

	Standardized regression coefficients	p-value
Mother's smoking status	0.037	.50
Ex-smoker (vs. never-smoker)	−0.091	.11
Current smoker (vs. never-smoker)		
Father's smoking status	0.024	0.38
Ex-smoker (vs. never-smoker)	1.250	0.043
Current smoker (vs. never-smoker)		
Subgroup (consistent exposure[‡] vs. low exposure[†])	3.112	.0042
Gender (male vs. female)	−0.110	.031
Smoking frequency	0.036	< .0001
Smoking quantity	0.001	.49
Other tobacco use	−.146	.0033

[†]Based on the screening phase of the study, youths who indicated smoking in the past 90 days and who have smoked fewer than 100 cigarettes in their lifetime.

[‡]Based on the screening phase of the study, youths who indicated smoking in the past 30 days, smoked more than 100 cigarettes in their lifetime, but smoke 5 or fewer cigarettes a day.

Table 3
Standardized path coefficients from the path analyses for each smoking group

Standardized path coefficients have the same meaning as standardized multiple regression coefficients, i.e. the change in the dependent variable (in standard deviations) resulting from a 1 standard deviation change in the independent variable.

Path	Under 100 cigarettes/life		Over 100 cigarettes/life	
	Standardized path coefficient	<i>p</i>	Standardized path coefficient	<i>p</i>
Baseline to outcome				
Mother's smoking → 48-month smoking freq.	.097	.017	.043	.603
Father's smoking → 48-month smoking freq.	.051	.233	.106	.222
Smoking freq. → 48-month smoking freq.	-.064	.372	.477	.002
Smoking quant. → 48-month smoking freq.	.093	.146	-.423	<.001
NDSS → 48-month smoking freq.	.007	.894	-.201	.160
Other tobacco use → 48-month smoking freq.	-.089	.018	.089	.245
Gender → 48-month smoking freq.	.168	<.001	-.093	.252
Baseline to intermediate				
Mother's smoking → int. smoking freq.	.099	.017	.047	.561
Mother's smoking → int. smoking quant.	.041	.357	.027	.734
Mother's smoking → int. NDSS	.088	.029	.076	.328
Father's smoking → int. smoking freq.	.044	.319	.016	.849
Father's smoking → int. smoking quant.	.033	.491	.007	.935
Father's smoking → int. NDSS	.025	.562	-.077	.331
Smoking freq. → int. smoking freq.	.480	<.001	.685	<.001
Smoking freq. → int. smoking quant.	.317	<.001	.082	.523
Smoking freq. → int. NDSS	.215	.001	.324	.008
Smoking quant. → int. smoking freq.	-.038	.567	-.092	.374
Smoking quant. → int. smoking quant.	.079	.258	.446	<.001
Smoking quant. → int. NDSS	-.012	.853	-.111	.264
NDSS → int. smoking freq.	.166	<.001	-.076	.523
NDSS → int. smoking quant.	.141	.004	.108	.358
NDSS → int. NDSS	.470	<.001	.403	<.001
Other tobacco use → int. NDSS	.021	.359	.088	.054
Intermediate to outcome				
Int. smoking freq. → 48-month smoking freq.	.272	<.001	.061	.707
Int. smoking quant. → 48-month smoking freq.	.035	.561	.273	.041
Int. NDSS → 48-month smoking freq.	.260	<.001	.143	.392