ORIGINAL INVESTIGATION

Exploring Alternate Processes Contributing to the Association Between Maternal Smoking and the Smoking Behavior Among Young Adult Offspring

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Received September 27, 2012; accepted April 30, 2013

ABSTRACT

Introduction: Maternal smoking during pregnancy (MSP) is a known risk factor for regular smoking in young adulthood and may pose a risk independently of mother's lifetime smoking. The processes through which MSP exerts this influence are unknown but may occur through greater smoking quantity and frequency following initiation early in adolescence or increased sensitivity to nicotine dependence (ND) at low levels of smoking.

Methods: This study used path analysis to investigate adolescent smoking quantity, smoking frequency, and ND as potential simultaneous mediating pathways through which MSP and mother's lifetime smoking (whether she has ever smoked) increase the risk of smoking in young adulthood among experimenters (at baseline, <100 cigarettes/lifetime) and current smokers (>100 cigarettes/lifetime).

Results: For experimenters, MSP was directly associated with more frequent young adult smoking and was not mediated by adolescent smoking behavior or ND. Independently of MSP, the effect of mother's lifetime smoking was fully mediated through frequent smoking and was heightened ND during adolescence. Controlling for MSP eliminated a previously observed direct association between mother's lifetime smoking and future smoking among experimenters. For current smokers, only prior smoking behavior was associated with future smoking frequency.

Conclusions: These results seem to rule out sensitivity to ND and increased smoking behavior as contributing pathways of MSP. Further, the impact of MSP on young adult smoking extends beyond that of having an ever-smoking mother. Future work should test other possible mediators; for example, MSP-related epigenetic changes or gene variants influencing the brain's nicotine response.

INTRODUCTION

Cigarette smoking is a serious public health issue that remains the primary cause of preventable death in the United States (Armour, Woolery, Malarcher, Pechacek, & Husten, 2005; Mokdad, Marks, Stroup, & Gerberding, 2004). One fifth of U.S. adults currently smoke (Pleis, Ward, & Lucas, 2009), putting them at risk for a myriad of health problems and a reduced life expectancy. The majority of smokers initiate smoking by their mid-teens (Anderson, Burns, Dodd, & Feuer, 2012; Rath, Villanti, Abrams, & Vallone, 2012), highlighting the importance of studying smoking development during adolescence.

Parental smoking is one of several well-known risk factors that increase the likelihood of regular smoking in offspring. Parental smoking increases rates of smoking initiation (Bricker et al., 2007; Burchfiel, Higgins, Keller, Butler, & Donahue,

1989), progression to heavy/daily smoking (Bricker et al., 2007; Griffin, Botvin, Doyle, Diaz, & Epstein, 1999; Hu, Davies, & Kandel, 2006; Melchior, Chastang, Mackinnon, Galera, & Fombonne, 2010), and nicotine dependence (ND; Avenevoli & Merikangas, 2003; Hu et al., 2006). As a complex risk factor, it potentially includes several environmental and hereditary mechanisms. Environmental explanations include behavioral modeling of parents' smoking (Kalesan, Stine, & Alberg, 2006; Pedersen & Lavik, 1991), parental permissiveness (Banks, Bewley, & Bland, 1981) and attitude toward smoking (Flay et al., 1994; Nolte, Smith, & O'Rourke, 1983), and perceived cigarette availability (Doubeni, Li, Fouayzi, & Difranza, 2008; Woodruff, Candelaria, Laniado-Laborin, Sallis, & Villaseñor, 2003). Smoking has hereditary components (Boomsma, Koopmans, Van Doornen, & Orlebeke, 1994; Koopmans, Slutske, Heath, Neale, & Boomsma, 1999; Swan, Carmelli,

doi:10.1093/ntr/ntt072

Advance Access publication June 12, 2013

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Rosenman, Fabsitz, & Christian, 1990), such as inheritable personality characteristics (Heath, Madden, Slutske, & Martin, 1995) and gene variants that affect one's nicotine response (Arinami, Ishiguro, & Onaivi, 2000; O'Loughlin et al., 2004; Timberlake et al., 2006; Weiss et al., 2008).

A special case of parental smoking is mother's smoking during pregnancy (MSP), which is also associated with offspring smoking behavior and ND (Lawlor et al., 2005; O'Callaghan et al., 2009; Porath & Fried, 2005; Tehranifar, Liao, Ferris, & Terry, 2009). Though both risk factors share many of the above environmental and hereditary mechanisms, MSP poses additional negative behavioral risks to offspring beyond mother's lifetime or postnatal smoking. After controlling for mother's smoking, MSP is associated with higher ND in offspring (Kardia, Pomerleau, Rozek, & Marks, 2003; Lieb, Schreier, Pfister, & Wittchen, 2003), increased or earlier smoking initiation, and heavier smoking among adolescent girls and adult offspring (Cornelius, Leech, Goldschmidt, & Day, 2005; Kandel, Wu, & Davies, 1994). Additionally, heavy MSP ($\geq$ half pack/day) is associated with a higher risk of tobacco dependence among adult offspring (Buka, Shenassa, & Niaura, 2003) and heightened susceptibility to substance use as manifest in disruptive behavior (Jacobsen, Slotkin, Westerveld, Mencl, & Pugh, 2006; Wakschlag et al., 2010) and early initiation of multiple substance use (Goldschmidt, Cornelius, & Day, 2012).

Several theories describe possible mechanisms through which MSP could act. First, MSP has been suggested to have direct behavioral teratologic effects, that is developmental abnormalities stemming from the *in utero* environment (Wakschlag et al., 2010; Wakschlag, Pickett, Cook, Benowitz, & Leventhal, 2002): evidence suggests that MSP disrupts developing neural systems that promote addictive behaviors (Jacobsen et al., 2006; Kandel et al., 1994). Alternately, behavior genetic studies suggest that MSP may instead signify parental smoking liability (D'Onofrio et al., 2008). Additionally, heavier smoking shortly following initiation, due to environmental and/or genetic sources, may contribute to continued heavy smoking in young adulthood. For example, certain genetic effects on smoking in adulthood are mediated by heavier smoking in adolescence (Ducci et al., 2011). Finally, MSP may also act by altering dopaminergic systems, increasing offspring sensitivity to ND. For example, MSP is hypothesized to increase an offspring's initial sensitivity to ND, even prior to smoking initiation, resulting in more persistent smoking patterns (Kandel et al., 1994). A longitudinal examination of the association between MSP and lifetime maternal smoking on youth smoking outcomes, and the contributions of simultaneous mediating pathways, are needed to disentangle these issues.

Following these existing hypotheses, this study specifically tests whether the effects of MSP and mother's ever-smoking status on young adults' smoking behavior are mediated through smoking exposure and/or through ND earlier during adolescence. Mother's ever-smoking may act through increased cigarette consumption in adolescence (e.g., via perceived availability of cigarettes or genetic risk) or alternatively through increased sensitivity to ND (e.g., via secondhand smoke exposure or genetic risk). MSP could also act through increased smoking exposure during adolescence (e.g., via different genetic risks) and/or through ND (e.g., through teratological or epigenetic effects). Genetic risks and teratological effects could additionally manifest in direct associations with young adult smoking. A recent study showed the association

between mother's smoking and young adult smoking to be partially mediated through both adolescent smoking frequency and early emerging ND symptoms, with a remaining direct link that suggests an as yet unaccounted-for pathway (Selya, Dierker, Rose, Hedeker, & Mermelstein, 2012).

This study takes advantage of a unique longitudinal cohort oversampled for novice smokers to examine whether MSP and maternal ever-smoking are independent risk factors, and which of several mediating pathways explain their associations with young adult smoking behavior. Two smoking groups (experimenters and current smokers) were analyzed separately within a longitudinal sample of 9th and 10th grade adolescents. This study tests the hypotheses that (a) MSP and mother's ever-smoking are independently associated with offspring smoking frequency in young adulthood, (b) one or both of these effects are mediated in adolescence through mediating pathways of smoking frequency, smoking quantity, and/or ND, and (c) MSP explains the previously unaccounted-for association between mother's ever-smoking and young adult smoking frequency.

METHODS

Participants

The sample was drawn from the longitudinal Social and Emotional Contexts of Adolescent Smoking Patterns (SECASP) Study, which has been described previously (Dierker & Mermelstein, 2010). Sampling was designed to mirror the racial/ethnic diversity of the greater Chicago metropolitan area. Approximately 35 high schools were invited to participate, of which 16 agreed. Within these, all 9th and 10th grade students completed a brief screener survey of smoking behavior ($N = 12,970$). All novice (<100 cigarettes/lifetime) and light (≤ 5 cigarettes/day) smokers, and random samples of nonsmokers were invited to participate. Of the 3,654 invited, 1,344 agreed to participate (36.8%). Agreement to participate did not differ by race/ethnicity, smoking status, or parental smoking, but was higher for females. Of these 1,344, 1,263 (94.0%) completed the baseline assessment.

The SECASP Study contains additional assessment waves 6, 15, 24, 33, and 48 months following baseline. Retention at 48 months was 86.5% ($N = 1,092$). Participation through 48 months did not differ by gender, race/ethnicity, or age, but was lower among youth whose parent did not complete the parent questionnaire at baseline ($\chi^2 = 29.3$, $df = 1$), $p < .0001$. Participants with parent noncompleters reported higher 7-day smoking rates at baseline ($M = 0.85$ cigarettes/day, $SD = 2.47$ vs., $M = 0.42$, $SD = 1.33$), $p = .025$; more frequent past 30-day smoking ($M = 5.5$ days, $SD = 8.75$ vs., $M = 3.6$, $SD = 7.50$), $p = .007$; more cigarettes smoked per smoking day in the past 30 days ($M = 1.37$ cigarettes, $SD = 3.05$ vs. $M = 0.82$, $SD = 1.74$), $p = .022$; and lower grade point averages ($M = 3.50$, $SD = 0.68$ vs. $M = 3.77$, $SD = 0.76$), $p < .0001$. Adolescents' eligibility was not affected by parent participation; however, this study excludes participants with parent noncompleters, based on missing MSP data ($N = 147$). Two smoking groups categorized at baseline were the focus of the present analyses ($N = 599$): experimenters (472 adolescents who smoked in the past 90 days but smoked <100 cigarettes/lifetime, falling short of the criterion for lifetime smoking status [Center for Disease Control, 2002]); and current smokers (127 adolescents

who smoked >100 cigarettes/lifetime and smoked in the past 30 days, but smoked ≤5 cigarettes/day, in order to study light and novice smokers). These groups were modeled separately, since they have been shown to have different risk factors and pathways (Dierker & Mermelstein, 2010; Selya, Dierker, Rose, Hedeker, & Mermelstein, 2012). Demographic and smoking characteristics from the baseline measurement wave are presented in Table 1.

Measures

Smoking Frequency and Quantity

Participants were asked at baseline on how many days they smoked cigarettes in the past 30 days (frequency) and the total number of cigarettes they smoked over the past 7 days (quantity). Smoking frequency was assessed categorically: 0, 1, 2–3, 4–5, 6–7, 8–10, 11–20, 21–29, and 30 days smoked, and the midpoint of each category was used numerically. Smoking quantity was assessed numerically. Intermediate measures of smoking frequency and quantity were obtained by averaging responses from the 6-, 15-, and 24-month assessments. The outcome is smoking frequency at 48 months during young adulthood (approximately age 19).

Maternal Smoking During Pregnancy

The parent questionnaire was completed by 513 mothers and 86 fathers (who reported on the mother's prenatal smoking). Father's proxy reports of pregnant mothers' current smoking behavior are reasonably accurate (Hatch, Misra, Kabat, & Kartzmer, 1991) though retrospective accuracy is unknown. MSP was assessed using methods that increase reliability of retrospective self-reports (Pickett, Kasza, Biesecker, Wright, & Wakschlag, 2009), that is asking the amount smoked per trimester (did not smoke, <1 cigarette/week, ≥1 cigarette/week,

1–9 cigarettes/day, 10–19 cigarettes/day, and ≥20 cigarettes/day). MSP was dichotomized into any smoking during ≥2 trimesters versus during 0 or 1 trimesters, based on preliminary analyses showing no effect of any versus no MSP; this ensured that children of “spontaneous quitters” (i.e., mothers who quit smoking upon learning of their pregnancy) would not be considered persistently exposed. Mothers who smoked during one trimester were overwhelmingly (26 of 30) spontaneous quitters, smoking only during the first trimester; the remaining four smoked only during the third trimester. Only two mothers smoked during two trimesters, both during the first and second. Ninety-nine mothers smoked throughout pregnancy. Overall, 16.9% had MSP according to this dichotomization of ≥2 versus ≤1 trimesters. Of those, 90.1% had MSP to daily smoking, and 33.7% were exposed at a level (≥half pack/day) found to predict behavioral risks in offspring (Wakschlag et al., 2002).

Mothers' Ever-Smoking

Adolescents reported the current smoking status (current, ex-smoker, or never-smoker) of each biological parent at baseline. Preliminary analyses showed no effects of father's smoking or of mother's current smoking, thus only mother's ever-smoking (current and ex-smoker vs. never-smoker) was examined. Adolescents' reports of parents' smoking have been shown to be very reliable (Harakeh, Engels, Vries, & Scholte, 2006). Within the sample, 50.6% had ever-smoking mothers. Of the 479 mothers who did not persistently prenatally smoke, 43.4% had ever smoked by baseline.

Youth Nicotine Dependence

ND was assessed with a shortened version of the Nicotine Dependence Syndrome Scale (NDSS; Shiffman, Waters, & Hickcox, 2004), shortened for use in adolescents (Sterling

Table 1. Demographic and Smoking Characteristics by Subgroup

Baseline characteristic ^a	Experimenters ^b , <i>N</i> = 472	Current smokers ^c , <i>N</i> = 127
Female sex, <i>n</i> (%)	277 (58.7)	60 (47.2)
Age, years, mean (<i>SD</i>), (range)	15.6 (0.59), (13.9–17.2)	15.8 (0.62), (14.4–16.7)
Caucasian ethnicity, <i>n</i> (%)	330 (69.9)	105 (82.7)
Smoked in the past 24 hr, <i>n</i> (%)	60 (12.7)	81 (64.3)
Smoked in the past week, <i>n</i> (%)	177 (38.0)	105 (84.7)
Any other tobacco use in past 30 days, <i>n</i> (%)	144 (30.5)	55 (43.3)
Ever smoked daily in lifetime, <i>n</i> (%)	74 (15.7)	102 (81.6)
Smoked daily in the past month, <i>n</i> (%)	4 (0.8)	38 (29.9)
Age of initiation (puff or more), years, mean (<i>SD</i>)	12.3 (3.11)	11.8 (1.88)
Number of days smoked in past month, median (IQR)	1 (0, 4.5)	25 (9, 30)
Number of cigarettes smoked in past week, mean (IQR)	0 (0, 2)	13 (3, 27.5)
NDSS score, mean (IQR) of 10 items on a scale of 0–3	0.1 (0, 0.5)	1.2 (0.8, 1.9)
Maternal smoking during pregnancy, <i>n</i> (%)	72 (15.3)	29 (22.8)
Mother's smoking status, <i>n</i> (%)		
Current smoker	106 (23.5)	52 (42.3)
Ex-smoker	114 (25.3)	31 (25.2)
Never-smoker	231 (51.2)	40 (32.5)
48-Month smoking frequency outcome, mean (IQR)	3 (0, 25)	28 (4, 30)

Note. IQR = Interquartile range; NDSS = Nicotine Dependence Syndrome Scale; *SD* = standard deviation.

^aPercentages based on valid responses.

^bBased 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.

^cBased 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.

et al., 2009), retaining Drive and Tolerance items that have predictive validity (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 were answered on a 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 by averaging these scores from the 6-, 15-, and 24-month assessments.

Other Tobacco Use

Participants reported how many days in 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. Baseline reports were dichotomized into any versus no other tobacco use.

Analyses

Simple linear regressions examining the effect of (a) MSP and (b) mother's ever-smoking on 48-month smoking frequency were performed using the software AMOS (version 19.0) in order to obtain each variable's direct, unadjusted effect.

Path analysis was performed using AMOS to investigate potential mediating roles of post-initiation smoking behavior and early emerging ND on the relationship between MSP and 48-month smoking frequency. Path analysis is an extension of regression models that allows multiple simultaneous outcomes or mediators. The model included MSP, baseline measures of maternal ever-smoking, smoking quantity, smoking frequency, NDSS, gender, and other tobacco use, as well as intermediate measures of adolescent smoking quantity, smoking frequency, and NDSS. Every baseline variable had a direct path to the outcome (i.e., direct effects). Mediating pathways (i.e., from baseline to each of the three intermediate variables, and from these to the outcome) were modeled for MSP, and mother's ever-smoking, adolescent smoking quantity and frequency, and ND at baseline. Finally, a mediating pathway from other tobacco use through intermediate NDSS was added, due to their correlation at baseline. A multiple-group model examined experimenters and current smokers separately. In order to test the contribution of each significant path to the overall model fit, nested models were run, in which the original model was compared with one without each of the significant paths.

RESULTS

Characteristics of the two groups of adolescent smokers at baseline are shown in Table 1. The groups did not differ by gender, Caucasian ethnicity, or age. The current smokers were marginally more likely to have had MSP ($\chi^2 = 3.579$, $df = 1$, $p = .058$) and less likely to have never-smoking mothers ($\chi^2 = 12.819$, $df = 1$, $p = .0003$). The current smokers smoked more heavily than the experimenters on several quantity and frequency measures (all $p < .0001$), consistent with the difference in definitions of these two groups.

Linear regressions examining each direct, unadjusted effect of MSP and mother's ever-smoking revealed that, for experimenters, MSP ($B = 5.834$; 95% confidence interval

[CI] = 2.580, 9.088; $p < .001$) and mother's ever-smoking ($B = 4.143$; $CI = 1.750, 6.540$; $p < .001$) were each significantly associated with 48-month smoking frequency. For current smokers, neither MSP ($B = 5.479$; $CI = -0.150, 11.108$; $p = .056$) nor mother's ever-smoking ($B = 0.976$; $CI = -4.226, 6.178$; $p = .713$) was significantly associated with the outcome.

The path analysis investigating the direct and mediated effects of MSP and mother's ever-smoking on young adult smoking at 48 months after baseline fit well according to the following guidelines: adjusted $\chi^2(\chi^2/df) \leq 5$ (Marsh & Hocevar, 1985; Wheaton, Muthen, Alwin, & Summers, 1977), Comparative Fit Index (CFI) ≥ 0.95 (Hu & Bentler, 1998), Tucker-Lewis Index (TLI) ≥ 0.9 (Bentler & Bonett, 1980), and root mean square error of approximation (RMSEA) $\leq .05$ (Browne & Cudeck, 1993). Though the χ^2 value of the present model was high ($\chi^2 = 84.899$, $df = 38$, $p < .001$), the other indices showed a good fit ($\chi^2/df = 2.234$; CFI = 0.983; TLI = 0.942; RMSEA = 0.045).

Among experimenters (Figure 1; Table 2), the association between mother's ever-smoking and 48-month smoking frequency was fully mediated through both intermediate smoking frequency ($B = 1.763$, $p = .008$) and ND ($B = .143$, $p = .005$). In contrast, MSP was directly associated with 48-month smoking frequency ($B = 5.07$, $p < .001$) and was not mediated by intermediate smoking behavior or ND.

Among current smokers (Figure 2; Table 2), neither MSP nor mother's ever-smoking was associated with young adult smoking frequency. Instead, only baseline smoking behavior was associated with 48-month smoking frequency, including a direct effect of smoking frequency ($B = .687$, $p < .001$), and a partially mediated effect of smoking quantity (direct: $B = -.288$, $p < .001$; through intermediate smoking quantity: $B = .466$, $p < .001$). The direct component was negative only after controlling for smoking frequency.

Nested models showed that removing the direct path from MSP to 48-month smoking frequency significantly decreased the model fit ($\chi^2 = 12.129$, $df = 1$, $p < .001$), as did removing the mediating path from mother's smoking to intermediate smoking frequency ($\chi^2 = 6.972$, $df = 1$, $p = .008$), and the mediating path from mother's smoking to intermediate ND ($\chi^2 = 7.862$, $df = 1$, $p = .005$). These results confirm that these paths contribute significantly to the fit of the original model.

DISCUSSION

This study investigated the effects of MSP and mother's ever-smoking on young adults' smoking frequency, and whether these effects are mediated through smoking frequency, smoking quantity, and ND during adolescence. Among experimenters, MSP was directly associated with young adults' smoking frequency 4 years after baseline, without mediation. Mother's lifetime smoking had an independent, fully mediated effect on young adults' smoking frequency, suggesting that mother's smoking signals a risk for regular smoking in young adult offspring through (a) more frequent smoking soon after initiation and (b) greater sensitivity to ND at low levels of smoking.

The current results reveal that MSP poses a heightened risk of smoking to young adult offspring that extends beyond that of mother's lifetime smoking. This finding is consistent with previous research reporting that MSP poses risks beyond that of mother's ever-smoking (Kardia et al., 2003) or postnatal

Experimenters

Figure 1. Path analysis model for experimenters. Baseline variables are shown on the left, intermediate mediating variables are shown in the middle, and the outcome (48-month smoking frequency) is shown on the right. Solid lines represent significant ($p < .05$) paths, and dotted lines are not significant.

smoking (Kandel et al., 1994; Lieb et al., 2003), particularly maternal smoking late in pregnancy (O’Callaghan et al., 2006). Since MSP was examined independently of mother’s ever-smoking, its effects are likely independent from sharing an environment with a smoking mother and genetic liabilities for mother’s lifetime smoking.

The direct effect of MSP seems to rule out heavier smoking and ND during adolescence as a risk factor. However, MSP may be associated with ND beyond the current time frames; previous research has shown MSP to predict higher ND among adult offspring (Buka et al., 2003; Kardia et al., 2003). Alternatively, this result may indicate the presence of an unaccounted-for mediator, potentially one involving time delays that could explain the late emergence of MSP’s effect during young adulthood. One likely candidate is MSP’s direct teratologic effects on the developing brain (Wakschlag et al., 2002): MSP may cause epigenetic changes (Toledo-Rodriguez et al., 2010) and disrupt neural development (Dwyer, McQuown, & Leslie, 2009) in a way that results in a long-term abnormal response to nicotine (Blood-Sieffried & Rende, 2010; Gold, Keller, & Perry, 2009; Slotkin, Tate, Cousins, & Seidler, 2006). If the development of these neural pathways is still incomplete during adolescence, the effects of MSP may not manifest until young adulthood. Alternatively, MSP’s effect on only young adult smoking frequency is consistent with genetic effects being weak during adolescence but stronger in adulthood (Vink, Willemsen, & Boomsma, 2003; White, Hopper, Wearing, & Hill, 2003) and under conditions of lower parental monitoring (Dick et al., 2007), which likely occurs upon entering young adulthood.

The fully mediated effect of mother’s smoking, via more frequent smoking and ND among adolescents soon after initiating smoking, is consistent with prior studies showing that early emerging ND symptoms (i.e., those occurring soon after smoking initiation and before smoking daily; DiFranza et al., 2000, 2002) predict future smoking behavior (Dierker & Mermelstein, 2010). These effects of mother’s lifetime smoking

likely capture a variety of environmental factors, for example exposure to secondhand smoke, which increases the risk of ND (Belanger et al., 2008; Okoli, Richardson, Ratner, & Johnson, 2009), behavioral modeling (Kalesan et al., 2006; Pedersen & Lavik, 1991), parental views of smoking (Banks et al., 1981; Flay et al., 1994; Nolte et al., 1983), and perceived availability of cigarettes (Doubeni et al., 2008; Doubeni, Li, Fouayzi, & DiFranza, 2009; Woodruff et al., 2003). Additionally, mother’s lifetime smoking may transmit separate genetic risks than does MSP, since specific genetic factors influence different aspects of smoking independently (Maes et al., 2004). In particular, genetic factors associated smoking initiation (Kendler et al., 1999; Koopmans et al., 1999), risk-taking/disinhibited behavior (Hopfer, Crowley, & Hewitt, 2003; Keyes, Legrand, Iacono, & McGue, 2008), or ND (Greenbaum et al., 2010; O’Loughlin et al., 2004; Weiss et al., 2008) may partially explain the effects of mother’s lifetime smoking on adolescent smoking frequency and ND.

Taken together with a previous study investigating mediating effects of mother’s and father’s smoking on young adult smoking frequency (Selya, Dierker, Rose, Hedeker, & Mermelstein, 2012), the current results reveal that when examining MSP independently of mother’s ever-smoking, the previously observed direct effect of mother’s smoking no longer reaches significance. This finding indicates that MSP is an important component of maternal smoking history that may act through different pathways, and suggests the importance of examining the two independently. Future research is needed to further examine what may explain MSP’s direct effect and to disentangle environmental versus genetic contributions.

Future studies utilizing forthcoming genetic data from the current sample have the potential to elucidate specific genetic factors associated with MSP or mother’s ever-smoking. Further, it is possible that MSP exacerbates genetic predispositions to smoking. A gene-by-MSP interaction has been identified for other behavioral outcomes, including antisocial behavior among youth (Wakschlag et al., 2010), raising the

Table 2. Unstandardized Path Coefficients (B) and 95% CI

Path	Experimenters		Current smokers	
	B	95% CI	B	95% CI
Baseline to outcome				
MSP → 48-month smoking frequency	5.07	2.14, 7.98	3.21	-2.29, 8.71
Mother's smoking → 48-month smoking frequency	-0.14	-2.28, 2.00	-1.29	-6.01, 3.43
Smoking frequency → 48-month smoking frequency	-0.10	-0.44, 0.24	0.69	0.32, 1.05
Smoking quantity → 48-month smoking frequency	0.14	-0.20, 0.49	-0.29	-0.42, -0.15
NDSS → 48-month smoking frequency	1.14	-2.01, 4.29	-3.54	-8.45, 1.36
Other tobacco use → 48-month smoking frequency	-3.59	-5.66, -1.52	-3.74	-8.10, 0.62
Gender → 48-month smoking frequency	4.28	2.35, 6.21	1.78	-2.33, 5.90
Baseline to intermediate				
MSP → intermediate smoking frequency	-0.67	-2.46, 1.12	4.15	-0.02, 8.31
MSP → intermediate smoking quantity	-1.07	-4.13, 1.99	4.81	-6.99, 16.6
MSP → intermediate NDSS	-0.10	-0.24, 0.03	0.10	-0.20, 0.40
Mother's smoking → intermediate smoking frequency	1.76	0.46, 3.06	-1.44	-5.04, 2.17
Mother's smoking → intermediate smoking quantity	2.17	-0.06, 4.39	-1.54	-11.76, 8.67
Mother's smoking → intermediate NDSS	0.14	0.04, 0.24	0.06	-0.20, 0.32
Smoking freq. → intermediate smoking frequency	0.62	0.42, 0.81	0.53	0.28, 0.79
Smoking frequency → intermediate smoking quantity	0.39	0.05, 0.73	0.11	-0.61, 0.82
Smoking frequency → intermediate NDSS	0.02	0.01, 0.04	0.02	0.00, 0.04
Smoking quantity → intermediate smoking frequency	-0.08	-0.29, 0.13	-0.00	-0.10, 0.09
Smoking quantity → intermediate smoking quantity	0.41	0.05, 0.77	0.47	0.20, 0.73
Smoking quantity → intermediate NDSS	-0.00	-0.02, 0.01	-0.00	-0.01, 0.01
NDSS → intermediate smoking frequency	3.40	1.78, 5.02	-0.52	-3.89, 2.85
NDSS → intermediate smoking quantity	4.82	2.04, 7.60	6.99	-2.55, 16.53
NDSS → intermediate NDSS	0.67	0.54, 0.79	0.38	0.14, 0.63
Other tobacco use → intermediate NDSS	0.01	-0.04, 0.07	0.23	0.09, 0.39
Intermediate to outcome				
Intermediate smoking frequency → 48-month smoking frequency	0.49	0.22, 0.76	-0.03	-0.43, 0.37
Intermediate smoking quantity → 48-month smoking frequency	0.05	-0.08, 0.17	0.16	0.05, 0.27
Intermediate NDSS → 48-month smoking frequency	4.347	0.99, 7.70	1.59	-3.75, 6.94

Note. CI = confidence interval; MSP = Maternal smoking during pregnancy; NDSS = Nicotine Dependence Syndrome Scale. CI from the path analysis for each smoking group.

Unstandardized path coefficients are to be interpreted in the same way as unstandardized coefficients from multiple regression, that is the change in units of the dependent variable resulting from a 1-unit change in the independent variable. Coefficients for the categorical variables of mother's ever-smoking, MSP, gender and other tobacco use reflect differences in the outcome relative to the reference group (never-smoking mother, no MSP, female gender, no other tobacco use). Bold: $p < .05$.

question of whether MSP similarly moderates genetic susceptibility for regular smoking. Alternatively, future studies could examine whether MSP is associated with different physiological responses to nicotine. For example, premature stimulation of nicotinic receptors may lead to hypersensitivity to ND upon initiating smoking in adolescence (Abreu-Villaca, Seidler, Tate, Cousins, & Slotkin, 2004). Since adolescents alter their smoking topographies (e.g., puffs/cigarette, puff volume, inter-puff interval) in response to nicotine content (Kassel et al., 2007), it is possible that MSP causes or signals higher tolerance at initiation and prompts adolescents to smoke in a way that extracts more nicotine. In turn, they may develop more severe ND, which mediates the effect of MSP on more frequent future smoking.

The current findings include negative results among current smokers, for whom neither MSP nor mother's smoking predicted young adult smoking frequency. This difference between experimenters and current smokers may reflect different sets of risk factors across heterogeneous subpopulations of smokers (Dierker & Mermelstein, 2010), or across different stages of smoking (Selya, Dierker, Rose, Hedeker, Tan

et al., 2012). Alternatively, given the relatively large path coefficients between MSP and intermediate and 48-month smoking frequency, it is possible the lack of significance reflects low statistical power due to the much smaller sample size of current smokers relative to experimenters. Another negative result is the lack of significant gender differences in path coefficients (data not shown), despite previous literature showing an association between MSP and smoking only among female offspring (Roberts et al., 2005; Rydell, Cnattingius, Granath, Magnusson, & Galanti, 2012). It is possible that gender differences would emerge with a larger sample size. Also, this discrepancy could be due to the current study's use of lifetime (rather than postnatal) maternal smoking and incorporation of mediating pathways.

Several limitations should be taken into account when interpreting the current results. First, the small sample size may limit the ability to detect significant results (especially for current smokers) and precluded investigation of MSP dose effects. Though study participants and nonparticipating invitees were similar (excluding gender), these findings should be generalized with caution. Since the participants who dropped out by

Current Smokers

Figure 2. Path analysis model for current smokers. Baseline variables are shown on the left, intermediate mediating variables are shown in the middle, and the outcome (48-month smoking frequency) is shown on the right. Solid lines represent significant ($p < .05$) paths, and dotted lines are not significant.

48 months and participants with parent noncompleters tended to be heavier smokers at baseline, the results may underreport the effects of MSP and mother's ever-smoking. Also, the current model cannot distinguish environmental from genetic contributions of pathways. Individual variability may exist within significant pathways, especially in terms of specific mechanisms (e.g., certain environmental mechanisms may not be relevant for offspring of ex-smoking mothers). Additionally, the accuracy of these results depends on adolescent self-reports of smoking behavior, cumulative cigarettes smoked/lifetime, and their mother's concurrent/lifetime smoking, which may be underreported (Harakeh et al., 2006); and on retrospective parent reports of MSP; however, reliability is likely given the trimester-specific assessments (Pickett et al., 2009). Multiple preliminary analyses examining coding variants of MSP and mother's smoking increased the Type I error, and thus the current results are exploratory in nature. Finally, these results using observational data cannot be used to conclude causation.

This study also has several advantages that strengthen the relevance of these findings. Though previous studies have focused on adolescent offspring (Cornelius et al., 2005; Kandel et al., 1994), this is the first to utilize longitudinal, prospective data of smoking behavior among youth who were oversampled for early stages of smoking. This is one of few studies to simultaneously examine multiple potential mediating pathways of MSP and mother's ever-smoking. The current results add to a small but growing body of literature on early emerging ND. Finally, modeling parallel mediating pathways allows an examination of their separate contributions, which has important implications for intervention strategies.

The results presented here are relevant to efforts to reduce youth smoking. Intervention strategies aimed at preventing progression to heavier smoking could identify high-risk youth based on jointly having an ever-smoking mother and experiencing early emerging ND. Targeting ND and/or smoking frequency during adolescence may prevent further

increases in smoking regularity though these approaches' effectiveness is uncertain. Finally, the findings can help to inform families of the specific contributing pathways through which MSP and mother's smoking acts. Education about the mechanisms of MSP and mother's lifetime smoking, as well as an improved ability to identify and intervene with high-risk youth, will help curb smoking behavior before it progresses to more serious levels.

FUNDING

This work was supported by the National Cancer Institute (grant number P01 CA098262 to RM); and the National Institute on Drug Abuse (grant numbers R01 DA022313-01A2, R01 DA022313-02S1 to LD, R21 DA024260 to JR, and Center Grant P50 DA010075 to Penn State University). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Cancer Institute, the National Institute on Drug Abuse, or the National Institutes of Health.

DECLARATION OF INTERESTS

None declared.

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